COUNTWAY LIBRARY
HC 18ZL 8

THE DISEASES OF INFANCY AND CHILDHOOD

COPYRIGHT, 1897, 1902, 1905, 1907, 1909, 1911, 1916,
BY D. APPLETON AND COMPANY

Printed in the United States of America

PREFACE TO THE SIXTH EDITION.

In the preparation of this edition the author has associated with him Dr. John Howland, his former assistant, who will hereafter be connected with the work as joint author.

Progress along many lines in pædiatrics has been rapid during the last two years. To make room for new knowledge without unduly enlarging the size of the volume has made it necessary to cut out about seventy-five pages of old material. It is believed that this has been accomplished without impairing the value of the chapters which have been abridged. The decision of the publishers to make entirely new plates has made this comparatively easy.

There is scarcely a page in the book which has not been subject to some revision. Many articles have been entirely rewritten and several new ones appear for the first time in this edition. The greater part of the new material will be found in the chapters upon Nutrition and Infant Feeding, Infant Mortality, Intestinal Intoxication, Pyloric Stenosis, Appendicitis, Acute Peritonitis, Endocarditis and Pericarditis, Cerebro-spinal and Other Forms of Acute Meningitis, Acute Poliomyelitis, Hereditary Syphilis and Tuberculosis.

A number of the old illustrations have been omitted as no longer necessary; others have been replaced by better ones. In all, thirty-six new illustrations have been introduced, including twelve radiographs. All illustrations are from original sources unless otherwise stated.

The authors desire to acknowledge their indebtedness to Dr. F. H. Bartlett for much assistance rendered in every way in the work of revision; to Dr. H. H. Mason for correction of the proof sheets, and to Dr. N. C. Holt for the preparation of the index.

14 West Fifty-fifth Street,
New York.

Digitized by the Internet Archive
in 2025

https://archive.org/details/diseasesofinfanc0000unse_s8n0

PREFACE TO THE SEVENTH EDITION

In this Seventh Edition the authors have endeavored to bring the book abreast of the science of the day. In the five years which have elapsed since the last revision there have been great advances in our knowledge of many of the subjects which are considered in a general textbook upon Pediatrics. The endeavor has been made to introduce this new knowledge without greatly changing the general arrangement of the book. To do this without increasing the size of the volume has made it necessary to cut out nearly one hundred pages of old material, and to condense other portions of the book. It is believed that this has been done without impairing the value of the chapters which have been abridged. The decision of the publishers to make entirely new plates has made this comparatively easy.

There is scarcely a page in the book but has been subjected to revision. Sixteen new articles appear for the first time in this edition. The more important ones are Acidosis, Neuropathic and Exudative Diathesis, Cardiac Arrhythmia, Acute Lymphatic Leukemia, Banti's Disease, Osteogenesis Imperfecta, Still's Disease, Syphilis of the Nervous System, Pellagra, Epidemic Catarrh, Duodenal Ulcer, and Idiosyncrasies to Food Stuffs.

More than twenty chapters have been almost entirely rewritten, the most important being those upon Birth Paralyses, Milk and Infant Feeding, Digestion in Infancy, Chronic Intestinal Indigestion, Hirschsprung's Disease, Asthma, Accidental Heart Murmurs, Hydronephrosis, Gonococcus Vaginitis, Tetany, Convulsions, Epilepsy, Hydrocephalus, Poliomyelitis, Diseases of Ductless Glands, Diabetes, Hodgkin's Disease, and Tuberculous Adenitis.

Many old illustrations have been omitted and fifteen new ones introduced, all of them from original sources. Especial attention has been devoted by the authors to the newer methods of diagnosis and treatment.

The authors desire to acknowledge the assistance of Dr. N. Curtice Holt in the correction of the proof sheets and the preparation of the index.

L. Emmett Holt,
John Howland.

CONTENTS

PART I

CHAPTER PAGE

I.—Hygiene and General Care of Infants and Young Children . 1

Care of the newly-born child; bathing; clothing; care of the eyes; care of the mouth and teeth; care of the skin; care of the genital organs; vaccination; training to proper control of rectum and bladder; general hygiene of the nervous system; sleep; exercise; airing; the nursery; the nurse; the amount of air space required by infants; the care of premature and delicate infants; incubators; the feeding of the premature infant.

II.—Growth and Development of the Body 15

Weight; height; growth of extremities as compared with the trunk; the head; the chest; the abdomen; muscular development; development of special senses; speech; dentition.

III.—Peculiarities of Disease in Children 30

Etiology; symptomatology and diagnosis; pathology; prognosis and infant mortality; prophylaxis; therapeutics.

PART II

Section I.—Diseases of the Newly Born

I.—Asphyxia 69

II.—Congenital Atelectasis 74

III.—Icterus 77

IV.—The Acute Infections of the Newly Born 82

The acute pyogenic diseases; ophthalmia; tetanus; epidemic hemoglobinuria; fatty degeneration of the newly born; pemphigus.

V.—Hemorrhages 96

Traumatic or accidental hemorrhages; spontaneous hemorrhages.

VI.—Birth Paralyses 106

Cerebral paralysis; facial paralysis; brachial paralysis.

CHAPTER PAGE

VII.—Tumors of the Umbilicus, Mastitis, etc. 114

Umbilical hernia; mastitis; intestinal obstruction; diaphragmatic hernia; congenital stridor; sclerema; inanition fever.

Section II.—Nutrition

I.—Introductory 127

The food constituents and the purposes they subserve in nutrition.

II.—The Infant's Dietary 134

Woman's milk; cow's milk; top milk—skimmed milk; milk sterilization; commercial pasteurization of milk; pasteurization vs. sterilization; methods of heating milk; frozen milk; peptonized milk; condensed milk; dried milk; buttermilk and other forms of fermented milk; protein milk; junket, curds and whey; beef preparations; cereals; infant foods.

III.—Infant Feeding 165

Choice of methods; breast feeding; maternal nursing; wet-nursing; weaning; mixed feeding; artificial feeding; formulas for whole milk; schedule for healthy infants during the first year; feeding in difficult cases.

IV.—Feeding after the First Year 209

Healthy infants during the second year; feeding from the third to the sixth year; feeding during acute illness; idiosyncrasies to foodstuffs; acidosis.

V.—The Derangements of Nutrition 218

Inanition; marasmus; malnutrition.

VI.—Diseases Due to Faulty Nutrition 231

Scorbutus; rickets.

VII.—Diatheses 260

The exudative diathesis; the neuropathic diathesis.

Section III.—Diseases of the Digestive System

I.—Diseases of the Lips, Tongue, and Mouth 267

Malformations; diseases of the lips; diseases of the tongue; dental caries; alveolar abscess; difficult dentition; catarrhal stomatitis; herpetic stomatitis; ulcerative stomatitis; thrush; gonorrheal stomatitis; syphilitic stomatitis; diphtheritic stomatitis; gangrenous stomatitis.

II.—Diseases of the Pharynx 288

Acute pharyngitis; uvulitis; elongated uvulva; retropharyngeal abscess; adenoid vegetations of the vault of the pharynx.

CHAPTER PAGE

III.—Diseases of the Tonsils 300

Membranous tonsillitis; ulceromembranous tonsillitis; follicular tonsillitis; phlegmonous tonsillitis; chronic hypertrophy of the tonsils.

IV.—Diseases of the Esophagus 311

Malformations; acute esophagitis; retro-esophageal abscess.

V.—Diseases of the Stomach 315

Digestion in infancy; malformations and malpositions of the stomach; hypertrophic stenosis of the pylorus; vomiting; cyclic vomiting; acute gastritis; chronic gastric indigestion; dilatation of the stomach; ulcer of the stomach; duodenal ulcers; tumors of the stomach; hemorrhage from the stomach; the swallowing of foreign bodies.

VI.—Diseases of the Intestines 348

Malformations and malpositions; diarrhea; acute intestinal indigestion and diarrhea.

VII.—Diseases of the Intestines (*continued*) 373

Acute ileocolitis—dysentery; amebic colitis; amyloid degeneration of the intestines; tuberculosis of the intestines and mesenteric lymph nodes.

VIII.—Diseases of the Intestines (*continued*) 395

Chronic intestinal indigestion; intestinal colic; chronic constipation; hypertrophy and dilatation of the colon; intussusception.

IX.—Diseases of the Intestines (*continued*) 418

Appendicitis; intestinal worms.

X.—Diseases of the Rectum 430

Prolapsus ani; fissure of the anus; proctitis; ischiorectal abscess; rectal polypus; hemorrhoids; incontinence of feces.

XI.—Diseases of the Liver 436

Catarrhal jaundice; new growths; acute yellow atrophy; congestion of the liver; abscess of the liver—suppurative hepatitis; cirrhosis; amyloid degeneration; fatty liver; hydatids; biliary calculi.

XII.—Diseases of the Peritoneum 444

Acute peritonitis; chronic (non-tuberculous) peritonitis; tuberculous peritonitis; ascites; subphrenic abscess.

Section IV.—Diseases on the Respiratory System

I.—Nasal Cavities 457

Acute rhinopharyngitis; chronic nasal catarrh; chronic rhinitis; epistaxis.

CHAPTER PAGE

II.—Diseases of the Larynx. 465

Catarrhal spasm of the larynx; acute catarrhal laryngitis; submucous laryngitis—edema of the glottis; chronic laryngitis; new growths; foreign bodies in the larynx and bronchi.

III.—Diseases of the Lungs 476

The peculiarities of the lungs in infancy and early childhood; acute catarrhal bronchitis; fibrinous bronchitis; chronic bronchitis; asthma.

IV.—Diseases of the Lungs (*continued*) 492

Pneumonia; acute bronchopneumonia.

V.—Diseases of the Lungs (*continued*) 526

Lobar pneumonia; pleuropneumonia; hypostatic pneumonia; chronic bronchopneumonia—chronic interstitial pneumonia—bronchiectasis; abscess of the lung; gangrene of the lung; acquired atelectasis—pulmonary collapse; emphysema.

VI.—Pleurisy 557

Dry pleurisy; pleurisy with serous effusion; empyema.

Section V.—Diseases of the Circulatory System

I.—Peculiarities of the Heart and Circulation in Early Life . . 575

II.—Congenital Anomalies of the Heart 579

III.—Pericarditis 588

Acute pericarditis; chronic pericarditis with adhesions.

IV.—Endocarditis and Valvular Disease of the Heart 594

Malignant endocarditis; myocarditis; accidental murmurs; functional disturbances of the heart; diseases of the blood vessels.

Section VI.—Diseases of the Urogenital System

I.—The Urine in Infancy and Childhood 615

Lordotic, orthostatic or cyclic albuminuria; hematuria; hemoglobinuria; pyuria; anuria; diabetes insipidus.

II.—Diseases of the Kidneys 623

Malformations and malpositions; uric-acid infarctions; chronic congestion of the kidney; acute degeneration of the kidneys; acute diffuse nephritis; chronic nephritis; tuberculosis of the kidney; tumors of the kidney; pyelitis—pyelocystitis; renal calculi; perinephritis.

CHAPTER PAGE

III.—Diseases of the Genital Organs 650
Malformations; diseases of the male genitals; diseases of the female genitals; gangrenous vulvitis.

IV.—Enuresis 662
Vesical calculus.

Section VII.—Diseases of the Nervous System

I.—Introductory 669

II.—General and Functional Nervous Diseases 671
Convulsions; tetany; epilepsy; chorea; other spasmodic affections; hysteria; headaches; disorders of speech; disorders of sleep; injurious habits of infancy and childhood.

III.—Diseases of the Brain and Meninges 719
Malformations; pachymeningitis; acute meningitis; cerebrospinal meningitis; acute meningitis due to other causes; tuberculous meningitis; chronic basilar meningitis in infants; thrombosis of the sinuses of the dura mater; cerebral abscess; cerebral tumor; hydrocephalus; chronic internal hydrocephalus; infantile cerebral paralysis; amaurotic family idiocy; mental deficiency; Mongolian idiocy; deaf-mutism.

IV.—Diseases of the Spinal Cord 796
Malformations; spinal meningitis; myelitis; compression-myelitis; acute poliomyelitis; tumors of the spinal cord; hereditary ataxia, diseases associated with progressive wasting; congenital myatonia.

V.—Diseases of the Peripheral Nerves 828
Multiple neuritis; diphtheritic paralysis; facial paralysis.

Section VIII.—Diseases of the Blood, Lymph Nodes, Ductless Glands, Bones and Joints

I.—Diseases of the Blood 839
Secondary anemia; chlorosis; pseudoleukemic anemia; pernicious anemia; leukemia; hemophilia; purpura.

II.—Diseases of the Lymph Nodes 860
Simple acute adenitis; simple chronic adenitis; syphilitic adenitis; tuberculous adenitis; Hodgkin's disease.

III.—Diseases of the Ductless Glands 876
The spleen; enlargement of the spleen; diseases of the thyroid; sporatic cretinism; hypothyroidism; Graves' disease; hyperthyroidism; diseases of other ductless glands; diseases of the thymus; status lymphaticus.

CHAPTER PAGE

IV.—Diseases of the Bones and Joints 896
Osteogenesis imperfecta; chondrodystrophy; acute arthritis of infants; chronic arthritis; tuberculous diseases of the bones and joints.

V.—Diseases of the Skin 920
Congenital ichthyosis; miliaria; seborrhea; eczema; furunculosis; gangrenous dermatitis; impetigo contagiosa; urticaria; scabies; tinea tonsurans—ringworm of the scalp.

VI.—Diseases of the Ear 938
Acute otitis.

Section IX.—The Specific Infectious Diseases

I.—Scarlet Fever 952

II.—Measles 975

III.—Rubella 991

IV.—Varicella 994

V.—Vaccinia—Vaccination 997

VI.—Pertussis 1003

VII.—Mumps 1015

VIII.—Diphtheria 1020

IX.—Typhoid Fever 1059

X.—Tuberculosis 1067

XI.—Syphilis 1103

XII.—Influenza 1130

XIII.—Malaria 1139

Section X.—Other General Diseases

I.—Rheumatism 1149

II.—Diabetes Mellitus 1155

III.—Pellagra 1158

Index 1163

LIST OF ILLUSTRATIONS

PLATES

PLATE		FACING PAGE
I.	Chart showing by months the mortality of New York City for the different ages for three consecutive years	44
II.	A, Costochondral junction in early rickets; B, Normal costochondral junction	244
III.	Typical rickets	248
IV.	Deformity of the chest in severe rickets	252
V.	Extensive superficial ulceration of the colon	376
VI.	Deep follicular ulcers of the colon	378
VII.	Membranous inflammation of the ileum	386
VIII.	Acute bronchopneumonia	498
IX.	Acute pleuropneumonia	546
X.	Chronic bronchopneumonia	548
XI.	Acute pneumococcus meningitis, complicating pleuropneumonia	744
XII.	A, Blood of an eight-months' fetus; B, Simple anemia; C, von Jaksch's anemia; D, Acute lymphatic leukemia	840
XIII.	Tuberculosis of the tracheobronchial lymph nodes	1078

ILLUSTRATIONS IN THE TEXT

FIGURE		PAGE
1.	Weight curve for the first twenty days	16
2.	Weight curve for the first year	17
3.	Skull, showing premature ossification	23
4.	Deaths, New York City, per 1,000 of population	41
5.	Deaths by months, New York City	45
6.	Chief causes of death first year	47
7.	Colon of a child six months old	65
8.	Pemphigus neonatorum	95
9.	Triple cephalhematoma	98
10.	Meningeal hemorrhage of the newly born	107
11.	Erb's paralysis	112
12.	Umbilical fistula and tumors	115
13.	Diaphragmatic hernia	119
14.	Temperature chart in inanition fever	124
15.	Apparatus for examination of human milk	139

FIGURE PAGE
16. A, Babcock tubes; B, Lewi's modification for human milk . . . 140
17. Chart showing effect of pregnancy on weight of nursing infant . . . 178
18. Case of marasmus 223
19. Case of scurvy 235
20. Costochondral junction in marked rickets 247
21. Rachitic skull, inside view 249
22. Rachitic head 250
23. Rachitic skull, external view 250
24. Rachitic thorax in outline 251
25. Rachitic spine 252
26. Multiple fractures in rickets 253
27. Epithelial desquamation of the tongue 270
28. Thrush 282
29. Adenoid vegetations 295
30. Temperature chart, streptococcus angina following measles . . . 303
31. Gastric peristalsis in pyloric stenosis 323
32. Malformations of the rectum 349
33. Chart showing mortality from diarrheal diseases in New York . . 352
34. Chart showing deaths under one year per 1,000 of population under one year, New York City, summer months 353
35. Temperature chart of acute intestinal intoxication with fatal relapse . 362
36. Acute catarrhal ileocolitis, severe form 376
37. Follicular ulceration of the colon, early stage 378
38. Follicular ulceration of the colon, later stage 379
39. Membranous colitis 380
40. Temperature chart in ileocolitis 383
41. Temperature chart in membranous colitis 385
42. Temperature chart in membranous colitis 386
43. Chronic intestinal indigestion 396
44. Ileocecal intussusception 411
45. Mechanism of intussusception 413
46. An air vesicle in bronchopneumonia 493
47. An air vesicle in lobar pneumonia 494
48. Bronchopneumonia with thickened bronchus 499
49. Bronchopneumonia with emphysema 501
50. Bronchopneumonia, diffuse purulent infiltration 502
51. Persistent bronchopneumonia 504
52. Temperature chart in mild uncomplicated bronchopneumonia . . . 509
53. Temperature chart, prolonged bronchopneumonia 510
54. Temperature chart, relapsing bronchopneumonia 510
55. Temperature chart, rapidly fatal bronchopneumonia 511
56-59. Physical signs in bronchopneumonia 513
60. Temperature chart, persistent bronchopneumonia 515

FIGURE PAGE
61. Temperature chart, bronchopneumonia following pertussis . . . 516
62. Temperature chart, bronchopneumonia complicating influenza . . 518
63. Bronchopneumonia—X-ray 520
64. Temperature chart, typical lobar pneumonia 531
65. Temperature chart, remittent type, lobar pneumonia 532
66. Temperature chart, lobar pneumonia, subnormal temperature after crisis 532
67. Temperature chart, abortive pneumonia 533
68-70. Physical signs, lobar pneumonia 536
71. Lobar pneumonia—X-ray 537
72. General subcutaneous emphysema 557
73. Section of lung, showing distribution of fluid in chest 564
74. Empyema—X-ray 565
75. Empyema—X-ray 565
76. Temperature chart, empyema following pneumonia 566
77. Temperature chart, empyema following pneumonia 566
78. Deformity after old empyema 572
79. Apparatus for inducing pulmonary expansion after empyema . . . 573
80. Congenital cardiac disease 581
81. Clubbing of fingers in congenital cardiac disease 584
82-83. Pericarditis with effusion—X-ray 591
84. Congenital malformations of the kidneys and ureters 626
85. Sarcoma of the kidney 641
86. Tetany 679
87. Meningocele 720
88. Encephalocele 720
89. Hydrencephalocele 720
90. Meningocele 720
91. Frontal meningocele 721
92. Nasofrontal meningocele 721
93. Incidence of cerebrospinal meningitis 727
94. Posture in cerebrospinal meningitis 731
95. Temperature chart, cerebrospinal meningitis, recovery 735
96. Temperature chart, cerebrospinal meningitis, treated by serum . . 739
97. Temperature chart, cerebrospinal meningitis, with late injection of serum 740
98. Seasonal occurrence of tuberculous meningitis 749
99. Tracing of respiration in tuberculous meningitis 751
100. Temperature chart in tuberculous meningitis 752
101. Chronic basilar meningitis 755
102. Chronic basilar meningitis 756
103. Brain in external hydrocephalus 770
104. Brain in internal hydrocephalus 771
105. Section of a normal brain 772

FIGURE PAGE
106. Vertical transverse section of a brain in congenital hydrocephalus . . 774
107. Oxycephaly with exophthalmus 777
108. Scaphocephaly 778
109. Brain showing atrophy 779
110. Convulsions in spastic paraplegia 781
111. Spastic paraplegia 782
112. Recent meningeal hemorrhage 784
113. Infantile hemiplegia showing contractures 786
114. Brain in idiocy 791
115-117. Various types of mental defect 792
118-120. Mongolian idiocy 794
121. Spina bifida, meningocele (partially diagrammatic) 797
122. Spina bifida, meningocele 798
123. Spina bifida, meningomyelocele (partially diagrammatic) . . . 798
124. Spina bifida, sacral 800
125. Infantile spinal paralysis of lower extremity 815
126. Infantile spinal paralysis of shoulder 816
127. Muscular pseudohypertrophy 825
128. Alcoholic neuritis 830
129. Diphtheritic paralysis 831
130. Facial paralysis 836
131. Acute suppurative adenitis, cervical 863
132. Acute suppurative adenitis, inguinal 864
133. Cicatrices following tuberculous adenitis 871
134-135. Cretins, showing effect of thyroid treatment 883
136-137. Cretins, showing effect of thyroid treatment 885
138. Infantile myxedema 887
139. Enlarged thymus 892
140. Osteogenesis imperfecta—X-ray 896
141. Chondrodystrophy, radiograph of skull 898
142. Chondrodystrophy, long bones 898
143. Chondrodystrophy, infantile figure 899
144. Chondrodystrophy, trident hand 899
145. Chondrodystrophy, adult figure 899
146. Section of the spine in Pott's disease 908
147. Hip-joint disease 913
148. Tuberculous dactylitis 919
149. Congenital ichthyosis 921
150. Temperature chart, acute otitis, following influenza 940
151. Temperature chart, acute otitis, early paracentesis 941
152. Temperature charts in scarlet fever, mild cases 958
153. Temperature chart in scarlet fever, typical curve 959
154. Temperature chart in severe uncomplicated scarlet fever 960

FIGURE PAGE
155. Temperature chart in fatal septic scarlet fever 961
156. Temperature chart in scarlet fever with late otitis 965
157. Temperature chart in scarlet fever with late nephritis 966
158-159. Temperature charts in measles, typical curve 981
160. Temperature chart in measles, occasional course 982
161. Temperature chart in measles, prolonged course 982
162-163. Temperature charts in measles complicated by pneumonia . . . 983
164. Table showing protective power of vaccination 998
165-169. Vaccination vesicles 1000
170. Generalized vaccinia 1001
171. Temperature chart in typhoid fever, short course 1062
172. Temperature chart in typhoid fever, with relapse 1062
173. Tuberculous bronchopneumonia, diffuse consolidation 1076
174. Cavity from tuberculous bronchopneumonia 1076
175. Pulmonary tuberculosis, extensive caseation 1077
176. Miliary tuberculosis of the lungs 1085
177. Temperature chart of tuberculosis following measles 1088
178. Temperature chart of tuberculous bronchopneumonia, general tuberculosis 1089
179. Temperature chart of tuberculous bronchopneumonia with softening . 1090
180. Tuberculous bronchial glands 1098
181. Early eruption of hereditary syphilis, legs 1111
182. Early eruption of hereditary syphilis, face 1112
183. Syphilitic scaling of the sole 1112
184. A later form of eruption in hereditary syphilis 1113
185. Hereditary syphilis 1115
186. Syphilitic periostitis of the fibula, radiograph 1115
187-188. Syphilitic dactylitis 1116
189-190. Syphilitic dactylitis, radiograph 1117
191. Syphilitic notched teeth 1117
192. Syphilitic teeth, deformed 1118
193. Syphilitic osteoperiostitis of the tibia 1119
194. Syphilitic osteoperiostitis of the tibia, radiograph 1120
195. Temperature chart of severe influenza in an infant 1132
196. Temperature chart of acute bronchopneumonia complicating influenza . 1133
197. Temperature chart, influenza, bronchitis, otitis 1134
198. Temperature chart, double tertian intermittent fever 1141
199. Temperature chart, tertian intermittent fever 1142
200. Temperature chart in malaria, irregular type 1143
201. Pellagra 1160

THE DISEASES OF INFANCY AND CHILDHOOD

PART I

CHAPTER I

HYGIENE AND GENERAL CARE OF INFANTS AND YOUNG CHILDREN

The physical development of the child is essentially the product of the three factors—inheritance, surroundings, and food. The first of these it is beyond the physician's power to alter; the second is largely and the third almost entirely within his control, at least in the more intelligent classes of society. These two subjects, infant hygiene and infant feeding, are the most important departments of pediatrics.

The Care of the Newly-Born Child.—After the ligature of the cord the child should be wrapped in a thick blanket and placed in a warm room. In hospital practice the eyes should be cleansed with absorbent cotton and water which has been boiled, and then two or three drops of a two per cent solution of nitrate of silver, after Credé's method, instilled into each eye by means of a glass rod or eye-dropper. In private practice a ten per cent solution of argyrol may be substituted, unless the mother has had a purulent vaginal discharge, in which case the silver solution should always be used. The bath should now be given in a warm room; the body being first oiled thoroughly in order to remove the vernix caseosa and then washed in water at a temperature of 100° F. The mouth should be cleansed with sterile water and a soft cloth, and no violence employed. The cord may be covered with sterilized talcum or bismuth powder, and wrapped in sterile gauze or surgeon's lint. The abdomen should now be enveloped in a flannel band, eight or ten inches wide, and pinned rather snugly. Before dressing is completed, the child should be submitted to a thorough examination for injuries received during delivery, congenital deformities, also as to the condition of the respiration, circulation, etc.

After dressing, the child should be placed in his crib and covered with blankets, and if the feet are cold, or the fingers and lips a little blue, he should be surrounded by hot-water bottles covered with flannel, and placed near, but not in contact with, the body. The crib should be placed in a quiet, darkened room. The young infant should not occupy the same bed as the mother, unless he greatly needs the warmth of her body, other means of artificial heat not being at hand.

The cord should be kept dry and disturbed as little as possible until it falls off. Under ordinary circumstances the cord separates from the fourth to the seventh day, the average being the fifth day. The stump should then be covered with the sterilized talcum or bismuth powder, and a pad of sterile gauze about one-fourth of an inch thick and two inches square applied and secured in position by means of the abdominal band. The purpose of this is to prevent umbilical hernia. The pad should be continued for the first month. The use of stronger antiseptic dressings than those recommended is somewhat objectionable, since it preserves the cord too long and delays separation. The full bath should not be given until the cord has separated.

The physician should always see to it that the infant cries enough to keep the lungs properly expanded.

The question of food for the newly-born infant is considered in the chapter upon infant feeding.

Bathing.—For the first few months the bath should be given at 98° F. The room should be warm, preferably there should be an open fire. The bath should be short and the body dried quickly, without too vigorous rubbing. The addition of salt to the bath is an advantage where the skin is unusually delicate or excoriations are present. One large handful should be used to a gallon of water. By the sixth month the temperature of the bath for healthy infants may be lowered to 95° F., and by the end of the first year to 90° F. Older children who are healthy should be sponged or douched for a moment at the close of the tepid bath with water at 65° or 70° F. During childhood the warm bath is preferably given at night. In the morning a cold sponge bath is desirable. This should be given in a warm room and while the child stands in a tub partly filled with warm water. This cold sponge should last but half a minute, and be followed by a brisk rubbing of the entire body.

In some young infants and even older children there is no proper reaction after the bath, even when given at the temperatures mentioned; children being pale, slightly blue about the lips and under the eyes. All tub bathing, and especially all cold bathing, should then be stopped, since a continuance can only be a drain upon the child's vitality.

Clothing.—The clothing of infants should be light, warm, non-irritating to the skin, and loose enough to allow free motion of the extrem-

ities; nor should bands be pinned so tightly about the trunk as to embarrass the movements either of the chest or of the abdomen. The chest should be covered with a woollen shirt, high in the neck and with long sleeves. All petticoats should be supported from the shoulders and not from waistbands. Canton flannel and stockinet are both superior as absorbents to the more commonly used linen diapers. Stockinet has the advantage of being very soft and pliable. Care should be taken that in infants the feet be kept warm. If the circulation is very poor, a bag of hot water should always be in the crib. Chilling of the surface is sometimes responsible for attacks of colic.

The abdominal band is usually worn during infancy. It cannot be considered in any sense a necessity after the first few months, except in cases of very thin infants whose supply of fat in the abdominal walls is an insufficient protection to the viscera. For the first few weeks a band of plain flannel is to be preferred; later, a knitted band with shoulder-straps. The fashion of low neck and short sleeves for infants and very young children has fortunately passed away—let us hope, not to return.

During the summer the outer clothing should be light and the under clothing of the thinnest flannel or gauze. The changes in the temperature of morning and evening may be met by extra wraps. The custom of allowing young children to go with legs bare has many enthusiastic advocates; while it may not be objectionable during the heat of summer, its advantages at any season are very questionable in a changeable climate like that of New York or the Atlantic coast. Many delicate children are certainly injured by such ill-advised attempts at hardening.

The night clothing of infants should be similar to that worn during the day, but should be loose, the material being of the lightest flannel. The night clothing for older children should consist of a thin woollen shirt and a union suit with waist and trousers, and in some cases with feet, if there is a tendency to get outside the coverings. The common mistake is to overload all children, but especially infants, with covering at night. This is an explanation of much of the restless sleep which is seen, particularly in delicate children.

Care of the Eyes.—During the first few days at the daily bath the eyes should be cleansed with a saturated solution of boric acid. They should be carefully protected from too strong light during early infancy. It is desirable that a child should always sleep in a darkened room.

Care of the Mouth and Teeth.—The mouth of the newly-born infant should be gently cleansed at each morning bath with boiled water and a soft cloth. On the first appearance of thrush the mouth should be washed after every feeding with a solution of bicarbonate of soda or boric acid (ten grains to the ounce). It should be applied with a swab made by twisting a bit of cotton upon a wooden toothpick, and not by the

nurse's finger. Harm is often done by the use of too much zeal in cleansing the mouth of a young infant.

The primary teeth as well as those of the permanent set should receive daily attention. Too often they are neglected altogether. Dirty teeth are likely sooner or later to become carious; and carious teeth, besides being a cause of bad breath and pain, are a constant menace to the health of the child, since they are frequently the cause of severe infections. Such teeth should either be filled or removed.

Care of the Skin.—The skin of a young infant is exceedingly delicate, and excoriations, intertrigo, and eczema are of very common occurrence. These conditions are much easier of prevention than of cure. The first essential in the care of the skin is cleanliness, and this must be secured without the use of strong soaps or too much rubbing. Napkins must be removed as soon as soiled or wet. Some bland absorbent powder, like starch, talcum, or the stearate of zinc, should be used in all the folds of the skin, in the neck, in the axillae, groins, and about the genitals, and in the folds of the thighs, particularly in very fat infants. If plain water produces an undue amount of irritation, the salt or bran bath should be employed.

Care of the Genital Organs.—The female genitals need but little attention in young children, except as to cleanliness. This is more often neglected in older children than in infants.

In males the prepuce should receive attention during the first few weeks of life. If the foreskin is long and the preputial orifice small, circumcision should be done. If it is not long, but is only adherent, these adhesions should be broken up, the parts thoroughly cleansed and the foreskin retracted daily until there is no disposition to a recurrence of the adhesions. These operations will be discussed more at length in a subsequent chapter. The only thing to be emphasized in the present connection is that the prepuce should receive proper attention in early infancy, since this can now be done with less pain and discomfort to the child, and at the same time better results are obtained. If this matter is neglected during infancy, it is apt to be overlooked until harm has been produced by local or reflex irritation which phimosis or adherent prepuce may have excited.

Vaccination.—This, although considered elsewhere, should be mentioned in this connection as among the things requiring the physician's attention during the first months of life.

Training to Proper Control of Rectum and Bladder.—It is surprising to see what can be accomplished by intelligent efforts at training in these particulars. An infant can often be trained at three months to have its movements from the bowels when placed upon a small chamber. This not only saves a great amount of washing of napkins, but there is soon

formed a habit of having the bowels move at a regular time or times each day. The infant must be put upon the chamber soon after his feeding. The importance of training young children to regular habits regarding evacuations from the bowels can hardly be overestimated. It should be impressed upon every mother and nurse by the physician, and especially the necessity of beginning training during infancy. Much of course will depend upon the food and the digestion; but habit is a very large factor in the case.

The training of the bladder is not quite so important, but the proper education of this organ adds much to the comfort of the child and the ease with which he is cared for. Before the end of the first year many intelligent children can be trained to indicate a desire to empty the bladder. Many mothers and nurses succeed in training children so well that by the tenth or eleventh month napkins are dispensed with during the day. On the other hand, it is very common to see children of two and even two and a half years still wearing napkins because of the lack of proper training. Before he has reached the age of three years a healthy child will usually go from 10 P.M. until morning without emptying the bladder. The annoyance and discomfort from the neglect of early training in this particular are very great. Night feeding is responsible for much of the difficulty experienced in training children to hold the water during the night.

General Hygiene of the Nervous System.—Great injury is done to the nervous system of children by the influences with which they are surrounded during infancy, especially during the first year. The brain grows more during the first two years than in all the rest of life. Normal healthy development of the nervous centers demands quiet, rest, peaceful surroundings, and freedom from everything which causes excitement or undue stimulation.

The steadily increasing frequency of functional nervous diseases among young children is one of the most powerful arguments for greater attention by physicians to the subject of hygiene of the nervous system during infancy. Most parents err through ignorance. Playing with young children, stimulating to laughter and exciting them by sights, sounds, or movements until they shriek with apparent delight, may be a source of amusement to fond parents and admiring spectators, but it is almost invariably an injury to the child. This is especially harmful when done in the evening. It is the plain duty of the physician to enlighten parents upon this point, and insist that the infant shall be kept quiet, and that all such playing and romping as has been referred to shall, during the first year at least, be absolutely prohibited.

Sleep.—The sleep of the newly-born infant is profound for the first two or three days and under normal conditions almost continuous. In

the case of prolonged or tedious labor, or where from any cause undue compression has been exerted upon the head, it may approach the condition of semi-coma for twenty-four or forty-eight hours. This may be so deep as to excite apprehensions of serious brain lesions. If, however, there are associated with it no convulsions and no rigidity, this early stupor usually passes away on the second or third day.

The sleep of early infancy is quiet and peaceful, but not very deep after the first month. After the third year the heavy sleep of childhood is commonly seen. A healthy infant during the first few weeks sleeps from twenty to twenty-two hours out of the twenty-four, waking only from hunger, discomfort, or pain. During the first six months a healthy infant will usually sleep from sixteen to eighteen hours a day, the waking periods being only from half an hour to two hours long. At the age of one year most infants sleep from fourteen to fifteen hours, viz., from eleven to twelve hours at night, and two or three hours during the day, usually in two naps. When two years old usually thirteen to fourteen hours' sleep are taken; eleven or twelve hours at night and one or two hours during the day, generally in a single nap. At the age of four years children require from eleven to twelve hours' sleep. It is always desirable, and in most cases with regularity it is possible, to keep up the daily nap until children are five years old. From six to ten years the amount of sleep required is ten or eleven hours, and from ten to sixteen years nine hours should be the minimum.

Training in proper habits of sleep should be begun at birth. From the outset an infant should be accustomed to being put into his crib while awake and to go to sleep of his own accord. Rocking and all other habits of this sort are useless and may even be harmful. An infant should not be allowed to sleep on the breast of the nurse, nor with the nipple of the bottle in his mouth. Other devices for putting infants to sleep, such as allowing the child to suck a rubber nipple or anything else, are positively injurious. If such means of inducing sleep are resorted to the infant soon acquires the habit of not sleeping without them. We have known of one instance where the habit of rocking during sleep was continued until the child was two years old; the moment the rocking was stopped the infant would wake, and in consequence this practice was continued by the devoted but misguided parents. A quiet, darkened room, a warm and comfortable bed, an appetite satisfied, and dry napkins are all that are needed to induce sleep in a healthy child.

The periods of sleep in young infants are usually from two to three hours long, with the exception of once or twice in the twenty-four hours, when a long sleep of five or six hours occurs. The purpose of training is to have the child take this long sleep at night. The habit of regular sleep is best established by wakening the infant regularly every three or

four hours during the day for feeding, and allowing him to sleep as long as possible during the night. This training goes hand-in-hand with regular habits of feeding. Such habits are easily formed if the plan be systematically followed from the outset.

By the fourth month all feeding between 10 P.M. and 6 A.M. should be discontinued. If this is done most infants can be trained by this time to sleep all night. If the room is lighted, and the child taken from the crib or rocked or fed as soon as he wakens at night, there is no such thing as the formation of good habits of sleep. Regularity in sleep and feeding not only makes the care of young infants very much easier, but is of a good deal of importance for the health of the child.

The causes of disturbed or irregular sleep in young infants are mainly two—hunger and indigestion. In nursing infants it is usually the former; in those artificially fed usually the latter. Sleeplessness from hunger is often seen in children who are nursed thirty or forty minutes and then fall asleep, but wake in fifteen or twenty minutes crying and fretful. After being quieted they may fall asleep again for half an hour, but wake at short intervals. The peaceful sleep of two or three hours which should follow a proper feeding is never seen. With this restlessness from indigestion other signs are usually present, stationary weight, etc. The disturbed sleep due to overfeeding shows itself by much the same symptoms, except that the first sleep after the meal is usually longer.

Exercise.—This is no less important in infancy than in later childhood. An infant gets his exercise in the lusty cry which follows the cool sponge of the bath, in kicking his legs about, waving his arms, etc. By these means pulmonary expansion and muscular development are increased and the general nutrition promoted. An infant's clothing should be such as not to interfere with his exercise. Confinement of the legs should not be permitted. In hospital practice we have often had a chance to observe the bad results which follow when very young infants are allowed to lie in the cribs nearly all the time. Little by little the vital processes flag, the cry becomes feeble, the weight is first stationary, then there is a steady loss. The appetite fails so that food is at first taken without relish, then at times altogether refused; later, vomiting ensues and other symptoms of indigestion. This, in many cases, is the beginning of a steady downward course which goes on until a condition of hopeless marasmus is reached. Such infants must be taken up every few hours and carried about the wards; the position should be frequently changed, and general friction of the entire body employed at least twice a day. Every means must be made use of to stimulate the vital activity. The value of systematic attention to these matters cannot be overestimated in hospitals for infants. Infants who are old enough to creep or stand usually take sufficient exercise unless they are restrained. At this age

they should be allowed to do what they are eager to do. Every facility should be afforded for using their muscles. Exercise may be encouraged by placing upon the floor in a warm room a mattress or a thick "comfortable," and allowing the infant to roll and tumble upon it at will. A large bed may answer the same purpose.

In older children every form of out-of-door exercise should be encouraged—ball, tennis, and all running games, horseback riding, the bicycle, tricycle, swimming, coasting, and skating. Up to the eleventh year no difference need be made in the exercise of the two sexes. Companionship is a necessity. Children brought up alone are at a great disadvantage in this respect, and are not likely to get as much exercise as they require. The amount of exercise allowed delicate children should be regulated with some degree of care. It may be carried to the point of moderate muscular fatigue, but never to muscular exhaustion. The latter is particularly likely to be the case in competitive games.

Exercise should have reference to the symmetrical development of the whole body. In prescribing it the specific needs of the individual child should be considered. By carefully regulated exercises very much may be done to check such deformities as round shoulders and slight lateral curvature of the spine, and also to develop narrow chests and feeble thoracic muscles. For purposes like these, gymnastics are exceedingly valuable to supplement out-of-door exercise, but they can never take their place.

There are two important points with reference to exercise indoors. First, the playroom should be cool—about 60° F. Secondly, during all active exercise the clothing should be loose and light, so as to allow the freest possible motion of the body.

Airing.—In summer there can be no possible objection to a young infant being allowed out of doors at the end of the first week. He should be kept in the open air as much as possible during the day. In the fall and spring this should not be permitted until the child is at least a month old, and then only when the out-of-door temperature is above 60° F. During his outing the head should be protected from the wind and the eyes from the sun. The duration of the outing at first should be only fifteen or twenty minutes, the time being gradually lengthened to two or three hours. The child should be gradually accustomed to changes of temperature in the room by opening wide the windows for a few minutes each day even before he is taken out of doors, the child being dressed meanwhile as for an outing. In the case of children born late in the fall or in the winter this means of giving fresh air may be advantageously begun at one month and followed throughout the first winter. It is only necessary in all such cases that the changes be made very gradually both as to the length of the airing and as to the temperature. The great

advantage of this plan over that more commonly followed of keeping young infants closely housed for the first six months in case they are born in the fall or early winter, we can positively affirm from quite a wide observation of both methods. It is a matter of very serious importance that every infant be furnished an abundance of pure fresh air in winter as well as in summer. When the plan above outlined is carefully and judiciously followed, the tendency to catarrhal affections instead of being increased is thereby greatly lessened.

When four or five months old, there is no reason why a healthy child should not go out of doors on pleasant days if the temperature is not below 20° F. While there is a prejudice on the part of many mothers and some physicians against a child's sleeping out of doors in cold weather, it is a practice which we have always urged upon mothers, and have never seen followed by any but the most beneficial results. The days of all others when infants and very young children should not be out of doors are when there are high winds, especially those from the northeast, an atmosphere of melting snow, and during severe storms. Delicate infants must of course be more carefully guarded during the cold season. With most of these the plan of house-airing is all that should be attempted.

Nursery.—This should be the sunniest and best-ventilated room in the house. It is the physician's duty to see that proper attention is paid to the hygiene of the room in which the child spends at least four-fifths of his time during the first year, and two-thirds of his time during the first two or three years of life. Sunlight is absolutely indispensable. Sunny rooms always contain less organic matter and less humidity, and hence a room upon the north side of the house should always be avoided; preferably one in the second story should be chosen. Nothing which can in any way contaminate the air of the room should be allowed. There should be no washing and drying of clothes or of napkins. No food should be allowed to stand about the room. Gas should not be allowed to burn at night; a small wax night-light furnishes all that is needed in the nursery. If possible the heat should be from an open fire; the next best thing is the Franklin heater. Nothing in the room is worse than steam heat from a radiator unless it be a gas stove, which under no circumstances should be allowed, except possibly for a few minutes each morning during the bath.

The temperature of the room during the day should not be over 70° F. It is important that every nursery should have a thermometer, and that this and not the sensations of the nurse should be the guide. It is almost invariably true that the nursery is overheated. Often no other explanation can be found for chronic indigestion and falling weight excepting a nursery whose habitual temperature ranges from 75° to 80°

F. At night for the first few weeks the temperature should not be allowed to fall below 65° F. After two months the night temperature may fall to 60° or even 50° F.

Free ventilation without draughts is an absolute necessity. This is best accomplished by ventilators in the windows, of which there are many excellent devices sold in the shops. While the child is absent from the room the windows should be widely opened and free airing of the nursery accomplished. The room should always be thoroughly aired at night before the child is put to bed. After the first year the window may be open, unless the outside temperature is as low as 20° F. If the window is open the door of the nursery should be closed, that currents of air may be avoided. The ventilation by means of an open fire is the most efficient.

The furniture of the nursery should be as simple as possible, heavy hangings should be positively forbidden, and upholstered furniture used only to a small extent. Floors covered by large rugs are much more cleanly than carpets, and hence are to be preferred.

The child, whenever it is possible, should have a separate bed; and so should the newly-born infant, in order to prevent the danger of overlying by the mother, which is seen as an occasional cause of death, and also to avoid the danger of too frequent night nursing, which is injurious alike to mother and child. Separate beds for older children will prevent the spread of many forms of infection. The crib for infants should be one which does not rock, in order that this unnecessary and vicious practice may not be carried on. The mattress should be of hair and quite firm. The pillow should be small; in the summer, hair pillows are an advantage but not a necessity. The position of the child during sleep should be changed from time to time from one side to the other and then to the back. Attention to all these details should not be beneath the physician's notice, since the violation of these plain rules of hygiene is at the bottom of many of the milder disorders and even of some of the more serious diseases seen in infancy.

The Nurse.—The nurse of a young child should be healthy, young or in middle life, free from tuberculous or syphilitic taint, from catarrhal affections of the nose and throat, and not of a nervous or excitable temperament. She should be neat in habit, of quiet disposition, and, most of all, she should be a person of intelligence.

The Amount of Air Space Required by Infants.—The nursery should always be as large a room as possible. One of the reasons why young infants do so badly in institutions is because of overcrowding. In a well-ventilated ward there should be allowed to each infant at least 1,000 cubic feet. Children over two years old are not so sensitive to their surroundings, and may thrive in wards where only 700 or 800 cubic feet are allowed to each child.

THE CARE OF PREMATURE AND DELICATE INFANTS

Infants born before term, and some exceedingly delicate ones who are born at full term, require very special and particular care. The vitality is so feeble in these children that if they are handled in the ordinary way they survive at most but a few weeks. The symptom which indicates that such special care is necessary is most of all the weight of the child. Either congenital feebleness or prematurity may be assumed in most of the children weighing less than five pounds; also if the length of the body is less than nineteen inches. In these children all the organs are likely to be imperfectly developed and they are not ready for their work. Especially is this true of the lungs and of the organs of digestion.

The clinical picture presented by these cases is quite characteristic. The body is limp; the skin very soft and delicate and almost transparent; the cry, a low feeble whine not unlike the mew of a kitten; the respiratory movements, extremely irregular, sometimes scarcely perceptible for several seconds; the movements of the extremities infrequent and never vigorous. The general appearance is one of torpor. The muscles of the mouth and cheek and tongue may lack the requisite force for sucking, so that this is practically impossible, and even deglutition is slow, difficult, and prolonged. It is difficult to maintain the normal body temperature; unless closely watched this may fall far below the normal, and may rise quite as much above it with the use of too much artificial heat. We once saw a fluctuation of 13° F. occur in a few hours from such causes. All the symptoms mentioned vary much according to the degree of prematurity.

In the management of these cases there are three problems to be solved: the first to maintain the animal heat, the second to nourish the infant, the third to prevent infection. Difficult as it always is to rear a premature infant, these difficulties are much increased in cases where proper means are not adopted immediately after birth. The loss which these children sustain during the first few days is in very many cases so great that subsequent measures, however well carried out, are futile. The heat-producing power is so feeble that the body temperature quickly falls below normal unless artificial heat is constantly used. The effect of cold upon these delicate infants is very serious, and not only growth but even life depends upon maintaining the body temperature steadily and uniformly. Their extreme susceptibility is something which it is difficult for one to appreciate who has not had experience in these cases.

One of the simplest means of maintaining the temperature is to oil the skin and then roll the entire body, including extremities, in absorbent cotton or lamb's wool; even the neck and cranium may be covered, leaving only the face exposed. The usual diaper may be replaced by a pad of

gauze and absorbent cotton. The body is then wrapped in blankets, placed in a clothes-basket or bassinet with protected sides, and surrounded by bottles or bags containing hot water. A blanket or sheet should partially cover the top of the basket, forming a sort of hood to protect the eyes from light and the face and head from draughts. In using hot-water bags, some caution must be exercised or too much heat may be secured. We have seen the temperature of an infant raised six or seven degrees from this cause. The temperature of the child should at first be taken every few hours to make sure that a proper amount of external heat is supplied.

A more efficient means of furnishing artificial heat is by the electric pad. These small heaters may be attached like a drop-light to any electric fixture. A convenient size is ten by fifteen inches. The pad, which can be obtained of any electric supply company, is placed beneath two or three thicknesses of blanket, upon which the infant lies in its basket. Since the pads occasionally get out of order they must be used with some caution, as they have been known to burn the bedclothes and even the baby.

With such means as those described it is possible to maintain the body temperature at normal even in a room kept at the ordinary temperature. It is preferable to have a warmer room; 80° or even 85° F. is desirable for feeble infants. Adequate ventilation, however, is indispensable. With intelligent care excellent results can, however, often be obtained with no other means for maintaining heat than the padded basket and hot-water bottles; but the other accessories make the problem an easier one.

Premature infants should be fed without being removed from the basket, until they are strong enough to take the breast. The position should be frequently changed and some freedom of movement of the limbs permitted, but the infants should be handled as little as possible. The body should be oiled and fresh cotton applied every other day. The rectal temperature at first should be taken several times a day in order to be sure that sufficient artificial heat is being supplied, but not too much. The latter condition is one that often obtains. So long as the rectal temperature varies only between 98° and 100° F. one should be satisfied.

Incubators.—Personally, we have not found the usual small incubator a very satisfactory means of caring for the premature infant. The difficulties in successful operation are many and the dangers consequent upon the mode of ventilation are considerable. Except by persons experienced, their use is not to be advised. In hospitals with specially trained nurses they may give excellent results, but in the average home the simpler measures above described are much safer and quite efficient.

Every institution receiving and caring for premature infants should have a specially equipped room for that purpose. It should be of sufficient size to accommodate several patients. We have had such a room constructed in the Babies' Hospital which seems to fulfill all the requirements. The room has a floor space of thirteen by sixteen feet with ceiling eleven feet high. This is arranged for five infants, which gives each child 450 cubic feet of air. The cribs are separated by glass plates, which project three feet from the side wall and are four feet in height, forming an alcove for each infant. The purpose of this is to diminish the chances of bed-to-bed infection. The room has double partition walls and double windows. The temperature is controlled by a thermostat regulator and is maintained at about 90° F. The room is provided with a special ventilating apparatus by means of which the entire air of the room can be changed in a few minutes. This is done several times a day. Such a room possesses all the advantages of the small incubator without any of its drawbacks. The infants are clothed in a single loose garment of absorbent cotton and cheese-cloth and lightly covered. In this room the normal body temperature is easily maintained. For wet-nursing, bathing, and changing of napkins, the children are removed to an anteroom which is kept at a temperature of about 75° F. When the bottle is given they are fed in their cribs. After reaching the weight of about five pounds they are removed to the anteroom for a few days, after which they are placed in the ward or sent home.

Feeding.—The feeding of the premature infant is not less important than the maintenance of heat and proper ventilation. Infants at eight months and those weighing five pounds or thereabouts can usually be made to take the breast after the first few days. Few below this age or weight will do so. Some will suck from a bottle, but the majority must be fed by other means. A medicine dropper may be used, or the Breck feeder; the smallest and feeblest, however, must be fed by gavage, using a funnel and small rubber catheter. The food should be slowly given; if rapidly, some is liable to be regurgitated, and this may produce attacks of asphyxia or even an aspiration pneumonia. The quantity of food and frequency of feeding will depend upon the size and age of the child. A seven months' baby weighing three and a half pounds should have, for the first twenty-four hours, only water, one to three teaspoonfuls every hour. Then regular food every three hours beginning with half an ounce, increased to one ounce in a few days and gradually to one and a half or two ounces at the end of about three weeks.

Artificial feeding is seldom very successful with premature infants. With some of the larger and more vigorous, cow's milk modified according to the directions given in the chapters on Infant Feeding gives good results. We once succeeded with a child of three pounds two ounces. For

most of them under four and a half pounds, breast-milk is essential. If the child is born near term, the mother may be able to nurse it. Occasionally this may be done at eight months, but seldom earlier, so that the milk of some other woman must usually be depended upon.

As the premature baby requires only from six to eight ounces of breast-milk a day for the first few weeks, this may be secured from some other nursing woman; a friend might be willing to furnish it or it could be purchased from any healthy woman who has an abundant supply. It is sufficient if it is drawn fresh twice a day, the utmost precautions, of course, being taken to secure cleanliness. At first equal parts of breast-milk and a four- or five-per-cent solution of milk sugar may be given; the degree of dilution being gradually lessened until pure milk is taken. Eight feedings a day are usually necessary, the amount at one feeding may be from two drams to one ounce depending upon the size, age, and digestive powers of the infant. It is not important that the baby of the woman furnishing the milk should be of the same age as the foster infant. The milk of any woman whose baby is between one and eight months old will answer. We have successfully fed premature infants with breast-milk from women whose children were older than this. Another plan is to secure a wet-nurse and permit her to bring her own baby into the house. She expresses for the premature infant the required amount of milk three or four times a day, and the rest of the time nurses her own child. In this way her flow of milk is maintained; if the breasts are pumped exclusively the supply rapidly diminishes. The secretion of the milk in the mother may be promoted by her suckling the wet-nurse's baby or some other vigorous infant. The above are temporary expedients and in most instances need not be continued more than two or three weeks, at the end of which time the mother may be able to nurse her own child.

The results with premature babies will depend very much upon how soon after birth they receive proper care. Immediately after birth measures should be taken to secure the best care and provide adequately for

Age.	Tarnier saved without incubators.	Tarnier saved with incubators.	Voorhees saved with incubators.	Voorhees saved excluding cases dying a few hours after birth.
Born at 6 months.....	0.0%	16.0%	.....	.....
" " 6½ "	29.5%	36.6%	22.0%	66.0%
" " 7 "	39.0%	49.8%	41.0%	71.0%
" " 7½ "	54.0%	77.0%	75.0%	89.0%
" " 8 "	78.0%	88.8%	70.0%	91.0%
" " 8½ "	88.0%	96.0%	.....	.....

maintaining the body heat. If an incubator is to be used it should be in readiness, so that the child can be put into it as soon as he is breathing properly. The age and vigor of the infant are of the greatest impor-

tance in estimating the chances of survival. The table on the preceding page gives Tarnier's statistics, showing the percentage of premature infants saved during a period of five years without the incubator, and during the succeeding five years with the incubator; also the percentage saved at the Sloane Hospital for Women (New York), as published by Voorhees.

Results will improve with the experience of the physician in the feeding and care of these very sensitive patients. Much is yet to be learned about them.

CHAPTER II

GROWTH AND DEVELOPMENT OF THE BODY

Observations upon growth and development are of the utmost importance during infancy and childhood. Only by this means are very many diseases detected in their incipiency. Early recognition carries with it in most cases the possibility of checking such pathological processes as, if allowed to go on, may affect the health not only in infancy but even throughout life.

By familiarity with what is normal, detection of the abnormal soon becomes easy. Investigation in regard to these subjects should be made a part of the physical examination of every child.

WEIGHT

The weight of the infant is the best means we have to measure his nutrition. It is as valuable a guide to the physician in infant feeding as is the temperature in a case of continued fever. Although the weight is not to be taken as the only guide to the child's condition, it is of such importance that we cannot afford to dispense with it during the first two years. It is of great advantage to keep up regular observations during childhood.

Weekly weighings should be made for the first six months, bi-weekly for the rest of the first year, and monthly during the second year. Delicate children should be weighed even more frequently. Balance scales only should be used. The spring scales are not reliable.

Weight at Birth.—The following figures are taken consecutively in nearly equal proportion from the records of the Nursery and Child's Hospital, the Sloane Hospital, and the New York Infant Asylum, and include only full-term children:

Average weight of	568 females	7.16 lbs.	(3,260 grams).
" "	590 males	7.55 "	(3,400 ").
" "	1,158 infants	7.35 "	(3,330 ").

Weight Curve during the First Few Weeks.—The accompanying chart represents the variations in weight for the first twenty days. These observations were made upon one hundred healthy, nursing infants, fifty males and fifty females, at the Nursery and Child's Hospital. The children were weighed daily during the period of observation. The average weight at birth was 7.1 pounds. The curve shows a very marked loss of weight on the first day and a slight loss on the second day, the lowest point being touched at the beginning of the third day; but from this time there was a steady gain. The average initial loss in

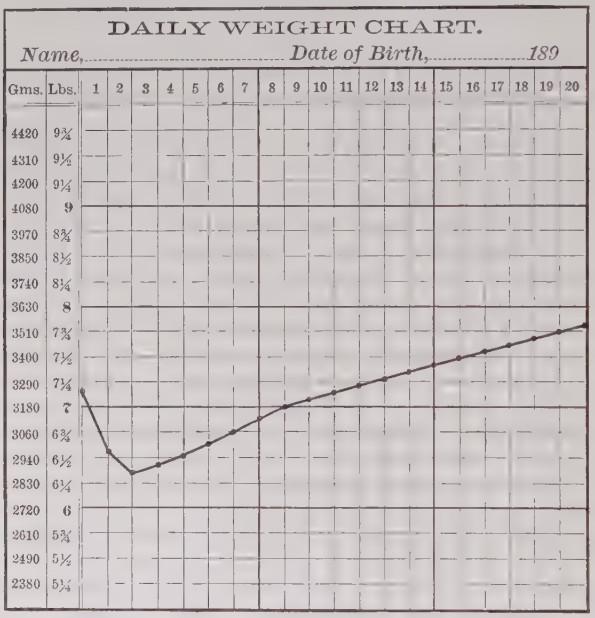


FIG. 1.—WEIGHT CURVE OF THE FIRST TWENTY DAYS.

these cases was ten ounces, being in each sex exactly eleven per cent of the body weight. In eight hundred and thirty-five cases, including those above mentioned, the average loss was nine and a half ounces. The loss of the first days is chiefly due to the discharge of the meconium and urine, but is in part from the excess of tissue waste over the nutriment derived from the breasts. After the third day, coincident with an abundant secretion of milk, there is a steady, daily increase in weight. If the milk is very scanty or is wanting altogether, the loss in weight continues.

The birth-weight of nursing children who thrive normally is regained on the average on the tenth day. The most frequent deviation from the normal curve consists in a continued loss or stationary weight after the

third day. This may be due to acute illness, such as bronchitis, diarrhea, pyemia, or hemorrhage, but in the majority of cases there is a disturbance of nutrition from improper or insufficient food.

The weight curve of infants who are artificially fed, even though they are strong and vigorous and the feeding properly done, rarely follows for the first month the same line as that of nursing infants. We

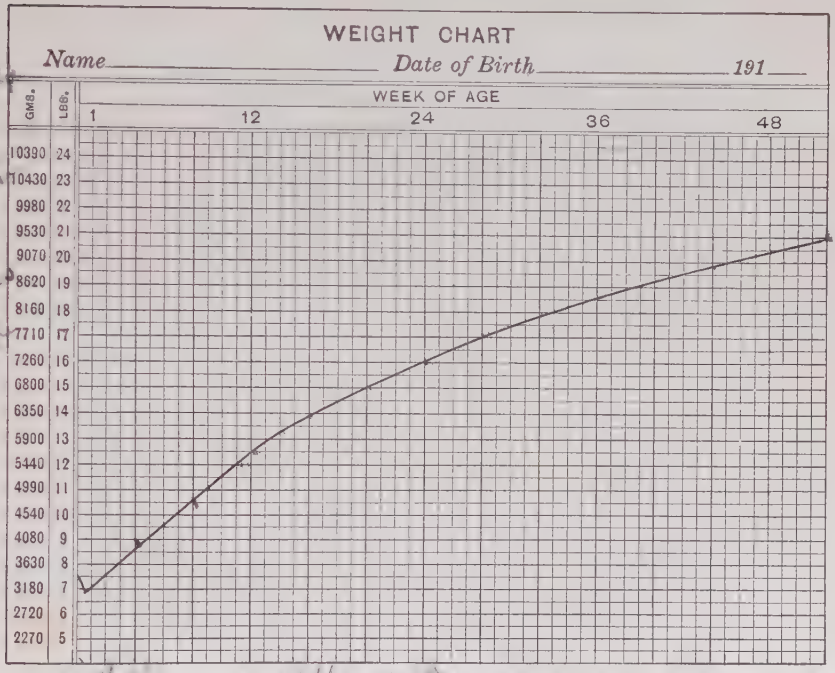


FIG. 2.—WEIGHT CHART.

usually see an initial loss which is about the same as in nursing infants, then a period of nearly stationary weight lasting from one to two weeks.

Excessive loss in weight during the first few days, from any cause whatsoever, seriously handicaps an infant during the first weeks of its life. The great importance of this has not been sufficiently appreciated.

Weight Curve of the First Year.—The curve of the accompanying chart is made up from complete weight charts of about two hundred healthy nursing infants who were thriving and weighed every week, and the incomplete charts of about five hundred other infants. There are represented in round numbers about thirty thousand observations on children under one year. The period of most rapid increase is during the

first three months. It is slowest from the sixth to the ninth month. This curve is not to be regarded as a normal line, like the normal line of the temperature chart, but as an average line. An infant who is at birth a pound above the average may keep this distance above the line for the whole year; another, weighing one pound less than the average, may be as far below it. Girls throughout the year are on the average half a pound lighter than boys. No single child exactly follows the line all the way, but it is surprising how close to it a very large number of the cases come.

In artificially-fed infants who are healthy and are properly fed, the curve does not differ essentially from that of breast-fed infants, except in the slower gain of the first month, although this difference is usually made up before the sixth month is reached.

At the end of the first year the average child weighs nearly three times as much as at birth. Perfect health during the first year is seen only in children who are gaining steadily in weight. A child may not always gain rapidly, but he should gain steadily, and if he does not, something is wrong. All the conditions surrounding the infant should be investigated, but especially the food. One should not be satisfied unless the average weekly gain during the first six months is at least four ounces. In the second six months it may be slightly less. As a rule, a child who gains regularly in weight is thriving; an exception must, however, be made in the case of some infants who are fed chiefly upon carbohydrate foods.

Weight from the Second to the Fifth Year.—Comparatively few observations have been published upon the weight during this period. From nearly two thousand personal observations, chiefly from private practice, it appears that the normal gain of a healthy child is about six pounds during the second year, about five during the third year, and about four pounds during the fourth year; the actual weights are given in the large table on page 20. During this period the gain is rarely uniform after the first year. With most children it is slowest or the weight is stationary in the summer months, while the most rapid increase is usually seen in autumn. Throughout this period girls gain in about the same ratio as boys, but remain on the average nearly one pound lighter. During almost every illness, no matter of what character, the gain in weight ceases, and usually there is a loss, the rapidity and extent of which are somewhat proportionate to the severity of the attack; but it is always much more rapid in diseases of the digestive tract than in any other form of illness.

Weight of Older Children.—The weights given in the table of children from five to fourteen years are from Bowditch. Observations were made upon children of American parentage in the public schools of Boston—

upon 4,327 boys and 3,681 girls.[1] It is to be remembered that these weights include the ordinary clothing, while those below five years are without clothing.[2] Our own observations upon children in private practice show that the average weight for the fifth and sixth years is one pound greater and from the seventh to the tenth year from two to three pounds greater than the averages of the public school children given by Bowditch.

The slowest gain is from the fifth to the eighth year, when it is about four pounds a year. From the eighth to the eleventh year it rises to about six pounds a year. Up to the eleventh year the two sexes gain in about the same ratio. From the eleventh to the thirteenth year the girls gain much more rapidly, passing the boys for the first time and maintaining this lead until the fifteenth year, when again the boys pass them.

HEIGHT

The figures showing the height at different ages are given in the table on page 20. The measurements of infants at birth, given on page 21, are taken in about equal numbers from the records of the New York Infant Asylum and the Sloane Hospital for Women. They were made upon full-term infants.

[1] W. T. Porter has published (1894) observations made upon 14,744 children of American parentage in the public schools of St. Louis. His figures show quite a variation from those of Bowditch, and are as follows:

Age.	Boys' weight.		Girls' weight.	
	Kilos.	Pounds.	Kilos.	Pounds.
6 years	19.66	43.2	18.76	41.3
7 "	21.67	47.7	20.82	45.8
8 "	23.91	52.6	22.71	50.0
9 "	26.08	57.4	25.07	55.1
10 "	28.49	62.7	27.43	60.3
11 "	31.26	68.8	29.93	65.8
12 "	33.45	73.6	33.17	73.0
13 "	35.96	79.1	38.29	84.2
14 "	40.34	88.7	43.12	94.9
15 "	47.25	103.9	46.90	103.2
16 "	52.10	114.6	50.06	110.1

[2] The average weight of the ordinary house clothing of school children, according to Bowditch, is at five years, 2.8 pounds for both sexes; at seven years, 3.5 for both sexes; at ten years, 5.7 pounds for boys and 4.5 pounds for girls; at thirteen years, 7.4 pounds for boys and 5.6 pounds for girls; at sixteen years, 9.7 pounds for boys and 8.1 pounds for girls. This must be deducted to obtain net weights.

Table showing Weight, Height, and Circumference of the Head and Chest from Birth to the Sixteenth Year.[1]

Age.	Sex.	Weight.		Height.		Chest.		Head.	
		Pounds.	Kilos.	Inches.	Cm.	Inches.	Cm.	Inches.	Cm.
Birth [2].....	**Boys.**	**7.55**	**3.43**	**20.6**	**52.5**	**13.4**	**34.2**	**13.9**	**35.5**
	Girls.	7.16	3.26	20.5	52.2	13.0	33.2	13.5	34.5
6 months [2]...	**Boys.**	**16.0**	**7.26**	**25.4**	**64.8**	**16.5**	**42.0**	**17.0**	**43.5**
	Girls.	15.5	7.03	25.0	63.6	16.1	41.0	16.6	42.2
12 months [2]..	**Boys.**	**21.0**	**9.54**	**29.0**	**73.8**	**18.0**	**45.9**	**18.0**	**45.9**
	Girls.	20.5	9.31	28.7	73.2	17.4	44.4	17.6	44.6
18 months [2]..	**Boys.**	**24.0**	**10.90**	**30.0**	**76.3**	**18.5**	**47.1**	**18.5**	**47.1**
	Girls.	23.5	10.68	29.7	75.6	18.0	45.9	18.0	45.9
2 years [2].....	**Boys.**	**27.0**	**12.27**	**32.5**	**82.8**	**19.0**	**48.4**	**18.9**	**48.2**
	Girls.	26.0	11.81	32.5	82.8	18.5	47.0	18.6	47.2
3 years [2]....	**Boys.**	**32.0**	**14.55**	**35.0**	**89.1**	**20.1**	**51.1**	**19.3**	**49.0**
	Girls.	31.0	14.09	35.0	89.1	19.8	50.5	19.0	48.4
4 years [2].....	**Boys.**	**36.0**	**16.36**	**38.0**	**96.7**	**20.7**	**52.8**	**19.7**	**50.3**
	Girls.	35.0	15.90	38.0	96.7	20.7	52.2	19.5	49.6
5 years.....	**Boys.**	**41.2**	**18.71**	**41.7**	**106.0**	**21.5**	**54.8**	**20.5**	**52.2**
	Girls.	39.8	18.06	41.4	105.3	21.0	53.5	20.2	51.3
6 years.....	**Boys.**	**45.1**	**20.48**	**44.1**	**112.0**	**23.2**	**59.1**	.....	.....
	Girls.	43.8	19.87	43.6	110.9	22.8	58.3	.....	.....
7 years.....	**Boys.**	**49.5**	**22.44**	**46.2**	**117.4**	**23.7**	**60.6**	.....	.....
	Girls.	48.0	21.78	45.9	116.7	23.3	59.5	.....	.....
8 years.....	**Boys.**	**54.5**	**24.70**	**48.2**	**122.3**	**24.4**	**62.2**	.....	.....
	Girls.	52.9	24.01	48.0	122.1	23.8	60.8	.....	.....
9 years.....	**Boys.**	**60.0**	**26.58**	**50.1**	**127.2**	**25.1**	**63.9**	.....	.....
	Girls.	57.5	26.10	49.6	126.0	24.5	62.5	.....	.....
10 years.....	**Boys.**	**66.6**	**30.22**	**52.2**	**132.6**	**25.8**	**65.6**	**21.0**	**53.5**
	Girls.	64.1	29.07	51.8	131.5	24.7	63.0	20.7	52.8
11 years.....	**Boys.**	**72.4**	**32.83**	**54.0**	**137.2**	**26.4**	**67.2**	.....	.....
	Girls.	70.3	31.87	53.8	136.6	25.8	65.8	.....	.....
12 years.....	**Boys.**	**79.8**	**36.21**	**55.8**	**141.7**	**27.0**	**68.8**	.....	.....
	Girls.	81.4	36.90	57.1	145.2	26.8	68.3	.....	.....
13 years.....	**Boys.**	**88.3**	**40.04**	**58.2**	**147.7**	**27.7**	**70.6**	.....	.....
	Girls.	91.2	41.36	58.7	149.2	28.0	71.3	.....	.....
14 years.....	**Boys.**	**99.3**	**45.03**	**61.0**	**155.1**	**28.8**	**73.3**	.....	.....
	Girls.	100.3	45.50	60.3	153.2	29.2	74.1	.....	.....
15 years.....	**Boys.**	**110.8**	**50.26**	**63.0**	**159.9**	**30.0**	**76.6**	**21.8**	**55.5**
	Girls.	108.4	49.17	61.4	159.9	30.3	76.8	21.5	54.8
16 years.....	**Boys.**	**123.7**	**56.09**	**65.6**	**166.5**	**31.2**	**79.2**	.....	.....
	Girls.	113.0	51.24	61.7	156.7	30.8	78.8	.....	.....

[1] The observations of Boas (Science, April 12, 1895) upon 4,319 children over six years old show that first born exceed children born at a later period both in height and weight.

[2] These weights are without clothes; after five years clothes are included.

Average length of 231 male infants born at term..	20.61 inches	(52.5 cm.);	
" " " 211 female " " " "	20.47 "	(52.2 ");	
" " " 442 infants.....	20.54 "	(52.35 ").	

The most rapid gain in length is in the first year. During this period the child grows on an average a little over eight inches (21 cm.). This gain is usually, but not always, proportionate to the increase in weight. During the second year the average increase is three and a half inches (9 cm.). From this time on the rate of increase is quite uniform in both sexes until the eleventh year, it being between two and three inches a year.

After the eleventh year in girls and the twelfth in boys the growth is much more rapid. In height the girls exceed the boys at the twelfth and thirteenth years for the only time in their growth.

In the figures given in the preceding table those of five years and over are taken from Bowditch, the observations being made upon the same children as those whose weights were taken. The observations from six months to four years inclusive are from original sources, and are drawn from about eight hundred cases. The height much more than the weight of children is modified by hereditary influences.

Rachitic children during infancy and early childhood are, as a rule, shorter than others. We have frequently measured such children during the third year who were six inches below the average for that age. The effect of malnutrition upon the length of the body is much less than upon the weight.

GROWTH OF THE EXTREMITIES AS COMPARED WITH THE TRUNK

At birth the trunk is relatively long and the extremities short. The middle of the body at birth, according to one hundred observations on normal infants made for us by Wilbur Ward at the Sloane Hospital, is three-quarters of an inch (2 cm.) below the center of the umbilicus. Subsequently the growth of the extremities is much more rapid than that of the trunk. Thus we have found at birth the length of the lower extremities (measuring from the anterior superior spine of the ilium to the sole of the foot) to be forty-three per cent of the length of the body; at five years, fifty-four per cent, and at sixteen years, sixty per cent. The above figures are from one hundred and fifty observations, which, although not numerous enough for exact percentages, are still sufficient to give a very good idea of the general relation of the length of the extremities to that of the body as a whole. These facts are of some assistance in the diagnosis of diseases attended by abnormalities of growth, such as rickets, cretinism, and chondrodystrophy.

THE HEAD

Circumference.—The average circumference of the head at birth in four hundred and forty-six full-term infants observed at the Sloane Hospital and New York Infant Asylum was as follows:

Average circumference of the head,	231 males.....	13.90 inches	(35.5 cm.);
	215 females.....	13.52 "	(34.5 ");
Total.....	446 infants.....	13.71 "	(35.0 ").

The occipitofrontal measurement was the one taken.

The growth of the head is most rapid during the first year, the increase being about four inches (10 cm.). It is about half an inch a month during the early months, and a fourth of an inch a month during the later months of the first year. During the second year the increase is about one inch (2.5 cm.). From the second to the fifth year the growth is slower, being only about one and a half inches (4 cm.) for the three years. After the fifth year the increase in the circumference of the head is very slow (see table).

Closure of the Sutures.—The main sutures of the cranium are not commonly ossified before the end of the sixth month, and very frequently some mobility may be detected at the end of the ninth month. Distinct separation of the cranial bones after birth is abnormal. It is most frequently seen in premature and in syphilitic infants.

Closure of the Fontanels.—The posterior fontanel is usually obliterated by the end of the second month. The anterior fontanel under normal conditions closes on an average at about the eighteenth month. The usual variations are between the fourteenth and twenty-second months. At the end of the first year the fontanel is generally about one inch in diameter. An open fontanel at the end of the second year may be considered abnormal. The closure of the fontanel is not always early in well-nourished children, nor is it always delayed in those suffering from malnutrition. In very rare cases the anterior fontanel may either be closed at birth or may close during the first few weeks of life. Closure of the fontanel by the middle of the first year is often seen in cases of arrested cerebral development. This indicates a serious condition, usually microcephalus. Closure of the fontanel in the early months of the second year may be due to the slow growth of the brain in a child suffering from general malnutrition but otherwise normal.

By far the most frequent cause of delayed closure of the fontanel is rickets, in which condition it may be open up to the end of the third year. A large fontanel is one of the striking features of cretinism, and

in untreated cases is often seen as late as the eighth year or later. In infancy an open fontanel with a rapid growth of the head should at once suggest hydrocephalus. There is an hereditary condition in which the fontanel remains open even to adult life. Two such cases in father and son were shown us by Marie in Paris. In both there was also lack of union between the two portions of the clavicle.

Shape of the Head.—The deformity which results from compression

FIG. 3.—PREMATURE OSSIFICATION OF THE SAGITTAL SUTURE. Death at six weeks.

during labor usually disappears by the end of the first month. During the first year the head often becomes flattened at the occiput in consequence of the child's lying too much upon the back. This is easily remedied by changing his position. A slight obliquity of the head may result from a habitual position during nursing or sleep. A marked degree of obliquity is sometimes congenital, but usually disappears by the third or fourth year.

The other abnormalities in the shape of the head are chiefly due to rickets and hydrocephalus, more rarely to congenital malformations of the brain. They will be considered in the chapter devoted to these topics.

Premature ossification of the sutures of the cranium occasionally gives rise to striking deformities of the head. Depending upon the sutures involved the head may be long and narrow or it may be short and high. These two types are known respectively as scaphocephaly and oxycephaly. They are referred to more fully in the chapter upon Internal Hydrocephalus. Fig. 3 shows a skull with complete obliteration of the sagittal suture. In this case there was a wide separation of the sutures at the junction of the parietal and temporal bones. Premature ossification of the *os tribasilare* at the base of the skull is largely responsible for the prognathism and peculiar formation of the cranium seen in chondrodystrophy.

THE CHEST

The figures showing the circumference of the chest at the different periods of childhood have already been given. The measurements up to and including five years are from personal observations, those from the sixth to the sixteenth are taken from Porter, and are drawn from observations on 31,371 school children. The measurement of the chest is that taken midway between full inspiration and expiration, and at the level of the nipples.

In the newly-born child the antero-posterior and the transverse diameters of the chest are nearly the same. As age advances, the transverse diameter increases very much more rapidly, so that the outline of the chest gradually assumes an elliptical shape, which it maintains during childhood.

At birth, the circumference of the chest is about one-half inch less than that of the head, but throughout infancy the two measurements are nearly the same. It is not until the third year that the average circumference of the chest exceeds that of the head. The chest measurement in infants is always much modified by the amount of fat; but, after making due allowance for this, a large chest always indicates a robust child and a small chest a delicate one. If at any age the circumference of the child's chest is found to be below the average, means should be taken, by gymnastics and otherwise, to develop it.

In infants deformities of the thorax result chiefly from rickets, sometimes from empyema, emphysema, and cardiac disease; in older children, from lateral curvature of the spine, or from Pott's disease. A peculiar deformity, usually congenital, but sometimes rachitic, is the funnel-shaped chest, the *Trichter brust* of the Germans. It consists in a deep pitlike central depression at the lower end of the sternum. It is usually permanent.

THE ABDOMEN

Throughout infancy the circumference of the abdomen is, as a rule, about the same as that of the chest. At the end of the second year the measurements of the head, chest, and abdomen are very often identical; after this time the chest measurement increases much more rapidly than the other two. Marked enlargement of the abdomen is seen in many varieties of chronic intestinal disorders. The tympanites that often accompanies rickets is a frequent cause of enlargement.

MUSCULAR DEVELOPMENT

The first voluntary movements are usually in the fourth month, when the infant deliberately attempts to grasp some object placed before him. During the fourth month, as a rule, the head can be held erect when the trunk is supported. In many infants this is possible in the early part of the third month. At seven or eight months a healthy child is usually able to sit erect and support the trunk for several minutes.

In the ninth or tenth month are usually seen the first attempts to bear the weight upon the feet. At eleven or twelve months a child usually stands with slight assistance. The first attempts at walking are commonly seen in the twelfth or thirteenth month. The average age at which children walk freely alone has been, in our experience, the fourteenth or fifteenth month. Quite wide variations are seen in healthy children. Very much depends upon the surroundings. We have known infants to walk at ten months and many others not until seventeen or eighteen months, although showing no evidences of disease, and although their development had not been retarded by previous illness. A very marked difference is seen in different families with respect to the time of walking.

The physician is often consulted because of backward muscular development, most frequently because the child is late in walking. General malnutrition, or any other severe or prolonged illness, may postpone for several months this or any of the other functions mentioned. When there is no such explanation of the backwardness, a child who does not hold up his head, sit alone, or make efforts to stand or walk at the proper time, should be submitted to a careful examination for mental deficiency or cerebral or spinal paralysis, but especially for rickets, which is the most frequent explanation of the symptoms.

Contrivances for teaching infants to walk are unnecessary, and their effect may even be injurious. An infant should be allowed the greatest possible freedom in the use of his limbs. He should not be restrained

from walking when inclined to do so, nor continually urged to walk when no voluntary attempts are made. Nothing short of mechanical restraint will prevent a healthy child from walking or standing when he is strong enough to do so.

DEVELOPMENT OF THE SPECIAL SENSES

Sight.—The newly-born infant avoids the light. The pupils contract in a light room, and if a bright light is brought before the eyes they close. During the first few weeks the infant indicates by every sign that excessive light is unpleasant. As early as the sixth day the eyes will sometimes follow a light in the room, and the child may even turn the head for this purpose. The muscles of the eyes of the newly-born infant act irregularly and not in harmony. Coördinate action for general purposes is not established until about the end of the third month. Even after this time incoördinate action is occasionally seen. The eyelids also move irregularly, and are often partly separated during sleep. The cornea is but slightly sensitive during the first weeks. In Preyer's child it was not until the third month that the lids closed when the water in the bath touched the lashes or the cornea. The recognition of objects seen is usually evident in the sixth month.

It is important that the room in which the newly-born child is placed should be darkened, and that for the first few weeks the eyes should be protected against strong light.

Hearing.—For the first twenty-four hours after birth infants are deaf. This deafness sometimes persists for several days. It is believed to be due to absence of air from the middle ear and to swelling of the mucous membrane which lines the tympanum. With the movements of respiration, air gradually finds its way into the middle ear, and the swelling subsides during the first few days. After this the hearing gradually improves, and during the early months of life it is very acute. The child starts at the slamming of a door, and even moderately loud noises will waken him from sleep. By the end of the second month he will sometimes turn his head in the direction from which the sound comes, and by the end of the third month this will usually be done. Demme found, in observations upon one hundred and fifty infants, that voices were recognized on an average at three and a half months.

Not only are the ears unusually sensitive to sound in infancy, but the impression produced upon the brain is often marked—very loud sounds causing great fright.

Touch.—Tactile sensibility is present at birth, but is not highly developed except in the lips and tongue, where it is very acute for the obvi-

ous necessity of sucking. After the third month it is fairly acute over the surface of the body generally. Two especially sensitive areas, according to Preyer, are the forehead and external auditory meatus.

Sensibility to painful impressions is present in early infancy, but very dull as compared with later childhood.

Temperature is also distinguished. This recognition is especially acute in the tongue. A young infant is often seen to refuse to take the bottle because the milk is only a few degrees too cold or too warm.

The localization of sensory impressions comes later, probably not much before the middle of the sixth month, and is very imperfect throughout the first year.

Taste.—This is highly developed, even from birth. According to the experiments of Kussmaul, the ability to distinguish sweet, sour and bitter, exists in the newly-born child—sweet exciting sucking movements, and bitter, grimaces. A young infant detects with surprising accuracy the slightest variation in the taste of his food, and the smallest difference is often enough to cause him to refuse the bottle altogether. Sweet substances are always easily administered, and in combination with syrups even very bitter substances can be given; but to aromatic powders and elixirs he usually objects.

Smell.—Observations upon the sense of smell in newly-born infants are few and not altogether conclusive. Kroner's experiments appear to show that smell is present in the newly born. It has been noted to be especially acute in infants born blind. The sense of smell is developed much later than the other senses. Detection of fine differences in odors is not acquired until quite late in childhood.

SPEECH

There is a very wide variation in children with reference to the time of development of the function of speech. Girls, as a rule, talk from two to four months earlier than boys. Towards the end of the first year the average child begins with the words "papa," "mamma." By the end of the second year he is able to put words together in short sentences of two or three words. Progress in speech from this time is very rapid, each month showing great improvement. Names of persons are commonly first acquired, then the names of objects. Next to this the verbs are learned, and then adverbs and adjectives. Conjunctions, prepositions, and articles follow in order, and last of all the personal pronouns.

If a child of two years makes no attempt to speak, some mental defect may usually be inferred or that the child is a deaf mute.

DENTITION

The teeth are enclosed at birth in dental sacs which are situated in the gums. Superficially they are covered by the submucous connective tissue and the mucous membrane; the dental sacs rest in depressions in the alveolar process of the jaw. The tooth grows in length mainly as the result of the calcification of its roots, and being thus fixed below, it pushes upward towards the mucous membrane. This growth undoubtedly goes on steadily from birth until the tooth pierces the gum.

The deciduous or milk teeth are twenty in number. The time at which they appear is subject to considerable variation even under normal conditions. The following is the order and the average time of appearance of the different teeth:

(1) Two lower central incisors	6 to 9 months.
(2) Four upper incisors	8 " 12 "
(3) Two lower lateral incisors and four anterior molars	12 " 15 "
(4) Four canines	18 " 24 "
(5) Four posterior molars	24 " 30 "
At 1 year a child should have	6 teeth.
At 1½ years " " "	12 "
At 2 " " " "	16 "
At 2½ " " " "	20 "

Quite wide variations on both sides of the average are common, and are not always easy of explanation. In many cases it seems to be a family idiosyncrasy, since in the different members of a family the teeth are apt to appear at about the same time. The order in which the teeth appear is much more regular than the time of their appearance. Slight variations are exceedingly common, but marked irregularities in the order of the appearance of the teeth are the rule in idiotic children or those suffering from slighter mental defects.

The teeth may pierce the gum without any local manifestations. Very frequently, however, just before a tooth comes through there is noticed a moderate swelling and redness of the mucous membrane of the gum overlying it, and to a slight degree this may affect the general mucous membrane of the mouth. This condition may be accompanied by a little fretfulness and increased salivation, or both of these may be entirely wanting. These symptoms usually disappear when the tooth has pierced the gum. The symptoms of difficult dentition will be discussed in connection with Diseases of the Mouth.

Infants may be born with teeth. We know of one family in which this occurred in three members of three successive generations. It is, however, rare. It is almost invariably one of the lower central incisors

that is present. In case this interferes with nursing, or if it is very loosely attached to the gum, it should be extracted, but under other circumstances it should be allowed to remain, since, if it is removed, a second tooth is not likely to appear in its place in the first set. It is not at all uncommon for the first teeth to appear in the fourth month. Such teeth, in our experience, do not usually differ in character from those appearing later, unless they are in children who are syphilitic. Syphilitic children are rather prone to early dentition, and under such circumstances rapid and early decay is likely to take place. Nursing infants are, as a rule, a little earlier in their dentition than those artificially fed.

Delayed dentition is usually due to rickets. However, in many healthy infants no teeth appear before the tenth month; and we have occasionally seen the first ones at thirteen months in those who seemed perfectly healthy and showed no other evidence of rickets. On the other hand, it is by no means invariable that dentition is late in rachitic children. The latest dentition is seen in cases of cretinism. In such children it is not rare for the first teeth to appear as late as eighteen months or two years. As a rule, dentition and ossification of the bones of the head go on in a corresponding manner; where one is early the other is likely to be rapid, and conversely. Great irregularities in dentition are common in children with defective cerebral development.

Provided an infant is well nourished and thrives properly for the first six or eight months, the eruption of the teeth is likely to go on steadily after this time, even though the child may later have chronic indigestion or suffer from extreme malnutrition from any cause except rickets. If, however, the symptoms of malnutrition date from birth, dentition is almost invariably delayed. It is often a matter of very great surprise to see children who are markedly emaciated as a result of chronic indigestion or ileocolitis and yet go on cutting their teeth regularly. We once had under our care a delicate infant of sixteen months, whose body length was twenty-eight inches and whose weight was less than nineteen pounds—almost exactly what they were eight months previously—and yet he had thirteen teeth.

Eruption of the Permanent Teeth.—The first to appear are the first molars, which usually come in the sixth year, and hence the name six year old molars, which is applied to them. These appear posterior to the second molars of the first set.

The incisors and canines replace the corresponding teeth of the first set. The eight bicuspids take the place of the eight molars of the first set. The molars of the permanent set appear back of the bicuspids, room being made for them by the growth of the jaw. As they grow and push upward the permanent teeth cause atrophy of the roots of the first teeth,

and gradually cut off their blood supply, so that they loosen and fall out.

The following table from Forchheimer gives the average time of the appearance of the second teeth:

First molars		6 years.
Incisors	7 to 8	"
Bicuspids	9 " 10	"
Canines	12 " 14	"
Second molars	12 " 15	"
Third molars	17 " 25	"

The place of dentition as an etiological factor in the diseases of infancy will be considered in the chapter on Difficult Dentition.

CHAPTER III

PECULIARITIES OF DISEASE IN CHILDREN

In many particulars disease in children differs from that of later life. These differences relate to etiology, pathology, symptomatology, diagnosis, and prognosis. The greatest contrast to adult life is presented by infancy and early childhood. After seven years, children in their diseases resemble adults more than they do infants.

ETIOLOGY

1. **Inheritance** is an important factor. The disease most frequently transmitted directly is syphilis. Occasionally tuberculosis and other infectious diseases have been conveyed directly from the mother to the child. In cases where no distinct disease is transmitted, children may inherit from parents constitutional weaknesses or tendencies, which may manifest themselves in infancy, or in some cases not until later childhood. Under this head we may place the influence of alcoholism, lead poisoning, rheumatism, gout, epilepsy, and insanity.

2. **Malformations** must be considered, particularly in the first two years of life. The most important of these, from a medical standpoint, are those of the heart, brain, stomach and intestines, and kidney. The various malformations of the mouth, nose, bladder, rectum, and genital organs belong more particularly to the domain of surgery.

3. **The Diseases or Accidents Connected with Birth.**—Some of these are distinctly traumatic, like the meningeal hemorrhages. A very large class are the infectious processes in the newly born. Infection usually

takes place through the umbilical wound, more rarely through the skin or mucous membranes. This class includes pyemia, with its varied lesions in the brain, lungs, and serous membranes, erysipelas, ophthalmia, and tetanus. In the class of infectious diseases may also be included many of the varieties of pulmonary and intestinal diseases in the newly born, and probably also some of the hemorrhagic affections.

4. **Conditions Interfering with Proper Growth and Development.**—These are among the largest etiological factors in the diseases of infancy. They are improper food or feeding, unhygienic surroundings, and neglect. These may cause specific diseases, like rickets or scurvy, or may lead to a condition of general malnutrition or marasmus. In this way they become most important predisposing factors, in infancy, to the acute diseases of the gastro-enteric tract, and later in childhood, to functional nervous diseases.

5. **Infection.**—This has already been mentioned as an important factor in diseases of the newly born. The number of diseases in later life directly traceable to this is very large. Under this head should be included not only the well-known classes of infectious and contagious diseases, but also a very large number of varieties of infection which as yet have not been differentiated, and the nature of which is but imperfectly understood.

SYMPTOMATOLOGY AND DIAGNOSIS

In older children the symptoms of disease are very much the same as in adults, and similar methods of examination may be employed. What is really peculiar to children belongs especially to the first three years of life, before speech has developed. During this period the chief and almost the sole reliance of the physician must be upon the objective signs of the disease. It is not so much that diseases in early life are peculiar, as that the patients themselves are peculiar.

Two fundamental facts are always to be kept in mind: First, that the common pathological processes are comparatively few, being chiefly of the gastro-enteric tract, the lungs, and the brain, but that the variations in clinical types are almost endless; the second is, that in infants, on account of the susceptibility of the nervous system, functional derangements are often accompanied by very grave symptoms, and may even prove fatal in twelve or twenty-four hours, or there may be speedy and complete recovery after very alarming symptoms. In many of these cases the symptoms are so indefinite that an exact diagnosis is impossible during life, and even the autopsy may throw but little light upon them.

At the bedside it is of great assistance to the physician if he can keep in mind the most frequent forms of acute disease that are likely to be met with. In the first group, including those which are very common, may be placed acute indigestion and ileocolitis, bronchitis, pneumonia, pharyngitis, tonsillitis, and otitis media; in the second group, which are less frequent, are placed the more common acute infectious diseases; in the third group, including the rarer forms of acute disease —meningitis, tuberculosis, rheumatism, and diseases of the kidneys. In all circumstances, the season, and the nature of the prevailing epidemic, if one exists, are to be considered.

In the examination of a sick infant quite a different method is to be followed from that pursued with adults. Much information is to be gained from a history carefully taken from an intelligent mother or nurse, and much more from a close observation of the child, whether asleep or awake, quiet or crying.

The History.—In view of the fact that but little information can be had from the patient, none at all in most cases, it is important to obtain from the mother or nurse as full and complete information as possible. A good history carefully obtained, puts the physician in possession of a fund of information about the patient which is not only of the greatest value in arriving at a diagnosis in the illness for which he is consulted, but is exceedingly helpful in the future management of the child. He may thus know the individual peculiarities and special pathological tendencies. The laity attach great importance, and justly so, to advice from the physician who "knows the child's constitution." Such a history should be taken at the first opportunity after the physician is placed in charge of a child, and with note-book in hand, or half its value will be lost.

Family History.—This should begin with the parents, going farther back, if possible, in many cases of hereditary disease. One must know regarding tuberculosis, syphilis, rheumatism, or alcoholism, the general vigor of constitution and physical condition of both father and mother. Health during pregnancy, and previous miscarriages, if any, are important facts in the mother's history. One should know the number of other children living and their general health, the number dead and from what causes. A knowledge of the surroundings in which the child has lived may be necessary to appreciate the chances of exposure to tuberculosis, malaria, and many other forms of infection.

Patient's Previous History.—This should begin with birth. One should inquire whether the child was premature or born at term, regarding the character of the labor, whether natural or instrumental, tedious or complicated, the condition and vigor of the child at birth, primary respirations, early convulsions, and the nutrition during the early days.

Next the methods of feeding should be taken up—how long entirely and how long partly breast fed, the date of weaning and the form of artificial feeding then employed. If the patient is an infant, and the problem presented is one of its nutrition, all the reliable data relating to the feeding should be obtained, even to the minutest detail. This may be wearisome and consume time, but in no other way can one appreciate the conditions present. The best idea of the child's growth and development may be obtained from a weight record if one has been kept. If not available, one must depend upon general statements as to how the child thrived at different periods. The date of the appearance of the first teeth and the time and the order in which the teeth came, are significant. The general muscular development may be best determined by learning when the child could first hold the head erect, sit alone upon the floor, bear the weight upon the feet, creep or walk alone; the mental development, by learning as to early recognition of mother or nurse, knowing the bottle, understanding the meaning of words, speaking in words or sentences. The muscular and mental development of a normal child during the first two years is a subject with which the physician should be familiar if he would detect early those differences, often slight at this age, in children whose development is backward owing to cerebral lesions.

All previous attacks of acute illness of whatever character should be noted, particularly the infectious diseases—measles, scarlet fever, diphtheria, pertussis, and influenza—with dates and details as to duration, severity, and complications. One should learn whether the child is especially prone to disorders of digestion or those of the respiratory system. Under the former head are included early difficulties in feeding, acute attacks of indigestion, diarrhea, or dysentery, also chronic disturbances of the stomach or bowels; under the latter head, frequent catarrhal colds, earache or otitis, catarrhal croup, bronchitis, pneumonia, or pleurisy. Other points to be investigated relate to attacks of tonsillitis, operations for the removal of hypertrophied tonsils or adenoids, and previous disorders of the nervous system. In infants, particularly important are extreme restlessness, insomnia, convulsions, or attacks of night terrors; in those who are older, hysterical manifestations, epilepsy, or chorea. Finally, one should know the date of successful vaccination. Inquiry should also be made concerning any recent exposure to infection in the community, school, or home.

Present Illness.—One should first note the chief complaints as stated by mother or nurse. It is important to obtain as definite statements as possible as to the time when the child was quite well, and whether the onset of the illness was abrupt or gradual, and with what particular symptoms. In all digestive disorders one should know exactly concern-

ing the child's food at the time of the onset, its quantity, character, and preparation; also any recent change in diet, the presence or absence of vomiting, and the condition of the bowels, whether loose or constipated, the frequency and character of the stools. General questions as to whether the bowels are regular or the stools normal are of no value, since the informant often is not capable of judging correctly.

Nervous symptoms, like the others, should be elicited in response to direct questions regarding sleep, restlessness, moaning, crying out, or other evidences of pain, excitement, delirium, or convulsions, or unnatural drowsiness. In any acute illness other important symptoms are fever, sweating, dyspnea, cough, hoarseness, nasal discharge, and the amount and character of the urine.

The Examination.—With infants, quite a different method should be followed from that pursued with adults. It may well begin with:

General Inspection.—What is learned in this way will depend almost entirely upon the acuteness of observation of the physician, but much that is of value can be ascertained before the clothing is removed for the physical examination by simply watching the patient, whether asleep or awake, for several minutes. In acute disease, the following points should be noted especially:

1. Nutrition and general development: whether the child is well nourished or the features pinched and wasted.

2. The facial expression: whether it is bright and intelligent or dull and stupid, peaceful or anxious, quiet or disturbed, and whether the features are contracted from time to time, as if from pain.

3. The character of the respiration: whether it is rapid or slow, easy or difficult; whether there is nasal obstruction, as indicated by snoring and mouth-breathing, suggesting in infants acute rhinitis, syphilis, or retropharyngeal abscess; in older children, diphtheria, scarlet fever, or adenoids. Marked dyspnea is usually accompanied by active dilatation of the alae nasi.

4. The posture: whether the child lies upon the back, side, or face; whether the head is drawn back with general flexion of the extremities as in meningitis.

5. The nervous condition: whether the child is restless, excitable, or drowsy and apathetic; if asleep, the nature of the sleep should be observed.

6. The color of the skin of the face: whether pale or cyanotic; and the lips, whether fissured or excoriated.

7. The amount of prostration: a practiced eye can usually tell with older children whether the condition is grave or not, but infants not infrequently deceive even the most experienced observer.

8. The cry: in conditions of restlessness or irritability, much infor-

mation may be obtained from its character. It is important, but not always easy, to determine whether a child cries from fright, as at the approach of a stranger, from nervousness or bad training, from general irritability which may come from any acute disease, or from actual pain. The cry of fright is usually evident, because it comes with the physician's approach and ceases when he goes away. Children of highly neurotic parents and those who have been much indulged and badly trained will often cry when anything out of the usual routine occurs. The cry of pain may be very distinctive; it may be sharp and acute and accompanied by some attempt at localization, as when a child puts his hand to an inflamed part, but in infancy the pain of acute inflammation is often indicated only by general restlessness and irritability. This is frequently true of acute otitis. The cry of pain is usually accompanied by contraction of the features and other evidences of distress.

The cry of some diseases is quite characteristic, as the short, catchy cry of acute pneumonia or bronchitis; the hoarse cry of laryngitis, whether catarrhal, membranous, or syphilitic; the feeble whine of extreme exhaustion or marasmus; the moaning cry of intestinal disease; and the sharp cry of a child with scurvy whenever its bed or body is touched.

Measurements.—These, though of greatest value in chronic diseases, particularly disturbances of nutrition, may be of assistance also in acute conditions. The important measurements are the circumference of the head, chest, and body length. The circumference of the abdomen is at times important, but varies so much with the degree of distention that it is not significant as to the general development. The measurements and weight furnish reliable data which are not only of assistance in the diagnosis of existing disease, but if recorded are useful for future comparison.

In taking the circumference of the head the largest measurement (over the occipital and frontal eminences) is preferable. The measurement of the chest is usually taken over the nipples. The body length of infants is best taken with a tape as the child lies upon his back upon a table or a firm bed. For older children a special measuring stick is convenient.

To estimate properly the significance of measurements they should be compared with the normal averages and with each other. It should be remembered that the head is normally larger than the chest until near the end of the second year; after this time, with a normal development, the chest should be larger. Any great disproportion between the size of the head and chest is suggestive of disease. The large head and the small chest belong especially to rickets. The measurements form important means of recognizing early such abnormalities as cretinism and

chondrodystrophy, the variations often being marked before the other symptoms are prominent. One who forms the habit of taking regular measurements soon appreciates the variations from the normal, and gains great assistance from these data. Such a record made from year to year in children whose development is in any way abnormal is of great value in indicating what should be done in the way of exercise to correct faulty conditions.

Vital Signs.—Pulse, Respiration, and Temperature.—The significance of these signs is not to be measured by adult standards, since the susceptible nervous system of infants and very young children greatly exaggerates their reaction to all forms of acute infection.

The rate, regularity, quality, and tension of the pulse should be noted. In young children, the rate of the pulse is of less importance than its force and quality. A slow, irregular pulse is always significant, and should suggest meningitis or brain tumor; a slight irregularity of the pulse during sleep has no special significance. The pulse rate is much increased from slight disturbances; the approach of a stranger or the examination by the physician may cause it to rise 20 or 30 beats. In acute disease, a pulse rate of 150 is common, and 170 or 180 is often seen where other symptoms are not particularly severe.

The rate, depth, and rhythm of respiration should be noted. The last often cannot be determined except by attentively watching the child for several minutes. In premature and very young infants a rather marked irregularity may be seen, often approaching the Cheyne-Stokes type. It is not to be taken as indicating a cerebral lesion, but seems rather to be due to the fact that the respiratory center is not yet fully able to control the movements. Respiration of this type is seen only during the first weeks of life. Irregularity of rhythm at other times should suggest cerebral disease, usually meningitis. The respiration rate is proportionately greater in infants than in adults. In acute diseases of the lungs it not infrequently rises to 70 or 80, and occasionally it may be over 100 a minute. The rate is generally in proportion to the extent of the pulmonary lesion.

The temperature of infants and very young children should be taken in the rectum, since groin or axillary temperatures are untrustworthy and those in the mouth difficult to obtain. Immediately after birth the temperature of the child is about the same as that of the mother, or a little higher. It falls from 1° to 3° F. in the course of the first few hours. Soon it again rises to 98.5° or 99° F.

From a large number of personal observations upon healthy infants, we have found that the rectal temperature under normal conditions varies between 98° and 99.5° F.; occasionally the range may be as wide as 97.5° to 100.5° F. in apparently perfect health. The heat-regulating

center in the brain acts only imperfectly in the young infant, and slight causes are enough to disturb the temperature.

The temperature in infants is always higher than from corresponding causes in adults. Moreover, very high temperatures may be met with in cases not serious, and not infrequently when no explanation can be found even after thorough examination. In such cases the temperature seldom remains at a high point for more than a few hours. It is a continuous high temperature rather than a single rise which is significant of disease in infancy. Nothing is more perplexing to the young practitioner than the frequency with which a high temperature is seen in infants in cases of comparatively mild illness.

It is common in chronic wasting diseases, in delicate infants and in those prematurely born, to find the temperature one or two degrees below the normal; 95° and 96° F. are of almost daily occurrence in hospitals, and much lower ones are not rare. Daily observations should be made with the thermometer in such conditions, just as in fever.

Puzzling and apparently alarming temperatures are seen in infants as a result of the application of artificial heat. In one of our patients, an infant two days old, a temperature of 107° F. was caused by the close proximity of two large hot-water bags placed in the baby's basket. The younger and feebler the child the more readily are such temperatures produced.

Muscular and Mental Development.—The general muscular development is determined by seeing how well the children can hold up the head, sit alone, stand, or walk; the mental development in young infants, by the intelligence of expression, the manner in which they respond to stimuli, the recognition of objects, fright at strangers, etc.; later in the first year, by the use of their hands, their understanding of speech, and their ability to pronounce words.

Local Examination.—For the purpose of making a complete routine examination of an infant the entire clothing, with the exception of the napkin, should be removed, and the infant placed preferably upon the nurse's lap upon a blanket. With older children the clothing may be removed and the body examined, one part at a time, but with all children it is essential that the examination be complete. A warm room is indispensable, and a table covered with a blanket in many respects better than the nurse's lap, although the latter has usually to be employed. The local examination should be deliberate, the physician should proceed cautiously, winning the child by gradual approaches, and avoiding excitement, force, or anything which may cause pain.

Skin.—The skin should first be inspected for eruptions, and it is important that the entire eruption be examined in order that the distribution as well as the character of the lesion may be seen. Marked wrin-

kling or loss of elasticity of the skin is one of the best indications of loss in weight. Bedsores are more frequently seen over the occiput than over the sacrum. Any large veins should be noted.

External glands should now be examined, especially the cervical, axillary, inguinal, and epitrochlear. The cause of a marked enlargement of any of these groups should be sought in the skin or mucous membranes with which they are connected. Marked swelling of the cervical glands may indicate diphtheria, scarlet fever, or a simple acute inflammation dependent upon a rhinopharyngitis. Enlargement of the epitrochlear glands is especially significant of syphilis. General enlargement of all the glands to a slight degree is seen in most cases of malnutrition and in many acute infectious diseases.

Head.—One should first note whether the sutures are ossified, unnaturally open or separated; also whether the fontanel is closed, or, if open, whether it is depressed or bulging. Prominences of the frontal or parietal regions when symmetrical are indicative of rickets. Irregular prominences of a smaller size, when present, are usually syphilitic. In the newly born, a tumor on the head, if in the median line, may indicate an encephalocele; if limited to either the parietal or occipital bone it is usually a cephalhematoma.

Eyes.—The condition of the conjunctivae and lids should be noted, also the presence of ptosis, strabismus, or other paralysis, but particularly the condition of the pupils, whether contracted or dilated, and the nature of their response to light. One should look also for the presence of corneal ulcers or of interstitial keratitis frequently seen in late hereditary syphilis.

Ears.—The presence of a discharge may be recognized by sight or by the odor. In any acute febrile condition one should look for tenderness or swelling over the ear or mastoid. The ears should invariably be examined otoscopically in all forms of febrile disturbance whose cause is doubtful and from time to time in pneumonia, scarlet fever, measles, diphtheria and other diseases involving the mouth and rhinopharynx.

Nose.—The presence of any nasal discharge should be noted and its character determined. An abundant discharge tinged with blood, in young infants, should suggest syphilis; in older children, diphtheria; a chronic discharge, adenoid growths; a purulent discharge of one side, a foreign body.

Mouth.—The appearance of the mucous membrane of the mouth and gums as well as the teeth may often be ascertained by watching the child while he is crying. It should be noted whether the tongue is dry or moist, clean or coated; whether thrush is present or any other form of stomatitis. If the gums are congested, swollen, or hemorrhagic, they should suggest scurvy. The number, position, and character of

the teeth are important. The general color of the mucous membrane may be significant in cases of cyanosis in congenital cardiac disease, and extreme pallor of the mucous membrane in anemia. On the mucous membrane of the hard palate may often be found the first local evidence of scarlet fever in the form of a minute punctate eruption, and on that portion of the cheeks opposite the molar teeth should be sought Koplik's sign, the earliest reliable symptom of measles.

Throat.—A careful examination of the pharynx and tonsils should never be omitted in any acute illness, no matter what other symptoms may be present. Not only tonsillitis, but often diphtheria, is overlooked from a failure to observe this as an invariable rule. A good light is essential, and one must train himself to take in all the appearances at a single glance. Marked general redness of the pharynx may be associated with scarlet fever, influenza, or simple acute pharyngitis. If other symptoms are present pointing to chronic nasal obstruction or to a catarrhal process of the rhinopharynx, a digital examination should be made to determine the presence of adenoids. Dyspnea with mouth-breathing when associated with difficulty in swallowing should, in an infant, always suggest retropharyngeal abscess. The examination of the mouth and throat may wisely be made the last step, since it usually disturbs a child so as to embarrass further investigation.

Neck.—One should consider the position in which the head is held and the amount of rigidity of the cervical muscles. Opisthotonus may be associated with meningitis or old cerebral palsy. A marked rigidity may indicate cervical Pott's disease or, if accompanied by torticollis, may be of rheumatic origin.

Chest.—In young children particular importance should be attached to the shape of the chest. Symmetrical deformities are usually due to rickets. Contraction of one side only is most frequently the result of an old empyema or an extensive interstitial pneumonia. Bulging of the precordial region is frequent in cardiac disease. One should notice also the recession of the soft parts—intercostal spaces, the suprasternal notch, or the epigastrium; the amount of this is usually the best means of judging the severity of obstructive dyspnea. Details regarding the physical examination of the lungs are discussed in the introductory chapter to Pulmonary Diseases.

Heart.—It should be remembered that under two years old loud murmurs are almost invariably of congenital origin, that soft murmurs at the base are very frequently functional, and that acquired cardiac disease is rare until after three years. For further details in the examination the reader is referred to the chapters upon Diseases of the Heart.

Abdomen.—There should be noted the presence or absence of tym-

panites or abdominal tenderness, whether general or localized, and the existence of retraction of the abdominal walls as in meningitis. The size and position of the liver and spleen are best determined by palpation. The lower border of the liver is usually slightly below the free border of the ribs. If the spleen can be easily felt below the ribs, it is, as a rule, enlarged. If it can not be felt in a satisfactory examination, it is not sufficiently enlarged to be of any diagnostic importance. In acute disease a large spleen suggests malaria, typhoid, or tuberculosis; in chronic disease, rickets, malaria, syphilis, leukemia, or anemia.

Spine.—The most frequent spinal curves seen in infancy are those due to muscular weakness. These disappear by placing the child in a prone position. Rachitic curvatures are of the same general character, but as they have usually lasted a longer time the spine is less flexible and the curvatures may not entirely disappear by change of posture. An angular deformity or even marked rigidity of the spine should suggest Pott's disease.

Extremities.—The color of the skin and the character of the peripheral circulation should be noted especially in pneumonia, diphtheria, and other diseases attended by weakened heart. Clubbing of the fingers or toes may be due to congenital heart disease or to chronic disease of the lungs. Desquamation of the palms or soles may indicate hereditary syphilis or scarlet fever, even though no other evidence may be present. The finger-nails may give valuable information in hereditary syphilis. In examining the extremities one should note especially the presence of tenderness, flaccidity, or rigidity of muscles, whether the limbs are wasted or plump, and the degree of muscular power; also any abnormal swelling on the shaft or near the extremities of the bones, and, finally, the function of the joints. A general relaxation of the ligaments is common in rickets, in paralytic conditions, and in the Mongolian type of mental deficiency. Flabbiness of the muscles belongs to malnutrition and rickets; rigidity, if chronic, is usually indicative of cerebral palsy. Weakness of special groups, with atrophy and flaccid muscles, suggests poliomyelitis. Acute tenderness of the legs in infants should suggest scurvy; in older children, osteomyelitis or rheumatism. Rachitic deformities are almost invariably bilateral. Tuberculous bone disease affects the epiphyses, while syphilis usually involves the shafts, the only exception to this being the epiphyseal separation which may occur in the first months of life.

The reflexes may be somewhat difficult to obtain in infants and when exaggerated may indicate cerebral palsy, meningitis, or, as in tetany, only an extreme irritability of the nervous centers without organic disease. The plantar reflex of Babinski has little significance in infants, and in older children it is present in many conditions. Kernig's sign is a form

of muscular spasm almost invariably present in meningitis, but often seen in other diseases.

Genital Organs.—Male children should be examined to determine the presence of phimosis or of undescended testicles. Hydrocele of the cord is a frequent condition, and may be mistaken for hernia. Both inguinal and umbilical herniae are very common. In female children it should be remembered that preputial adhesions may be considered normal, and are seldom the cause of the nervous symptoms attributed to them. Every vaginal discharge is significant, and if purulent should be examined bacteriologically. The great frequency of gonococcus infections is not appreciated, and they may be found when least expected.

The examination is not complete without the inspection of the *stools,* the chemical and microscopical examination of the *urine,* and an examination of the *blood.* All are more fully considered in special chapters.

PATHOLOGY

The pathological processes which result from intra-uterine disease and those which are connected with delivery are peculiar to early life. They have already been referred to in the section on etiology. Of the processes of early life which begin after birth, the first in frequency are those of the mucous membranes resulting from the various forms of irritation and infection. In summer, it is the stomach and intestines which suffer chiefly; in winter, the respiratory tract.

The serous membranes are rarely the seat of primary inflammation. The pleura is seldom the seat of primary disease, but is very often involved secondarily to disease of the lung itself. Affections of the pericardium and peritoneum are quite rare. Meningitis is fairly common, especially the tuberculous form.

Diseases of the lymph nodes (lymphatic glands) play an important part in connection with the acute diseases of the mucous membranes, with many affections of the skin, and even of the viscera. Acute infection tends to excite suppurative inflammation, particularly in infants; a less active process leads to chronic hyperplasia in the mesenteric, mediastinal, and cervical glands, in the tonsils, adenoid tissue of the pharynx, etc. The lymph nodes in the neck and thorax are frequently the earliest seat of tuberculous deposits, and in very many cases they are the foci from which secondary infection of the lungs, brain, or joints may occur.

Of the visceral inflammations those of the lungs are the most common, it being rare to find the lungs normal at autopsy after any acute infectious disease which has lasted a week. Up to the third or fourth year of life the heart usually escapes. In older children it may be involved, as in adults, in the rheumatic diseases. The liver and spleen

are not often the seat of organic disease in early life, nor is serious disease of the kidney likely to be met with except in connection with scarlet fever. Organic disease of the brain itself is rare, as is also organic disease of the spinal cord, with the exception of poliomyelitis. Chronic diseases of the different viscera are decidedly rare, except when resulting from acute processes. Diseases of the bones and joints are common, and of extreme importance. They are usually of tuberculous, less frequently

---

The following table gives in a general way a very good idea of the relative frequency of diseases of the different organs in infancy. It is based upon seven hundred and twenty-six consecutive autopsies in the New York Infant Asylum, extending over a period of eight years during our connection with that institution. Of these children seventy-two per cent were under one year, twenty-five per cent between one and two years, and only three per cent were over two years. The institution did not receive infants under one month, hence the absence of lesions peculiar to the newly-born:

Table showing principal lesions in seven hundred and twenty-six consecutive autopsies in the New York Infant Asylum.

Lungs:		
Pneumonia—Primary		139
	Complicating other acute infectious diseases	112
	Complicating other conditions	71
	Noted to be present in	322
Pleurisy—	No case uncomplicated with disease of lungs.	
	Empyema	5
	Serous pleurisy	1
	Dry pleurisy in nearly all the severe cases of pneumonia.	
Atelectasis (congenital)		6
Pulmonary abscess (always with pneumonia)		7
Pulmonary gangrene (always with pneumonia)		2
Pulmonary tuberculosis		56
Mouth:		
Noma		1
Peritoneum:		
Acute peritonitis (localized 2, with acute pneumonia and pleurisy 2)		4
Kidneys:		
Acute nephritis (complicating scarlet fever 4, diphtheria 1, pneumonia 4, measles 1, pertussis 1, ileocolitis 2, pyonephrosis 1, apparently primary 5)		19
Malformations of the kidney		7
Stomach and Intestines:		
Acute ileocolitis, with or without gastritis		116
Acute gastritis (without intestinal lesions)		None
Acute diarrheal disease (without gross lesions)		72
Intussusception		1

of syphilitic, origin. Diseases of the blood are quite common, but as yet but little understood. New growths are rare. The parts most frequently affected are the kidneys and the bones. Disorders of nutrition are extremely common and of great importance, particularly rickets and scurvy.

PROGNOSIS AND INFANT MORTALITY

The younger the patient the worse the prognosis in all the diseases of childhood. This is in consequence of the feeble resistance of the infantile organism to all diseases, particularly those which are of an acute nature. On the other hand, the rapid metabolism of childhood makes it possible for many conditions of an organic nature to disappear with time, or, as the phrase is, to be "outgrown," provided the patient can be so placed that the general nutrition can be carried to the highest point.

The accompanying chart (Plate I) shows the mortality of New York City by months during three consecutive years, representing a total mortality of 128,136.

The following table gives comparative figures of actual deaths for four periods of three years each, and shows the reduction in infant and child mortality which has taken place in the last twenty-five years:

Deaths—New York City (Boroughs of Manhattan and Bronx)

	1890–1892.	1898–1900.	1907–1909.	1912–1914.
Under 1 year.	32,916 = 26%	29,326 = 24%	30,626 = 22.5%	25,015 = 19.1%
1 to 2 years.	10,547 = 8%	9,012 = 7%	8,298 = 6.0%	6,527 = 5.0%
2 " 5 "	9,794 = 7%	7,292 = 6%	6,579 = 5.0%	5,408 = 4.1%
5 " 15 "	5,470 = 5%	6,922 = 5%	4,902 = 3.5%	4,533 = 3.5%
Over 15 "	69,409 = 54%	71,024 = 58%	85,741 = 63.0%	89,341 = 68.3%
Total.....	128,136	123,576	136,146	130,824

Heart:

Pericarditis (all with acute pneumonia)	3
Congenital malformations	3
Acute or chronic endocarditis	None

Brain:

Acute meningitis (7 with pneumonia, 2 cerebrospinal)	14
Tuberculous meningitis	11
Acute encephalitis	1
Chronic pachymeningitis	5
Chronic meningitis	1
Chronic hydrocephalus	3

There were twenty-six deaths from marasmus without gross lesions.

The deaths per 1,000 of population show the same reduction. The curves for the different age periods are indicated in the accompanying chart (Fig. 4).

The reduction in infant mortality in New York has been chiefly in acute gastro-intestinal diseases, marasmus and debility, especially in those over three months old. In older children it has been chiefly in acute infectious diseases, especially diphtheria. The mortality from

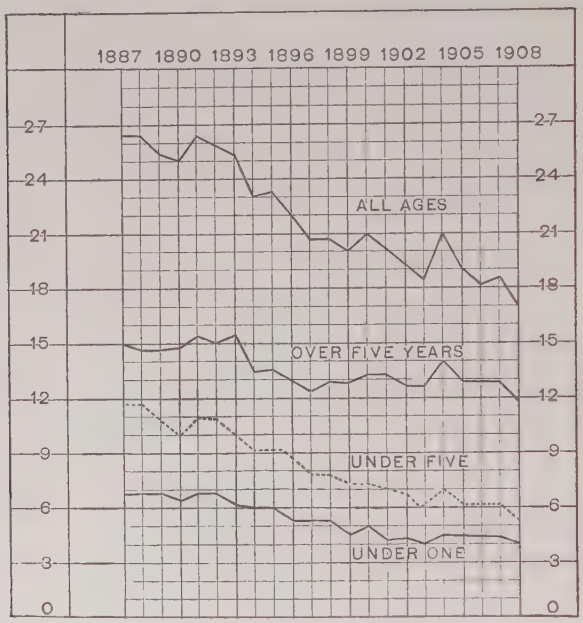


Fig. 4.—Deaths—New York City—per 1,000 of Population.

certain other causes is increasing, notably acute respiratory diseases and prematurity.

The only age in which the mortality is increased during the summer months is the first year. In Fig. 5 are given the curves indicating the deaths under one year and from one to five years by months.

The rise in the summer mortality during the first year is chiefly due to diarrheal diseases. As a result of the organized campaign for the reduction of infant mortality in New York which has been in full operation since 1911, the number of infant deaths has steadily fallen. That part of the mortality curve chiefly affected has been the sharp summer rise which has been almost obliterated. It will be noted that the curve for children from one to five years of age touches the highest point in the late winter and early spring months, the time when pneumonia and the common contagious diseases are most prevalent. The

PLATE I

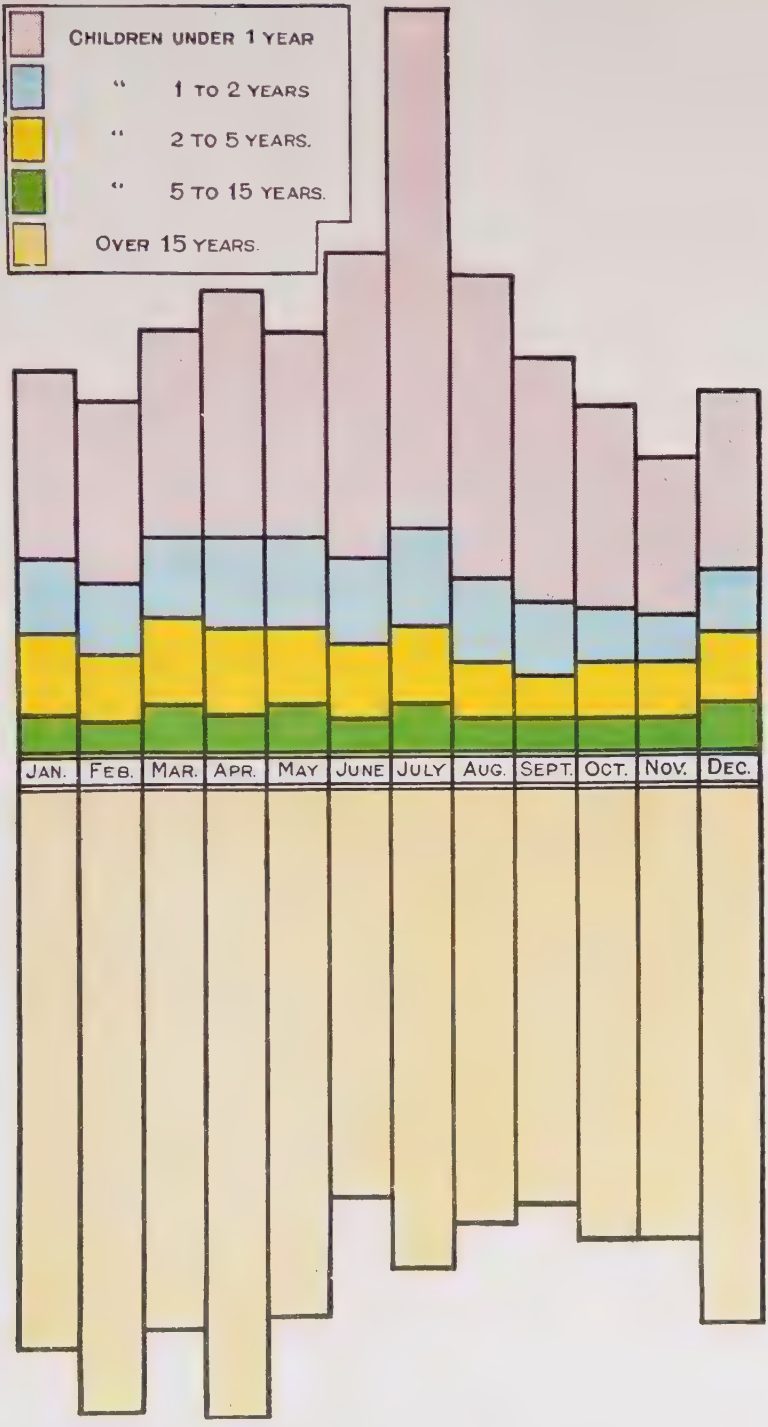


CHART SHOWING BY MONTHS THE MORTALITY OF NEW YORK CITY FOR THE DIFFERENT AGES FOR THREE CONSECUTIVE YEARS. Scale, 1 in. — 2,200 deaths.

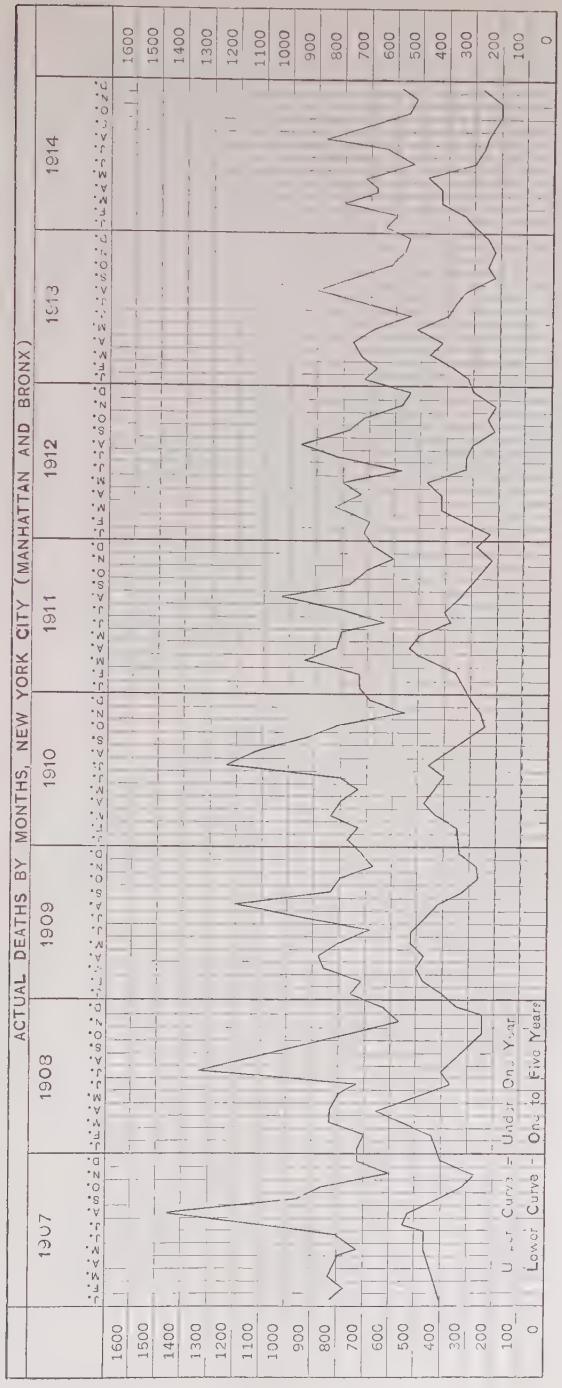

ACTUAL DEATHS BY MONTHS, NEW YORK CITY (MANHATTAN AND BRONX)
1907
1908
1909
1910
1911
1912
1913
1914
1600
1500
1400
1300
1200
1100
1000
900
800
700
600
500
400
300
200
100
0
Upper Curve = Under One Year
Lower Curve = One to Five Years

FIG. 5.

curve for both groups is lowest in the months of October and November, which may therefore be considered the healthiest months in New York. The highest mortality is in the first month of age. During this time twenty-five per cent of the deaths of the first year occur. Eröss, writing in 1894, states that from the records of sixteen large cities of Continental Europe nearly ten per cent of all the infants born died during the first month. These figures have been considerably reduced since that time.[1] The first weeks of life are the period of highest mortality, because in them takes place the adaptation of the organism to its environment. After this period each month shows a steadily declining death rate to the end of the first year.

Causes of Death at Different Periods.—The most frequent causes of infant mortality, according to the combined reports from the records of the cities of New York, Philadelphia, Boston, and Chicago, making a total of 44,226 deaths in the first year, are shown in the accompanying chart (Fig. 6).

The group, acute gastro-intestinal, includes chiefly diarrheal diseases in summer. The proportion of deaths from this cause is being greatly reduced; while the proportion due to acute respiratory diseases, chiefly pneumonia and bronchitis, is increasing. Marasmus, prematurity, etc., include also congenital debility, inanition, and other conditions in which the cause of death recorded is disorder of nutrition rather than some general or local disease. The group, congenital malformations, includes

[1] The relative frequency of the causes of death in the newly born has been greatly altered since the introduction of antiseptic midwifery. Some idea of the importance of the different factors has been gained from a study of the records of the Sloane Hospital for Women for a period of six years (1908-1914), embracing 10,000 consecutive births.

CAUSES OF DEATH DURING FIRST FOURTEEN DAYS

	Under One Day		Under 7 Days		Seven to 14 Days		Total Under 14 Days		Grand Totals
Congenital weakness	**93**	2	**120**	7	**14**	2	**134**	9	143
Accidents of labor	**1**	14	**1**	32	..	..	**1**	32	33
Pneumonia	..	..	**3**	9	**3**	13	**6**	22	28
Atelectasis	**3**	7	**3**	14	**1**	7	**4**	21	25
Congenital syphilis	**5**	..	**6**	1	**6**	..	**12**	1	13
Malformations	..	4	**2**	7	..	3	**3**	10	12
Hemorrhage	..	..	..	8	..	2	..	10	10
Sepsis	..	..	..	2	..	7	..	9	9
Asphyxia	..	7	..	8	..	..	..	8	8
Accidental	..	1	..	2	..	..	..	2	2
Undetermined	..	3	..	8	..	..	..	8	8
Totals	**102**	38	**135**	98	**24**	34	**159**	132	291

Ten thousand confinements: Abortions, 253; stillbirths, 429; living births, 9,318. (Prematures, heavy type.) Holt and Babbitt, Jour. American Med. Assn., Jan. 25, 1915.

Nearly half of the total mortality for the period covered was ascribed to congenital weakness, chiefly due to prematurity.

also deaths from accidents during birth. Whooping cough is the most important member of the group of acute infectious diseases, diphtheria coming next. Tuberculosis should, we believe, be rated higher than is shown in these figures. The mortality records of the Babies' Hospital show that the deaths from tuberculosis constitute 5.6 per cent of the first-year mortality of that institution.

The figures and charts preceding indicate that a very marked reduction in infant and child mortality has taken place especially within the last twenty years. Many causes have united to bring about this result. Among those which have affected infants may be mentioned:

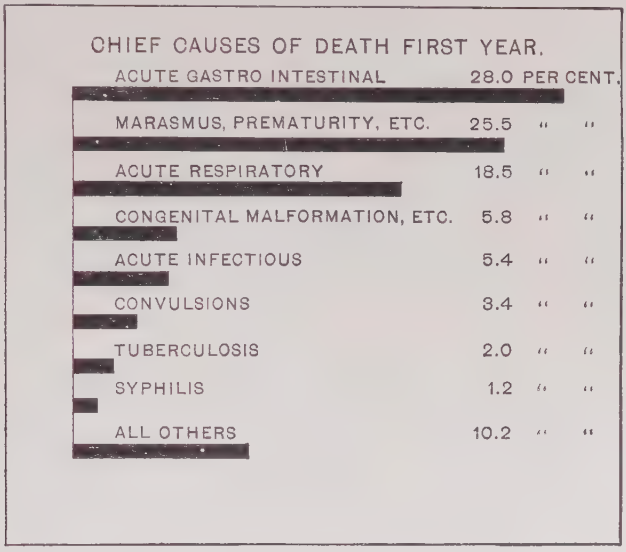

CHIEF CAUSES OF DEATH FIRST YEAR.
ACUTE GASTRO INTESTINAL 28.0 PER CENT.
MARASMUS, PREMATURITY, ETC. 25.5 " "
ACUTE RESPIRATORY 18.5 " "
CONGENITAL MALFORMATION, ETC. 5.8 " "
ACUTE INFECTIOUS 5.4 " "
CONVULSIONS 3.4 " "
TUBERCULOSIS 2.0 " "
SYPHILIS 1.2 " "
ALL OTHERS 10.2 " "

FIG. 6.

A wider diffusion of knowledge of infant-feeding and hygiene; a great improvement in the general milk supply; the furnishing of pure, whole milk and of modified milk gratis, or at small cost, from milk depots; a general adoption during hot weather of some form of milk sterilization; the sending of a large number of infants into the country in summer; the closer supervision of infants in cities during the summer by visiting physicians and nurses, and the operation of many other agencies to improve sanitary conditions. Besides these important factors in preventing disease there must be considered the recent advances in pediatrics and the more rational treatment of the sick infant by the average physician.

During the second year the diseases of the gastro-intestinal tract are still a large factor in the death rate, also the acute diseases of the lungs and the acute infectious diseases, especially measles, diphtheria, and per-

tussis. Deaths from scarlet fever are much less numerous. General tuberculosis and tuberculous meningitis are frequent.

From the second to the fifth year the deaths are mainly from acute infectious diseases—chiefly diphtheria and scarlet fever—much less frequently from measles or pertussis. In the next group come the acute diseases of the lungs, general tuberculosis, and tuberculous meningitis.

From the fifth to the fifteenth year the mortality in childhood is remarkably small, diphtheria and scarlet fever being still in the front rank in point of frequency. Next come the acute diseases of the lungs, meningitis, diseases of the bones, appendicitis, rheumatism, and cardiac disease.

By far the largest single factor in reducing mortality after the first year is without doubt the use of diphtheria antitoxin. The serum treatment of cerebrospinal meningitis is important, but not influential in vital statistics, as cases are relatively infrequent.

Sudden Death.—This is not a very uncommon occurrence in infants who are apparently healthy. They are sometimes found dead in bed under circumstances in which grave suspicion may unjustly rest upon the attendants. This usually happens with those who are delicate or suffering from malnutrition, especially in institutions where sudden death is by no means rare. The most frequent causes in infants are the following:

1. *Malformations.*—While in most cases malformations of a serious nature give rise to symptoms, they may be absent, or may be so slight as to be overlooked. Infants may succumb during the first few days of life from malformations of the heart, lungs, kidneys, stomach or intestines, and sometimes from diaphragmatic or umbilical hernia.

2. *Internal Hemorrhage.*—This is chiefly limited to the first two weeks of life. In the cases that have come to our notice the cause has been rupture of some subperitoneal hemorrhage into the general abdominal cavity, or meningeal hemorrhage. Such cases are reported in the chapter upon Visceral Hemorrhages in the Newly Born. Under these circumstances no symptoms may exist until the occurrence of collapse, with death in a few hours.

3. *Asphyxia from Overlying.*—This is not common, except among the lower classes, and is most frequently due to intoxication on the part of the mother. Such infants after death present the usual lesions of death from asphyxia, but without any evidence of violence. It is not improbable that overlying has been held responsible for many deaths that were in reality due to other causes.

4. *Asphyxia from Aspiration of Food into the Larynx or Trachea.*—This may be due to vomiting or to the regurgitation of food during sleep; in a very weak infant it may occur while awake. This is usually

seen in infants who are less than a year old, and most of the reported cases have been under six months. Such children are usually delicate. There seems to have been vomiting with an attempt at crying, during which the food is drawn into the air passages. In some cases, as that reported by Demme, a single large curd of milk has been found in the larynx. In others, food is found in the larynx, trachea, and large bronchi. Cases have also been reported by Partridge and by Parrot, and we have met with at least three. The infants have generally been found dead in bed within a few hours after feeding. This accident is more likely to happen when an infant lies upon his back.

5. *Enlargement of the Thymus.*—Although these cases are very imperfectly understood, they are not rare. We see two or three each year. The condition is most frequent in infancy, but is not confined to this period. When a child is suffering from some minor illness, often bronchitis, severe attacks of asphyxia may develop and sometimes convulsions may unexpectedly occur and death soon follow. Or the first attack may not be fatal. Sometimes sudden death follows the administration of an anesthetic, particularly chloroform. In most cases there is found besides an enlarged thymus, a general hyperplasia of the lymphatic tissues throughout the body known as *status lymphaticus,* more fully discussed elsewhere.

6. *Atelectasis.*—In very young infants there may be no symptoms noticed except those of general malnutrition until sudden death occurs, sometimes with convulsions and sometimes without any such symptoms. (See Atelectasis.)

7. *Marasmus.*—In this class of cases sudden death is of very common occurrence. These children are often apparently as well two or three hours before death as for several weeks. Death frequently occurs at night, the children being found dead in bed in the morning. In some of the cases the exciting cause seems to be the lowering of the temperature, while in many no exciting cause can be found; the vital spark simply goes out after burning for some time with a feeble intensity. In some of these cases the autopsy reveals atelectasis, but in many cases nothing abnormal is found, death apparently resulting from heart failure.

8. *Convulsions in Children Previously Showing no Special Signs of Disease.*—Many of these cases are seen in children who were previously rachitic; some are associated with the status lymphaticus, and many are manifestations of tetany. The autopsy may show no lesion except cerebral hyperemia. It is extremely rare for cerebral hemorrhage to produce death in this way. In some rachitic cases death is due to spasm of the glottis.

9. *Asphyxia in Older Infants and Young Children.*—This may result from the pressure of a retropharyngeal abscess upon the larynx or

trachea, or from the rupture of such an abscess into the air passages. Previous symptoms may have been wanting. Pressure upon the pneumogastric nerve leading to fatal asphyxia may be caused by tuberculous bronchial nodes, or by abscesses in the posterior mediastinum connected with caries of the spine. Sudden death may occur with spinal caries from dislocation of the upper cervical vertebrae.

Sudden asphyxia may follow the ulceration of tuberculous lymph nodes and the escape of cheesy masses into the trachea or primary bronchi. This usually occurs in children from two to five years old.

10. *Death after a Few Hours' Illness, in which the Chief Symptom is High Temperature.*—This is not an uncommon occurrence. Infants apparently well may be taken with great prostration and a high temperature, which may rise rapidly to 106° or even 107° F., and death follow in from six to twelve hours, sometimes preceded by convulsions. These are often examples of acute septicemia, most frequently from the pneumococcus, sometimes from the streptococcus, or other organisms. In older children death may be due to malignant scarlet fever or epidemic meningitis; however, unless these diseases are prevailing epidemically, it is somewhat hazardous to make such a diagnosis.

It does not fall within the scope of this chapter to consider such cases of sudden death as those which occur from heart failure after diphtheria, with pleurisy with effusion, or with myocarditis. These will be discussed elsewhere.

PROPHYLAXIS

There is no more promising field in medicine than the prevention of disease in childhood. The majority of the ailments from which children die it is within the power of man in great measure to prevent. Prophylaxis should aim at the solution of two distinct problems: (1) The removal of the causes which interfere with the proper growth and development of children; (2) the prevention of infection. The former can come only through the education of the profession and of the general public, in the fundamental principles of infant feeding and hygiene. This is a department which has received altogether too small a place in medical education. The latter must come through the profession and through legislation the purpose of which shall be more rigid quarantine, more thorough disinfection, and improved sanitation in all its departments. The subject of prophylaxis will be discussed in connection with the different diseases treated elsewhere.

THERAPEUTICS

Therapeutics in infancy consists in something more than a graduated dosage of drugs. Many therapeutic means which are valuable in adults are useless in children, and many others which are of little value in adults are extremely useful in children. Children in the past have suffered much from overzealous treatment, particularly from drug-giving. In early life more than at any other period the old dictum *non nocere* should be heeded. It should be a fundamental principle never to give a dose of medicine without a clear and definite indication. If this rule is followed, it is surprising to find how often medication can be dispensed with. A second rule is equally important: never to give a nauseous dose when one that is palatable will answer the purpose equally well. The simpler prescriptions are made, the better. As a rule, infants revolt against most of the highly seasoned sirups and elixirs which are used to disguise the taste of unpleasant doses. Bitter medicines, when mixed with water, are frequently administered without difficulty.

It is a common mistake to underestimate the importance of the hygienic surroundings of the patient, the value of good nursing, careful feeding, and judicious stimulation, just as it is to overestimate the beneficial effects of drugs. In the great majority of acute ailments not serious in character, for which a physician is called, the patient recovers quite as promptly without drugs as with them. This does not mean that such children require no treatment, but that the least important part of the treatment is drug-giving. In cases of severe illness, in infants especially, we must avoid all unnecessary medication, in order that the stomach may not be disturbed. Hence the importance of relying as far as possible upon local measures. The strong tendency to recovery from all acute processes, while seen in adult life, is even more striking in childhood, where, if we can but remove that which hampers the bodily functions, Nature will usually conduct the case to a satisfactory termination. Thus, after an attack of bronchitis, it is often seen that the disturbance of the stomach and intestines can be directly traced to the drugs employed, and continues long after the original disease has subsided. In diseases of the stomach and intestines especially there is a great amount of unnecessary medication. In all chronic disturbances of nutrition—chronic indigestion, malnutrition, and anemia—no tonic is so good as a change of air and surroundings.

Antipyretics.—The indications for the employment of antipyretics in children are somewhat different from those in adults. It is to be borne in mind that, where the cause is similar, all temperatures in children are

higher than in adults. Thus conditions, which in an adult would produce a rise of temperature of only 100° or 101° F., in a child are not infrequently accompanied by a temperature of 104°, or even 105° F. The height of the temperature, as measured by the thermometer, is not to be taken as the only or even the best guide for the employment of antipyretics. The nervous disturbance which accompanies such a temperature is much more important. The temperature may be 104°, or even 105° F., and yet the child exhibit no signs of unusual discomfort. Such a temperature manifestly does not call for interference. High temperature from apparently trivial causes is very common. It is only a continuously high temperature or a recurring high temperature which indicates serious illness. Whenever the temperature is found to be much above the normal it should be carefully watched, but not interfered with until a diagnosis has been made, unless the symptoms urgently demand it; otherwise the physician may lose one of the most valuable aids to diagnosis, since it is not the height of the temperature but its course which is significant. In many cases it is very important to know whether the temperature uninfluenced by drugs is remittent, intermittent, or steadily high, and hence the advantage of waiting until a diagnosis has been made before disturbing the temperature curve. This is, of course, not admissible when the temperature is itself a source of real danger, which after all is seldom the case. Since the cause of a great many obscure temperatures is found in the stomach and intestines, it very often happens that a purgative, stomach-washing, or intestinal irrigation may be the most efficient antipyretic. In cases of moderate elevation of temperature we need go no further than cold sponging.

The most reliable antipyretic measure for children is the use of cold. This may be employed—

(1) *As an Ice Cap to the Head.*—In many cases of quite high temperature and restlessness in infants this alone will reduce the temperature one or two degrees and allay the nervous symptoms.

(2) *Cold Sponging.*—For this purpose water at about 80° to 85° F., equal parts of alcohol and water, or equal parts of vinegar and water may be employed. In the case of infants, all the clothing except the diaper should be removed and the child laid upon a blanket. The body should be sponged for from ten to twenty minutes, and then wrapped in a blanket without further dressing. Cold sponging must be very frequently employed in order to be efficient in reducing high temperature. Its great value in allaying nervous symptoms, even when the temperature is not very high, is not sufficiently appreciated. Its effect is often more satisfactory than that of an anodyne.

(3) *Cold Pack.*—The child should be stripped and laid upon a blanket. The entire trunk should then be enveloped in a small sheet

wrung from water at a temperature of 100° F. Upon the outside of this, ice may now be rubbed over the entire trunk, first in front and then behind. By this method there is no shock and no fright, and any ordinary temperature can usually be readily reduced.

The rubbing with ice should be repeated in from five to thirty minutes, after which the child may be rolled in the blanket upon which he is lying without the removal of the wet pack. The head should be sponged with cold water while this is being carried on, and artificial heat, if necessary, should be applied to the feet. The pack is continued from one to twenty-four hours, according to circumstances.

(4) *Cold Bath.*—The child is put into a bath at a temperature of 100° F., the temperature being gradually lowered by the addition of ice or cold water to 75° or 80° F. The body should be well rubbed while the child is in the bath and water should also be applied to the head. On removal from the bath, the body should be quickly dried and rolled in a warm blanket. The bath is usually continued from five to ten minutes.

(5) *Evaporation Bath.*—The trunk is closely enveloped in two layers of surgeon's gauze, or some loosely woven equivalent, which is moistened from time to time with water at a temperature of 95° F., continuous evaporation being kept up by means of a hand, or better electric, fan. The evaporation bath would seem to possess some important advantages in the case of infants and young children, in that it is more efficient than sponging, involves little disturbance of the patient, and causes no shock or fright. Hot applications should constantly be made to the extremities.

(6) *Rectal Irrigations.*—These are easily given, disturb the patient very little, and are effective in reducing the temperature. A double tube or two catheters may be employed. It is best to use at first water at a temperature of 90° F., and gradually reduce this to 70° F. The irrigation should be continued for ten or fifteen minutes, or even longer if the desired fall in temperature is not obtained, and may be repeated as often as every three hours.

Antipyretic Drugs.—Except in cases of malaria, quinin should not be employed for the reduction of temperature in children.

Of the many coal-tar derivatives employed, phenacetin has the advantage for children of being tasteless and causing little depression, but the slight disadvantage of practical insolubility. None of the drugs of this group is, however, to be employed in large doses with the sole purpose of reducing the temperature. Their great value in pediatrics consists rather in allaying the nervous symptoms which accompany fever, and this purpose can be accomplished by the use of comparatively small doses. To an infant of one year, phenacetin can be given in one-grain doses every hour or two hours until the desired effect is produced. For

a child of five years a dose of two grains may be given in the same manner. When used as indicated, these drugs are of very great value in making the patient more comfortable, in promoting sleep, and in allaying headache and general pains. In cases of hyperpyrexia they are, however, much less certain and less safe than the use of cold.

Sedatives.—For most of the milder conditions where sedatives are required bromids are to be preferred. A preference should be given to the sodium salt. Young children require relatively large doses: e. g., in convulsive conditions five grains every two hours are often necessary at three months. Chloral is usually well borne even by quite young infants. Since it is often irritating to the stomach it may be advantageously given by the rectum. After rectal administration its effects are usually manifest in half an hour, and sometimes sooner. The rectal dose for an infant of one month is one grain; three months, two grains; one year, three to five grains. It may be repeated every two to four hours, according to indications. Doses by mouth should be about half as large. Other drugs may replace this in most diseases, but in the case of infantile convulsions nothing is so reliable as chloral.

Belladonna is well borne by children, and in relatively larger doses than most drugs. The eruption is more readily produced than the other physiological effects, and even quite small doses may be sufficient to bring out a very abundant blush. The parents should be advised of this fact, lest undue alarm be felt.

The drugs classed as antipyretics—phenacetin and antipyrin—are exceedingly valuable in the treatment of many diseases of infancy where irritative nervous symptoms are prominent. In many cases they may advantageously take the place of opium except when pain is present. In all conditions where spasm is a prominent symptom, whether of the larynx or bronchi, or local or general convulsions, antipyrin is especially valuable.

Stimulants.—Alcoholic stimulants are well tolerated even by young infants; yet all stimulants, alcohol in particular, are very greatly abused in the hands of practitioners, and their indiscriminate and protracted use can not be too strongly condemned.

The indications for the employment of stimulants are much the same in young children as in adults. In most of the acute fevers they are not to be given early in the disease, and in many cases they are not required at all. They must often be used very sparingly while the temperature is high, but may be necessary as soon as it falls. In many acute febrile diseases stimulants are not called for at any period.

The method of administering alcohol is of no little importance. Brandy and whisky are in most cases to be preferred to the wines, but not always. For infants under one year old, brandy should be diluted

with at least twenty parts of water. Altogether the best method of administration is to determine the amount to be given in every twenty-four hours, have it diluted sufficiently, and then administer it in small doses at short intervals.

An infant one year old for whom alcohol is indicated should not be given to begin with more than one-fourth of an ounce of brandy or whisky during the twenty-four hours, and even in bad conditions it is rarely advisable to give more than twice this quantity, except for a very short period. In children four years old double the amount may be employed in the corresponding conditions. Little good and much harm is likely to follow such amounts as four or five ounces daily of brandy or whisky for children of two or three years. There certainly is a strong tendency at the present time to use less and less alcohol in therapeutics and many would abandon it altogether.

Other stimulants, caffein, camphor, strychnin, digitalis, strophanthus, etc., are used in children with much the same indications as in adults. Their application is more fully discussed in the different diseases in which they are employed. They may be used in the following doses at the different ages indicated:

	3 months.	1 year.	5 years.
Digitalis, tincture	♏ i	♏ iii	♏ v
Strophanthus, tincture	♏ i	♏ iii	♏ v
Caffein citrated	Gr. $\frac{1}{4}$	Gr. i	Gr. ii
Strychnin sulphate	Gr. $\frac{1}{400}$	Gr. $\frac{1}{200}$	Gr. $\frac{1}{60}$
Camphor (10 per cent solution in oil)	♏ v	♏ x	♏ xx
Epinephrin (1–1000 Sol.)	♏ iii	♏ vi	♏ x

Note.—Camphor and epinephrin are for hypodermic use only.

Tonics.—Cod-liver oil is particularly useful in the convalescence after acute diseases of the respiratory tract, in anemia, and with a large number of children who are extremely delicate. In these patients it may be advantageously administered throughout the greater part of nearly every winter season. In convalescence after attacks of gastroenteric disease it should be withheld for a long time. When the tongue is coated, the digestion poor, and the stomach easily disturbed it should not be given at all. In the case of infants, as a rule, the pure oil is to be preferred to the emulsions. The administration of small doses—i. e., ten or twenty drops of the oil three times a day continued for a long period—is often better than the use of larger doses for a shorter time.

Preparations of malt are sometimes of even greater value than cod-liver oil, for they can be used in many conditions when the oil is contra-indicated. The two may often be advantageously combined.

The best preparations of iron for very young children are the bitter

wine, sweet wine, saccharated carbonate, and the wine of the citrate. These are only slightly constipating, and many of them can be given with milk. Most of the organic preparations on the market are less reliable than those mentioned. For older children nothing is better than reduced iron or Blaud's pills.

Arsenic is second only to iron in the treatment of the anemia of children, and in very many cases it is to be preferred to iron. The tablet triturates of arsenious acid, one one-hundredth of a grain, may be given immediately after meals three times a day, or one or two drops of Fowler's solution largely diluted with water.

Alcohol is useful in combination with some of the bitters, either small doses of quinin, nux vomica, or the bitter wine of iron. Usually wines, especially sherry, are to be preferred to spirits, although some children take spirits better. When combined with a bitter there is little danger of the formation of the alcoholic habit, even though its use may be long continued.

Of the bitter tonics, nux vomica is easily superior to all others.

Opiates.—Strong objections have been urged by many against the employment of opium in the diseases of infancy. While opiates have no doubt been abused, the fact remains that opium is almost as valuable a remedy in the treatment of disease during the first five years as at any other period of life. For infants relatively smaller doses are required than of most drugs. If the physician will accustom himself to the use of small doses, he will be surprised to see how satisfactory are the effects produced.

The most useful preparations for young children are paregoric, Dover's powder, the deodorized tincture, morphin, and codein. The following table gives what may be considered safe initial doses at the different ages:

	1 month.	3 months.	1 year.	5 years.
Paregoric	♏ i	♏ ii	♏ v to x	♏ xxx to xl
Deodorized tincture	♏ $\frac{1}{20}$	♏ $\frac{1}{10}$	♏ $\frac{1}{4}$ to $\frac{1}{2}$	♏ ii to iii
Dover's powder	Gr. $\frac{1}{10}$	Gr. $\frac{1}{6}$	Gr. $\frac{1}{2}$	Gr. ii to iii
Morphin	Gr. $\frac{1}{200}$	Gr. $\frac{1}{150}$	Gr. $\frac{1}{60}$	Gr. $\frac{1}{15}$
Codein	Gr. $\frac{1}{150}$	Gr. $\frac{1}{100}$	Gr. $\frac{1}{30}$	Gr. $\frac{1}{10}$ to $\frac{1}{8}$

Ordinarily doses like the above should not be repeated oftener than every two hours. In exceptional circumstances, as when very great pain is present, the dose may be given more frequently. In the hypodermic use of morphin it should be remembered that its effects are always more uniform and striking than when the drug is administered by the mouth, and the dose should therefore be smaller. In every instance where a full

dose of opium has been given the physician should wait until the effects have subsided before the dose is repeated.

Drugs Well Borne by Children.—In this list may be mentioned belladonna, the bromids, the iodids, chloral, quinin, calomel—in fact, all mercurials—and opium also, though not all of its products.

The drugs not well borne include particularly cocain and heroin. In the case of many others, while the constitutional effects are well tolerated, they must be given carefully to young infants, since they are irritants to the stomach. In this class may be mentioned the salicylates, salol, the astringent preparations of iron, and the acids.

Vaccines.—These are suspensions of dead bacteria in a normal salt solution. Their application in pediatrics is confined to therapeutics; as a prophylactic measure they are seldom called for, except for the prevention of typhoid fever. Vaccine therapy has been employed in almost every form of bacterial infection. In the great majority of these the results have been disappointing. They are of unquestioned value in localized staphylococcus infections, particularly those of the skin, e. g., general furunculosis and larger multiple abscesses. In other staphylococcus infections they are sometimes useful, but results are very uncertain. In streptococcus infections whether localized or general their effect is doubtful; although in rare cases they have seemed to be of benefit. Pneumococcus infections are apparently not at all influenced by their use. Regarding the effect of vaccines on gonococcus infections of mucous membranes, one must speak very guardedly. The great majority of patients with gonococcus vaginitis so treated have received but temporary benefit, although a few striking cures have been obtained. Colon bacillus infections of the urinary tract (pyelitis) sometimes appear to be decidedly improved by vaccines. With respect to most other conditions experience thus far does not warrant one in forming a sanguine opinion of their value. For the technic of vaccine treatment special works should be consulted.

Counterirritants.—These are of great value in a large variety of diseases.

The *mustard paste* is probably the most satisfactory means of producing quick counterirritation over a large surface. To make a mustard paste: Take one part powdered mustard and six parts of wheat flour, mix with lukewarm water, and spread between two layers of muslin. This should be removed as soon as a thorough redness of the skin has been produced—in most cases from five to eight minutes, according to the strength of the mustard employed. This may be repeated as often as every three hours, and continued for a week if necessary, without producing excoriations of the skin. For older children the paste may be made one part mustard to four parts flour. In pulmonary diseases it

should be large enough to surround the chest. When it is used to produce general reaction in heart failure it should cover the entire trunk.

The Mustard Pack.—The child is stripped and laid upon a blanket, and the trunk is surrounded by a large towel or sheet saturated with mustard water. This is made as follows: One tablespoonful of mustard to one quart of tepid water. In this a towel is dipped, and while dripping wound around the entire body. The patient should then be rolled in the blanket. This pack may be continued for ten or fifteen minutes, at the end of which time there will usually be a very decided redness of the whole body. It may be repeated according to indications. Where it is desired to produce a general counterirritation, the mustard pack is not quite as efficient as the mustard bath, but it has the advantage in causing much less disturbance to the patient. The mustard pack is useful in the condition of collapse or of great prostration from any cause whatever, in convulsions, and in cerebral or pulmonary congestion.

The *turpentine stupe* is made by wringing a piece of flannel out of water as hot as can be borne by the hand. Upon this is sprinkled ten or fifteen drops of the spirits of turpentine. The stupe is then applied to the body and covered with oiled silk or dry flannel. It is useful chiefly in abdominal pains or inflammations, but in infancy must be carefully watched or vesication will be produced. For frequent use it is not so valuable as the mustard paste.

Stimulating liniments containing turpentine and other irritants are useful in inflammations of the chest, although less reliable than the mustard paste. One of the mildest and most useful preparations is camphorated oil. Another is olive oil four parts and turpentine one part. These may either be rubbed upon the surface, or a piece of flannel may be saturated with them and then applied to the skin.

Local Blood-letting.—Leeches are sometimes useful in the early stages of acute inflammations of the mastoid or middle ear. They may also be applied to the precordium in acute pneumonia with signs of failure of the right heart, viz., great dyspnea and cyanosis.

Dry cups may be used even in young infants, to relieve acute congestion in pneumonia or bronchitis, and for pulmonary edema. Wet cups should never be used for young children.

Poultices are much too frequently employed and may with advantage be omitted in the treatment of most local inflammations. They have been largely replaced by wet dressings, especially those of aluminum acetate. In acute pulmonary inflammations their use is very limited.

Cold.—Cold is useful in almost all forms of local inflammation. In inflammation of the cervical lymph glands and of the joints it is of undoubted value, but its advantage over heat is questionable. The efficiency of both cold and heat in these cases depends largely upon the

method of use. The difficulties in the way of their proper application are great in young children. Sometimes in pleurisy much greater relief is obtained from the use of an ice bag to the chest than from hot applications, but this is not the general experience. The treatment of pneumonia by the application of the ice bag to the chest has many advocates, although in our hands it has not yielded the results claimed for it. It is admissible only in lobar pneumonia, and here chiefly in older and stronger children. The application of cold in young or very delicate children should be made with caution in all inflammations of the respiratory tract.

Cold is best applied to the head by an ice cap made like a helmet; an ordinary rubber or flannel bag filled with ice may answer the purpose. The rubber coil filled with ice water is also an excellent method. For inflamed glands or joints the ice bag or the coil should be used; for the eyes, cold compresses, changed every minute.

The Hot Pack.—All clothing is to be removed and the child's body covered with towels wrung from water at a temperature of from 100° to 108° F., after which the body should be rolled in a thick blanket. These hot applications may be changed every twenty or thirty minutes until free perspiration is produced, which may be continued as long as necessary. This is mainly useful in uremia.

The hot bath, like the mustard pack or the mustard bath, may be used to promote reaction in cases of shock or collapse. The patient should be put into the bath at a temperature of 100° F., the water being gradually raised to 103°, or even to 106°, but not above this point. The body should be well rubbed while the patient is in the bath. A thermometer should be kept in the water to see that the temperature does not go too high. Unless this precaution is taken the danger of burning the child is great. During the bath, in most cases, cold should be applied to the head.

The Hot-Air or Vapor Bath.—All the clothing should be removed and the patient laid upon the bed with the bedclothing raised above the body ten or twelve inches, and sustained by means of a wicker support. The bedclothing should be pinned tightly about the neck, so that only the head is outside. Beneath the bedclothing hot vapor is introduced from a croup kettle or a vaporizer. This will usually induce free perspiration in fifteen or twenty minutes. It may be continued from twenty to thirty minutes at a time. Instead of vapor, hot air may be introduced in the same way. The air space about the body is indispensable. The vapor bath is applicable chiefly to cases of uremia.

The Mustard Bath.—Four or five tablespoonfuls of powdered mustard should be mixed for a few minutes with one gallon of tepid water. To this should be added four or five gallons of plain water at a temperature

of 100° F. The temperature of the bath may be raised by the addition of hot water to 103° or 106° F., if desired. Nothing is more efficient than the hot mustard bath for a general derivative effect in bringing the blood to the surface in cases of shock, collapse, heart failure from any cause, or in sudden congestion of the lungs or brain. The bath should not usually be continued for more than ten minutes. If necessary, it may be repeated in an hour.

The Bran Bath.—Put one quart of ordinary wheat bran in a bag made of coarse muslin or cheese cloth and place this in four or five gallons of water. The bran bag should be frequently squeezed and moved about until the bath water resembles a thin porridge. It may be of any temperature desired, but usually about 90° to 95° F. is best. A bran bath is of great value in cases of eczema, excoriations about the buttocks, or in other cases where the skin is very delicate, and plain water seems to irritate it.

The tepid bath may be given at a temperature of 95° to 100° F. It is very useful in many conditions of excitement or extreme nervous irritability. To induce sleep it is often more efficient than drugs.

The cold sponge or the shower bath should be given in the morning before breakfast, and in a warm room. The child should stand in a tub containing warm water enough to cover the feet, then a large sponge holding half a pint of water at a temperature of from 40° to 60° F. should be squeezed three or four times over the chest, shoulders, and spine of the child, the skin being rubbed meanwhile. The bath should not last more than half a minute. It should be followed by a brisk rubbing until a thorough reaction is established. This is very useful at all ages, but it is a particularly valuable tonic in delicate children. It may be used in those only eighteen months old. Not the least of the beneficial results is the full expansion of the lungs from the strong cry which the bath usually excites. In younger infants a cold plunge may be substituted. This should be merely a single dip of the entire body in water at a temperature of 50° to 60° F. In order that beneficial effects shall follow the cold plunge or cold sponging, a good reaction must be established. If children lack sufficient vitality to secure this, and if they remain pale, pinched and blue for some time after the bath, it must be discontinued altogether, or water of a higher temperature used.

Nasal Spray.—This may be either of an aqueous or an oily solution. For the oil spray an atomizer should be employed. It is valuable in cases of dry catarrh, where there is a formation of crusts in the nose. A variety of oils may be used, benzoinol being perhaps as satisfactory as any.

There are many forms of hand atomizers to be found in the market for the production of aqueous or oil sprays. For a cleansing nasal spray,

Dobell's solution, Seiler's solution, or a two-per-cent solution of boric acid may be used.

Nasal Irrigation.—In cases of considerable nasal obstruction and in the more serious affections of the rhinopharynx, only the syringe can be considered an efficient means of cleansing the cavity.

The fountain syringe has the advantage of being easily regulated as to the force employed, this being determined by the height at which the bag is suspended above the bed. For ordinary purposes an elevation of one or two feet is sufficient, and rarely is a greater pressure than three feet advisable. The last is desirable when a thorough flushing of the rhinopharynx is required. The danger of forcing fluid into the middle ear is greatly lessened if the patient keeps the mouth wide open.

Where a smaller amount of fluid is sufficient a piston syringe may be employed. This should be small enough to be easily worked with one hand. It should have a soft rubber tip, to prevent injuring the nasal mucous membrane, and the tip should be large enough to fill the nostril. The piston syringe for nasal use is made either of glass or hard rubber, and fulfils all the conditions mentioned. It is easy of action, can be readily cleansed, and holds about half an ounce. The same syringe should not be used for more than one patient, unless it has been very thoroughly disinfected. In hospitals, and even in private practice, nasal syringes are frequent carriers of infection.

Either of two positions may be used in nasal syringing. In diphtheria, scarlet fever, or any constitutional disease attended by great depression, the child should not be removed from the bed. The syringing may be done by a single nurse, who stands at the head of the bed, alternately syringing the right and left nostril, turning the head from side to side. The other method is to hold the child erect on the lap, with the head inclined somewhat forward, the syringing being done by a second person standing behind. In either position the child's arms and hands should be securely pinioned to the sides by a sheet. To make sure that the rhinopharynx has been reached the water should return through the opposite nostril or the mouth. When properly done, no prostration and very little irritation are caused. The bulb (Davison) syringe should not be employed for nasal irrigation; as the pressure can not be satisfactorily regulated, fluids are likely to be forced into the Eustachian tubes.

Syringing the mouth and pharynx is useful in many pathological conditions of these parts, particularly in children too young to gargle. Either the fountain, piston, or bulb syringe may be used. If the pharynx is to be reached, the nozzle is used as a tongue depressor. This should be placed at the angle of the mouth between the back teeth. The child should lie upon the side or be held in the sitting posture, with the head inclined forward. Only bland solutions should be employed.

Inhalations.—These are of very great utility in all affections of the respiratory tract. To be efficient, the patient should be put under a tent. A satisfactory tent may be made by erecting a T-shaped piece of wood at the head and foot of the crib and throwing over this a large sheet folded and pinned at the corners. Another method is to stretch a cord around the top of each of the four posts of the crib, or simply from the center of the head piece to the center of the foot piece; the sheet should be used as in the first instance. A very good tent may be improvised by throwing a large sheet over an open umbrella. The better the tent the more satisfactory are the results.

Inhalations may be in the form of vapor or spray. The apparatus employed may be the croup kettle, the vaporizer, or the steam atomizer. As all of these are used with alcohol lamps, innumerable accidents from fire have occurred with them. Patients and nurses should always be cautioned regarding this. Whenever possible, the electric heater should be substituted. The ordinary croup kettle is a clumsy affair and especially likely to be the cause of accidents.

There are various forms of apparatus for the purpose of obtaining medicated inhalations.

Stomach-washing or gastric lavage consists in the introduction of water into the stomach through a flexible catheter or stomach tube and then siphoning it out. It is one of the most valuable therapeutic measures we possess. The procedure is very simple, and may be considered entirely free from danger; in fact, it is difficult to pass the tube anywhere else than into the esophagus. The amount of prostration produced by stomach-washing may be compared to that of an ordinary attack of vomiting.

The apparatus for gastric lavage consists of a soft-rubber catheter, size 16, American scale (24 French)—one with a large eye is preferred; a glass funnel, holding four to six ounces; two feet of rubber tubing, and a few inches of glass tubing to join this to the catheter. The child may be held in a sitting posture or placed upon the back; the body should be well protected by a rubber sheet, with a large basin conveniently near. The catheter should be moistened. While the tongue is depressed with the forefinger of the left hand, the catheter is passed rapidly back into the pharynx and down the esophagus. It is important that the first part of the introduction should be as rapid as possible, for if the child begins to gag from the pharyngeal irritation the introduction of the tube may be quite difficult. No resistance is ordinarily encountered after the tube reaches the esophagus. About ten inches of the catheter should be passed beyond the lips. When it has reached the stomach the funnel should be raised as high as possible, to allow the escape of gases almost invariably present. It should then be lowered, in order to siphon out the fluid con-

tents. If nothing escapes, the funnel is then to be raised and from two to six ounces of water poured into it from a pitcher; the funnel is then lowered and the water siphoned out. This procedure is repeated from four to ten times, or until the fluid comes back clear. About a quart of water is ordinarily used. Various solutions have been advised for stomach-washing, but nothing is better than boiled water, used at the temperature of from 100° to 110° F.—the higher temperature being employed when the gastric irritation is very great. If mucus is present in the stomach an alkaline solution (bicarbonate of soda, 5j to Oj) is preferable. Through the tube are easily discharged mucus and small curds; larger ones are gradually broken down by repeated washing. Vomiting may be induced by overdistending the stomach with water. If there is great thirst there is often an advantage in leaving one or two ounces of water in the stomach. To this water it is at times beneficial to add lime water.

Gastric lavage in its application is practically limited to children under two and a half years. It is easiest in those under eighteen months. Children of three years and over are usually so much alarmed and struggle so violently as to make it difficult and undesirable.

The indications for lavage are: (1) Acute gastric indigestion, either with or without persistent vomiting. Here the purpose is simply to clear the stomach of its irritating contents, and a single washing may be sufficient. (2) Chronic indigestion attended by the production of gastric mucus. (3) Dilatation of the stomach. (4) Hypertrophic stenosis of the pylorus. (5) Poisoning.

Gavage.—Gavage consists in the introduction of food into the stomach by a tube passed through the mouth. The same apparatus is employed as in lavage, and the method is similar, with the exception that for gavage the child should be placed upon the back, the head being steadied by an assistant. With older children a mouth-gag is often necessary. After the tube has entered the stomach the funnel should be raised to allow the gas to escape. The food is then poured into the funnel; as soon as it has disappeared the tube is tightly pinched and quickly withdrawn, to prevent food from trickling into the pharynx, since this is often a cause of vomiting. If the food is regurgitated this usually happens at once. It may then be introduced a second time. After feeding, the child should be kept absolutely quiet upon the back.

In cases where all the food is given by gavage the interval between feedings must be considerably longer than under other circumstances. Sometimes the food given may be partially predigested, since digestion in these cases is usually feeble. The stomach should be washed before each feeding, in order to remove mucus and to be sure that it is empty before the meal is given.

Gavage is valuable in the feeding of premature infants and after certain operations upon the mouth and neck. It is also useful, first, in the case of some very young infants, who, suffering from severe malnutrition, can not be induced to take food enough to sustain life; secondly, in many acute diseases, particularly in septic cases where the child will not readily take the necessary food, as in diphtheria, scarlet fever, typhoid, pneumonia, etc.; thirdly, in many cases of cerebral disease where food is refused on account of delirium or coma; and, fourthly, in some cases of persistent vomiting.

Gavage is a very simple procedure and one which a nurse can easily be taught. Not only may food be given, but also medicines and stimulants as may be required, with little or no trouble. The advantage of gavage over the continued coaxing or holding the nose and forcing the patient to swallow, will be at once apparent to one using it.

Nasal Feeding.—The method is similar to gavage, the only difference being that the tube is passed through the nose, and consequently a much smaller one is used. No. 10 American or No. 16 French scale is a proper size. Nasal feeding is applicable to children over two years old, in whom the tube can seldom be passed through the mouth without the use of a gag, and then only after much struggling. It is of value after intubation, tracheotomy, and other operations about the throat, also in some cases of throat paralysis, especially after diphtheria.

Irrigation of the Colon.—By irrigation of the colon is meant the flushing of the entire large intestine by fluids injected high up through a catheter or rectal tube.

The apparatus required for irrigating the colon is a fountain syringe, five or six feet of rubber tubing, and a flexible rectal tube or soft-rubber catheter—No. 26 or 27, French scale, being preferred. Kemp's double-current tube of hard or flexible rubber is useful. The same result can be obtained by using two catheters, the larger for outflow, the smaller for inflow. The child is placed upon the back, with the thighs flexed and the buttocks brought to the edge of the bed or table. He should lie upon a Kelly pad or a rubber sheet so arranged as to form a trough emptying into a large basin or tub. The bag containing the water is hung two or three feet above the bed. If a catheter is used it is inserted just within the sphincter before the water is turned on. As it flows the catheter is gradually pushed upward. The water distending the intestine in advance of the catheter usually makes its introduction quite easy. In Fig. 7 is shown the colon of an infant of six months in position. It is the peculiar curve and the great length of the sigmoid flexure that make the introduction of water difficult, unless the tube is inserted for some distance.

Usually a pint, and sometimes a quart, can be introduced before any

water returns. At least a gallon of water should be used for a single irrigation. The washing should be continued until the water returns quite clean. Change of posture and gentle kneading of the abdomen should be employed during the irrigation, particularly the early part of it, to facilitate the introduction of the water into the upper part of the colon. At the end of the irrigation the rubber tube is detached and the water allowed to escape through the catheter, which remains *in situ*. Sometimes as much as a pint of water remains in the intestine. This is usually passed within half an hour. As the irrigation of the colon almost invariably excites active peristalsis of the lower ileum, this part of the intestine is emptied as well. It is to be remembered that the colon of an infant six months old will hold about one pint without distention, and at the age of two years from two to three pints.

Fig. 7.—Colon of a Child Six Months Old, in Position. (From a photograph.)

Irrigation of the colon is useful to clear this part of the intestine of mucus, fecal matter, undigested food, and decomposing secretions. It may also be employed as a means of local medication in ileocolitis. Where the object is simply to cleanse the intestine, a saline solution—a teaspoonful of common salt to a pint of water—is preferred.

The temperature of the water used for irrigation may be varied according to the special indications. For ordinary purposes, where cleansing only is aimed at, a temperature of from 95° to 100° F. seems to be best. When the body temperature is high, or when there is much pain, tenesmus and straining, colder water has important advantages.

Irrigation under most circumstances is required only once in twenty-four hours. It is important to use a large quantity of water. It must be done thoroughly to be of value, and either by the physician himself or an experienced nurse.

In collapse or great prostration hot saline injections may be employed for purposes of stimulation; the temperature of these should be from 105° to 110° F.

Enemata.—Simple enemata are useful in infants and older children for constipation. When an immediate effect is desired the most efficient is one containing glycerin—e. g., for an infant, one teaspoonful to one ounce of water. Oil enemata (one-half to one ounce) are useful when

the fecal mass is hard and dry and expelled with difficulty. Enemata should always be given with care, and preferably a rubber catheter should be attached to the nozzle of the syringe.

Nutrient enemata have a limited application in infancy, as the rectum soon becomes intolerant. The quantity injected should be small, rarely more than one or two ounces, and the interval between injections should be at least four hours. In older children they may be used as in adults. Glucose can be given in this manner when the stomach is intolerant. It is doubtful if other substances are sufficiently absorbed to be of much benefit.

The administration of drugs *per rectum* is useful in certain cases when, on account of the unpleasant taste or vomiting, the administration by mouth is difficult—e. g., quinin and chloral. As a diluent, gruel is preferable to water. If quinin is used, the bisulphate is the best preparation, but this must be well diluted. The temperature of enemata which are to be retained should be about 100° F. It is necessary in infancy to press the buttocks together for half an hour afterward to prevent the expulsion of the injection.

Hypodermic Medication.—This is not so often used in young children as it should be, and is of the greatest service even in infancy. The use of morphin hypodermically in convulsions, of morphin and atropin in cholera infantum, of strychnin, camphor, caffein, epinephrin, or digitalis in circulatory failure, may be cited as examples.

Hypodermoclysis.—This is a therapeutic measure of much value especially in infants when great loss of fluid has been sustained, as, for instance, in severe diarrhea, or when fluids given by the mouth cannot be retained as in pyloric stenosis. It is at times useful in cases of marasmus when the tissues are dry, shriveled and wasted.

The solution employed is a normal saline (.9 per cent) prepared with sterile or preferably freshly distilled water. The amount injected may be from 100 to 120 c.c. (three or four ounces) to an infant of five or six pounds, and 150 to 250 c.c. (five to eight ounces) to one of nine or ten pounds. It is given once or twice in twenty-four hours. The fluid is contained in an inverted wash bottle suspended a foot or two above the patient and flows through a rubber tube and an ordinary hypodermic needle. The injection may be made into the subcutaneous tissue of any of the large areolar planes of the body, the back between the scapulae, or the abdomen being preferred. The apparatus should be sterilized before using. Before injecting, the solution should be warmed to body temperature and kept warm during injection by wrapping the bottle in flannel. It requires from one-half hour to two hours for the solution to flow into the tissues. Absorption usually takes place in four to six hours. Metabolism experiments have shown that a considerable part not only of

the water but of the salt so given is retained for two or three days by those whose tissues need it most. Healthy infants usually eliminate it very quickly, getting rid of most of it within twenty-four hours. A slight rise of temperature, rarely over 101.5°, occurs a few hours after the injection in about half the cases. Hypodermoclysis may often be repeated with advantage for several days.

Massage.—In older children massage is useful for the same conditions as those for which it is employed in adults; the most important are anemia general malnutrition and chronic constipation. It is necessary that in the beginning only the mildest movements of massage should be employed, and these but for a short time.

In infancy massage has a limited application and it is doubtful whether it really does more than can be accomplished by the general friction of the body. This rubbing, either with the bare hand or with cocoa butter, or with some form of fat, is useful in malnutrition, in rickets, and in wasting diseases when the circulation is feeble and the muscular tone low. Cocoa butter is cleanly and has a pleasant odor, and is, we think, quite as valuable as the more commonly employed cod-liver oil, which is exceedingly disagreeable. The inunctions should be given daily after the morning bath, before an open fire. The rubbing should be continued for fifteen to twenty minutes.

Anesthetics.—As a general anesthetic for routine use, ether is to be recommended for children. Its disadvantages can largely be overcome by proper administration; in point of safety it is immeasurably superior to chloroform for the very young. The administration of ether to young children may be advantageously preceded by a few whiffs of nitrous oxid or ethyl chlorid; both, however, are to be used with caution in infants. Ether should be given slowly, well diluted with air, and if used in this way its unpleasant features may be obviated. This can best be accomplished by the use of some special form of inhaler. Ether should not be selected as the anesthetic for patients suffering from nephritis, bronchitis, pneumonia, pleurisy, or any other disease attended by obstructed respiration.

The dangers from chloroform are greatest when it is given too rapidly or in too concentrated a form. Both are exceedingly likely to occur when it is administered to a struggling child. The greatest care and judgment should be exercised at such times, or disastrous consequences may follow. To produce and maintain the effect desired with the minimum amount of chloroform should always be the aim. All anesthetics, but especially chloroform, are dangerous in children with the so-called lymphatic diathesis. For the removal of tonsils or adenoids, chloroform should not be employed.

Nitrous oxid, while very useful in older children as in adults for

momentary operations, is not well borne by infants. It produces so early and so deep asphyxia that its prolonged use may be fraught with serious danger.

Transfusion.—Two methods of performing transfusion are in use: The first, the end-to-end anastomosis introduced by Carrel, is somewhat difficult of technic and requires a skilled surgeon; second, the syringe method popularized by Lindemann, which is much simpler and can be done by one of very moderate experience. In this the blood is drawn from the vein of the donor, preferably a member of the family, into a paraffin-coated glass syringe and immediately injected into the vein of the child, usually the external jugular, but any available superficial vein may be chosen. In most cases it can be done without any dissection. As the blood must be rapidly passed from one person to another before coagulation takes place, at least one assistant and the use of four or five syringes are needed. The amount of blood usually injected into infants is from two to six ounces.

The indications for transfusion are: first, in any acute hemorrhage, especially the hemorrhages of the newly born, where it is usually a specific remedy and acts at once; secondly, in loss of blood during or after operations. In some types of especially severe secondary anemia it is of benefit. In the slowly developing anemias, whether from disease of the blood-forming organs or as an accompaniment of malnutrition or marasmus, it is of very transient benefit.

PART II

SECTION I

DISEASES OF THE NEWLY BORN

CHAPTER I

ASPHYXIA

The lungs in the full-term fetus are of uniform dark red color, and show very distinctly upon their surface the lobular divisions. They are firm and solid and readily sink in water. The connective tissue is very abundant, and forms distinct fibrous septa, which stretch through the lungs in every direction.

Inflation of the lungs begins with the first cry uttered by the infant as it is born into the world. The parts first expanded are the anterior borders of the lungs, then the upper lobes, and finally the lower lobes posteriorly. The superficial lobules are nearly always expanded before those in the interior of the lung. The inflation is sometimes irregular, because of the accumulation of mucus in some of the bronchial tubes. The right lung is frequently stated to be expanded earlier than the left. Although this is often the case, there is no uniformity in this respect. The important point to be remembered is, that the parts last inflated are the posterior portions of the lower lobes. The expansion of the lungs is a gradual process, and in healthy infants it is probably not complete for two or three days. In delicate children it may be postponed for several days, or even weeks. The above statements are based upon post mortem observations upon infants dying from various causes during the first weeks. It has often been a matter of great surprise to find at autopsy on an infant two or three days old, that less than one-half of the lung tissue was expanded, although the child had breathed well and shown no signs of atelectasis. Under normal conditions at full term inflation of the lungs takes place very readily, but not so readily in premature or delicate infants, on account of the feebleness of the respiratory muscles. The longer it is postponed after birth the more

difficult does it become, on account of the changes which occur in the collapsed air vesicles. The condition of the child *in utero* may be described as one of fetal apnea, its oxygen being received and its carbon dioxid discharged through the placenta, which is essentially the organ of respiration at this period. This condition is interrupted by cutting off the supply of oxygen and the accumulation of carbon dioxid in the blood. Which of these is the important factor in inducing pulmonary respiration has been much debated; but the best experimental evidence seems to show that it is the latter which stimulates the respiratory centers.

Under the term "asphyxia" may be included all cases in which primary respiration is not spontaneously established with sufficient force to maintain life. Usually there is no attempt at pulmonary respiration until after the birth of the child, but it may occur *in utero* or at any stage of parturition. Asphyxia may be of intra-uterine or extra-uterine origin.

Etiology.—1. *Intra-Uterine Asphyxia.*—The maternal causes include any disturbance of the placental circulation during labor—anything which prolongs the second stage of labor, convulsions, hemorrhage, the use of ergot in the second stage, or, finally, the death of the mother. The causes relating to the child are pressure upon the cord, multiple winding of the cord about the neck, early separation of the placenta, and pressure upon the brain. If the respiratory stimulus comes before the birth of the child, the effort at respiration may cause the entrance into the mouth and air passages of amniotic fluid, mucus, blood, meconium, etc.

2. *Extra-Uterine Asphyxia.*—This condition is a much less common one. It arises from causes quite apart from those above mentioned, and depends upon malformations or intra-uterine disease of the organs of respiration, circulation, or of the brain. It may be secondary to an injury of any of these organs received during parturition. It is also seen in premature infants, where it depends upon the feeble development of the nerve centers and respiratory muscles and upon the soft, yielding chest walls.

Lesions.—In infants dying of intra-uterine asphyxia there are seen the usual changes found in death from suffocation, together with the effects of attempts at breathing *in utero*. There is general congestion of all the viscera, particularly of the brain and its meninges, the liver, and the lungs. They may show small, punctate hemorrhages, and occasionally large extravasations. Blood or bloody serum may be found in any of the serous cavities. The right heart is overdistended with dark, soft clots, and the blood generally is more fluid than normal. The lungs may contain no air, but more frequently there are small, scattered areas in which lobular inflation has taken place. If the child has lived several hours there are larger areas of expanded lung, especially in the upper

lobes, and these may even be emphysematous, if artificial inflation has been employed. In the mouth, nose, larynx, and even as far as the finest bronchi, there may be found aspirated materials—amniotic fluid, blood, mucus, or meconium. In extra-uterine asphyxia there may be organic changes in the viscera—malformations of the lungs or the heart, intra-uterine pneumonia or pleuritic effusion, malformation of the diaphragm and sometimes of the brain.

Symptoms.—Under normal conditions the newly-born infant begins at once to scream and to use his limbs, the purplish color of the skin giving place in a few moments to a rosy pink. In the first degree of asphyxia—*asphyxia livida*—the child is deeply cyanosed. Either no attempt whatever is made at respiration, or it is superficial and repeated only at long intervals. The pulse is slow, full, and strong. The vessels of the cord are distended. Muscular tone is preserved, and also cutaneous irritability, so that with the application of almost any kind of external stimulus respiration is excited and the symptoms disappear.

In the second degree—*asphyxia pallida*—the picture is quite a different one. The face is pale and deathlike, though the lips may still be blue. The heart's action is weak, and by palpation can rarely be felt at all. By auscultation the sounds are feeble, irregular, and usually slow. The cord is soft, pale, and flaccid, and its vessels nearly empty. The sphincters are relaxed and meconium oozes from the anus. There is entire loss of tone in the voluntary muscles, so that the extremities and entire body seem perfectly limp. Cutaneous sensibility is abolished. The extremities are often cold. There may occur a few short, convulsive contractions of the respiratory muscles, but these are without effect and soon cease. Unless such cases receive the most prompt and efficient treatment, the heart's action becomes more and more feeble until it ceases and death occurs. Other cases are partly resuscitated and may survive for a few hours or days, when they gradually sink, respiration becoming more and more feeble in spite of all efforts to maintain it. Between these two extremes all degrees of severity are seen.

In extra-uterine asphyxia there may be some attempts at voluntary respiration continuing for several hours, sometimes for a day or two, but this may be inadequate to sustain life.

Diagnosis.—Almost the only condition with which asphyxia is likely to be confounded is cerebral compression from a meningeal hemorrhage. The difficulties in the case are much increased by the fact that the two conditions are not infrequently associated. It may then be impossible to tell that in addition to asphyxia, intracranial hemorrhage is present. If the hemorrhage is extensive and the asphyxia only moderate, a diagnosis is possible in most of the cases. In hemorrhage there is often a history of undue compression during delivery—sometimes the use of forceps.

The fontanel is bulging; there is coma, and there may be paralysis. The respiratory murmur may be quite strong for several hours, but it gradually fails as the child becomes completely comatose. Anemia resulting from a large hemorrhage, like that due to rupture of the cord, may simulate the severe form of asphyxia.

Prognosis.—This depends upon the grade of asphyxia and the treatment employed. There is but little tendency to spontaneous recovery in any form. In the milder cases recovery is almost invariable with any intelligent treatment. In the severest cases the outcome is always doubtful, although by persistent effort many infants that are apparently hopeless may be saved. In a prognosis as to the ultimate result, the frequent complication of asphyxia with meningeal hemorrhage should always be kept in mind. Apart from this complication it is doubtful whether asphyxia has anything to do with the production of idiocy.

Treatment.—In every case the first step is to clear the mouth and pharynx of mucus by means of the finger covered with absorbent cotton. In the milder forms respiration is usually excited either by spanking the child or the alternate use of hot and cold baths. If the hot bath is employed, the water should be from 104° to 108° F. and always tested by a thermometer. After a moment the child should be dipped into very cold water, or the body may be douched with it. In the livid cases relief is often afforded by allowing the cord to bleed for a few moments before ligation. The loss of half an ounce of blood is ordinarily sufficient. Simply swinging the child in the air is a powerful stimulus to respiration. The above means will suffice in the great majority of cases. In the more severe forms, however, these are inadequate. There is no response whatever to external stimulation, either by heat or mechanical irritation. In these cases two methods of resuscitation may be employed: artificial respiration and direct inflation of the lungs.

One of the most widely employed methods of inducing artificial respiration is that of Schultze. The infant is grasped by both axillae in such a way that the thumbs of the physician rest upon the anterior surface of the chest, the index fingers in the axillae, and the remaining fingers extending across the back. The child is thus suspended at arm's length between the knees of the physician, the feet downward and the face anterior. The body is now swung forward and upward, until the physician's arms are nearly horizontal. This produces the inspiratory effort. When this point is reached, an arrest in the swinging causes flexion of the trunk, the head now being directed downward, the lower extremities fall toward the physician until the whole weight of the body rests upon the thumbs. In this way expiration is produced. Lusk cautions against the employment of this method if the heart's action is very feeble, as it may cause the heart to stop altogether. This method should

be used with care and skill; clumsy and too forcible manipulation has resulted in many serious injuries to the viscera and fractures of ribs or clavicles.

A method introduced by Dew has been extensively employed in New York. The infant is grasped in such a way that the neck rests between the thumb and forefinger of the left hand, the head being allowed to fall far backward, the upper portion of the back resting upon the palm of the hand; with the right hand the knees are grasped between the thumb and fingers, the thighs resting against the palm of the hand. Inspiration is produced by depressing the pelvis and lower extremities, thus causing the abdominal organs to drag upon the diaphragm, and at the same time gently bending the dorsal region of the spine backward. In expiration the movement is reversed, the head being brought forward and flexed upon the thorax, while at the same time the thighs are flexed so as to bring them against the abdomen. The body is thus alternately folded upon itself and unfolded as the movements are carried on. If there is much mucus in the mouth, the movement of expiration should first be made with the body completely inverted. This method is simple, efficient, and much less fatiguing than that of Schultze when it is to be maintained for a long time. It is also of great advantage in that it can be carried on while the child is in the hot bath, one of the greatest objections to the method of Schultze being the loss of animal heat incident to its use.

In all cases where artificial respiration is used the first movement should be that of expiration, to expel, so far as possible, mucus or other foreign substances from the air passages. The movements should be made from eight to twelve times a minute, and not too forcibly, the child being kept in the hot bath between the movements, and as much as possible during them. As long as the heart beats resuscitation is possible, and the case should not be abandoned.

Direct inflation of the lungs by the mouth-to-mouth method should not be employed.

An ingenious apparatus for artificial inflation of the lungs has been devised by Carrel of the Rockefeller Institute, making use of Meltzer's method of the continuous insufflation of air. A flexible catheter containing a wire stylet is introduced into the larynx. By means of a double bulb a continuous flow of air is maintained. A manometer measures the pressure employed and is a guide by which one is prevented from using an excessive amount of force. When the pressure employed is normal the mercury in the descending and ascending arms of the curved tube stands at about the same level; if an excessive amount of pressure is used, the mercury will be forced up into the bulb. Although this has been very little employed in infants it has been extensively used in resuscitating

animals and seems to fulfill all the indications better than any apparatus hitherto suggested. It is so simple of construction that it can easily be put together by any instrument maker.

The method introduced by Laborde, of making rhythmical traction upon the tongue ten or twelve times a minute as a means of exciting respiration, is sometimes very useful in conjunction with other methods. Faradization of the phrenic is of undoubted value, but somewhat difficult of application.

In cases of asphyxia it is not enough to make the child cry. The deep respirations should be made to continue, for very often it happens that resuscitation is only partial, and that the child after six or eight hours lapses into its previous condition. All severe cases require close watching for the first twenty-four or thirty-six hours, as a repetition of the treatment is often necessary.

CHAPTER II

CONGENITAL ATELECTASIS

This condition is one in which there is a persistence of the fetal state in the whole or in any part of the lung.

Atelectasis is the pathological condition with which asphyxia of the newly born is usually associated. In most of the cases the condition of atelectasis is completely overcome by the means employed in resuscitation; in some, however, these means are only partially successful, so that a portion of lung of variable extent remains in the fetal condition. These are the circumstances in which most of the cases of atelectasis arise. But there are others in which there is no history of early asphyxia, where the primary respirations, although taking place spontaneously, have not been of sufficient force and depth to produce full pulmonary expansion. This usually occurs in feeble infants, or in those who are premature. The causes of congenital atelectasis are therefore, in the main, those mentioned as producing asphyxia.

Lesions.—In cases where the child dies during the first few days the amount of expanded lung is often small, frequently not more than one fourth of the pulmonary area. The expanded portion is usually the anterior borders of the upper lobes. This is often the seat of acute emphysema. The rest of the lung is still in the fetal state; it is of a brownish-red color, very vascular, does not crepitate, and shows the lobular outlines both on the surface and on section. With a little force the atelectatic lung may be completely inflated.

If children have lived a longer time, nearly the whole of the upper lobes and the anterior portion of the lower lobes are usually well inflated. These portions are either normal or slightly emphysematous. The posterior portion of the upper lobes and the lower lobes are almost invariably the seat of the atelectasis. On the surface even these portions may present quite a large area of expanded vesicles, but the underlying portion may be solid to the touch, and crepitates but slightly. On section it is seen that only the most superficial part of the lung is inflated, while the interior of the lobe is unexpanded. Small hemorrhages are frequently seen beneath the pleura.

It is usual for both lungs to be affected, and often, but by no means uniformly, to about the same degree. It is frequently a great surprise to discover that a child has lived for some weeks without presenting any signs of cyanosis, although using not more than one-third of his pulmonary area. This variety of atelectasis closely resembles the hypostatic pneumonia of delicate infants, and very often the two conditions are associated. It may require the microscope to decide between them. If congenital atelectasis has existed for a considerable time, there are usually found evidences of pneumonia. Inflation is not so easy as in recent cases, but with force the greater part of the lung can usually be expanded. The heart commonly shows the right auricle and ventricle to be distended with dark clots, and there is occasionally found a patent foramen ovale or some other form of congenital lesion. The liver and spleen are in most cases congested, and the spleen may be considerably enlarged. The mucous membrane of the stomach and intestines is sometimes deeply congested.

Symptoms.—In one group of cases the children are asphyxiated at birth, but the attempts at resuscitation have been only partially successful. Although the patients may live for a few days, there is cyanosis, which gradually deepens, and death takes place from asphyxia, exhaustion, or convulsions.

In a second group of cases the infants have been asphyxiated at birth, and resuscitated perhaps with difficulty, but to all appearance completely. They do not thrive, however, remaining small and delicate, gaining very little or not at all in weight, and showing poor circulation, cold extremities, and occasionally subnormal temperature. It is characteristic of these cases that the cry is never loud, strong, and lusty. Some of them will not cry at all. Such children may live several weeks. There may develop at any time, often quite suddenly and without assignable cause, attacks of cyanosis with prostration. Children may have several such attacks, which do not excite suspicion since they pass away spontaneously. In other cases the symptoms are so severe that they may result fatally in a few hours, death being frequently preceded by convulsions. If ener-

getically treated the symptoms may pass away but, reappearing in a few hours, or again after a week or more, they gradually deepen in intensity until death occurs.

Two cases that came under our observation in the New York Infant Asylum illustrate this point: The infants were twins, ten weeks old and delicate. Suddenly at night one child was taken with convulsions, became deeply cyanosed, and died in two and a half hours. He had been suffering from a slight attack of indigestion for a week previous. The other twin had been apparently well on the previous day. Two hours after the death of the first child the second was taken with similar symptoms, dying in a few hours. At autopsy there was found very extensive atelectasis involving the posterior part of the upper and the greater part of both lower lobes. The lesions were almost identical in the two cases. In both, the stomach was greatly distended with food and gas. We have repeatedly seen the effect of overdistention of the stomach in producing cyanosis in young children, and in this instance we believe it to have been the exciting cause of the final symptoms. It was subsequently learned that during the six weeks of observation the nurse had witnessed several slight attacks of cyanosis in one of the infants. It is of course possible that the atelectasis in these cases may have been in part at least acquired.

We have seen a number of cases, in which there was nothing whatever to attract attention to the lungs until the final attack of cyanosis occurred. In not all of these cases is there a history of asphyxia at birth. Some are only puny, delicate or premature, exhibiting during the early weeks of life all the signs of feeble vitality. The subsequent course is the same as in those in which there is early asphyxia. The duration of life in these cases depends chiefly upon the extent of the atelectasis.

It is not to be supposed that all cases of congenital atelectasis terminate fatally. Infants in whom there is every reason to believe that atelectasis exists, from the occasional attacks during the first few weeks of cyanosis, feeble cry, poor circulation, etc., may under favorable conditions with improved nutrition recover completely, even though no special treatment is directed to the lungs.

Diagnosis.—The physical signs are of much less value than the symptoms. It should be remembered that the principal seat of the disease is the lower lobes posteriorly. Percussion usually gives resonance over the entire chest, although this may be somewhat diminished posteriorly. There is not, however, so much change as one would expect to find, for the collapsed areas are surrounded by others which are overdistended, and there are in the midst of the collapsed parts, especially upon the surface, lobules which are inflated. If the two sides are involved to about the same degree, as is often the case, we can get no difference in the percussion note over the two lungs, and the change from the normal may be so

slight as not to be appreciable. Where only one lung is affected a difference can usually be made out. The respiratory murmur is rarely bronchial, but generally only feeble in its intensity, and rather ruder in quality than normal. The cardiac sounds may be transmitted with abnormal intensity. As in the case of percussion, if only one lung is affected, this is of some value in diagnosis, but it is not sufficiently marked to be readily recognized when both sides are involved. Occasionally râles are present.

Treatment.—In the newly-born child, whether asphyxiated or not, the physician should see to it that the infant not only cries, but does so loudly and strongly, and that this cry is repeated every day. If children do not cry naturally they must be made to do so by the alternate use of the hot and cold bath, as in cases of asphyxia, or by mechanical means, like spanking. This should be repeated at least twice a day, and continued for from fifteen to thirty minutes. It may seem cruel but it is often the only means of saving life. Expansion of the lungs is much more easily induced during the first few days of life, becoming more and more difficult the longer it is delayed. Provided the condition is recognized, treatment is fairly successful. In institutions where delicate infants spend most of the time in their cribs, atelectasis is likely to be found. An infant needs exercise, and this is often only to be obtained by taking the child from its crib several times a day, by general friction, massage, the stimulus of fresh air, etc. Nothing is more certain to perpetuate atelectasis than to allow the infant a life of feeble vegetative existence. Food and feeding must be carefully attended to, but even these are of less importance than the maintenance of the animal heat. The temperature is often subnormal, and should be closely watched. If there is difficulty in keeping the child warm he should be rolled in cotton and surrounded by hot bottles, or kept in an incubator during the first few weeks. During attacks of cyanosis the same means are to be employed as in cases of asphyxia of the newly born—cutaneous stimulation and artificial respiration—the administration of drugs being of little or no value, but oxygen may be of assistance.

CHAPTER III

ICTERUS

Several varieties of icterus are met with in the newly born.

1. It is often seen in the various forms of pyogenic infection. In such cases the icterus is usually mild.

2. It may be due to congenital malformations of the bile-ducts.

3. It may depend upon interstitial hepatitis.

4. The most frequent of all varieties is the so-called idiopathic icterus, sometimes spoken of as physiological icterus.

In the cases included under the first head icterus is a minor symptom. The other varieties are sufficiently important to require separate consideration.

Malformations of the Bile-ducts.—The common bile-duct is the most frequently affected. There may be atresia at the point where it opens into the intestine, the duct may be represented by a fibrous cord, or it may be absent altogether. In many cases this is the only lesion; in others it is associated with an impervious hepatic or cystic duct; in still others the common duct is normal, but the cystic or hepatic ducts are impervious.

At autopsy all the organs are usually found intensely jaundiced, particularly the liver. In recent cases this is very much swollen, but presents no marked organic changes. In cases which have lasted several months there is commonly found chronic interstitial hepatitis, sometimes to a very marked degree. This was present in nine of the fifty cases collected by Thomson. The gall-bladder is usually small, and often rudimentary. In cases of atresia of the common duct it may be greatly distended.

The condition of the bile-ducts is ascribed to an error in development and subsequent catarrhal inflammation. There does not seem to be sufficient evidence to prove that hereditary syphilis is an etiological factor of much importance. This was present in but five of Thomson's cases.

Symptoms.—The most striking symptom is jaundice, which is usually noticed a day or two after birth, and steadily increases until it becomes intense. The other symptoms of obstructive jaundice are present. The urine is colored a dark brown or bronze by bile pigment, the stools are white, and bile pigment is absent or present only in traces, except in cases where malformation is limited to the cystic duct. The liver as a rule is much enlarged. The spleen is often swollen. Hemorrhages beneath the skin or from any of the mucous membranes are quite common. Vomiting is usually absent. In most cases there is progressive wasting, and death from inanition within the first few weeks. Of Thomson's fifty cases, nine lived less than a month, and only eighteen over four months. Lotze has reported a case of a child living eight months with an impervious hepatic duct. A frequent cause of death in the more rapid cases is convulsions.

These malformations cannot be influenced by any treatment.

Interstitial Hepatitis.—There is seen in newly-born children a form of icterus which resembles the foregoing in many particulars, but which

may end in recovery. In three such cases which have terminated fatally we have found the lesions of a general interstitial hepatitis, presumably of syphilitic origin. It is not certain that syphilis is always the cause of this condition, for the clinical history in some of them gives no evidence of this disease. While not a common condition we believe it to be more frequent than congenital malformations of the bile-ducts with which it is often confounded.

The symptoms and course may be illustrated by the following cases: A full-term, well-developed child of eight pounds' weight became jaundiced on the second day. By the fifth day the jaundice was intense; stools, pale yellow, and urine deeply bile-stained. Examination at three weeks showed both liver and spleen much enlarged. The jaundice was very marked for over a month; it was nearly two months before it faded entirely. The nutrition of the child was a matter of much difficulty for several weeks. The enlargement of the spleen and liver like the jaundice disappeared very gradually. There was no other evidence of syphilis in this patient nor in the two other children of the family, and no history of this disease could be obtained in the parents. Yet the improvement which began with the use of mercurial inunctions strongly suggested a syphilitic lesion.

In another case, the symptoms and course of which were almost identical, the stools, though nearly white, never failed to give the reaction for bile. A previous child in this family had died three years before at the age of six weeks with persistent jaundice, which had been diagnosticated congenital malformation of the bile-duct. There was no history of syphilis; but the mercurial inunctions seemed equally efficacious as in the first case cited.

Not much need be added to the symptoms described. In our cases which recovered and in the fatal cases there was no fever and no ascites; but there was much tympanites. The application of the Wassermann test will no doubt aid in clearing up the etiology of these cases. Other evidences of syphilis should always be carefully sought, but in all the cases we have seen, even those ending fatally and with syphilitic lesions at autopsy, clinical evidence of syphilis during life was wanting. A careful trial of antisyphilitic treatment should, therefore, be made in every case of protracted jaundice in a newly-born child. One should not be too ready to make the diagnosis of malformation of the bile-ducts and regard the case as hopeless. Nor does the fact that the child recovers without antisyphilitic treatment exclude syphilis as the cause, for one of Still's cases recovered from the jaundice and died at the age of nineteen months, the autopsy showing lesions evidently syphilitic.

Physiological or Idiopathic Icterus.—In 900 consecutive births at the Sloane Hospital for Women icterus was noted in 300 cases. In 88 it was

intense, in 212 it was mild. According to the statistics of various lying-in hospitals of Germany, it was found in from 40 to 80 per cent of all infants. In the 300 cases just referred to, icterus was noticed on the first day in 4, on the second day in 19, on the third day in 72, on the fourth day in 86, on the fifth day in 67, and on or after the sixth day in 44. From the second to the fifth day is therefore the usual period for its appearance.

It usually increases in severity for one or two days and then slowly disappears. The average duration in the mild cases is three or four days; in those of moderate severity about a week; in the most severe cases it lasts many weeks. Icterus neonatorum is regularly found in premature and very delicate infants. The course with them is also more prolonged and the icterus usually more severe.

The icterus is first noticed in the skin of the face and chest, then in the conjunctivae, then in the extremities. The skin varies in color from a pale to an intense yellow. The urine in most cases is normal. It sometimes is of a light brown color, and only in the most severe cases does it contain bile pigment in appreciable amount. The stools are unchanged, the normal yellow evacuations occurring in the icteric as early as in those not affected.

According to some observers, in infants who are icteric the initial loss in weight is greater and the subsequent gain slower than in other children. This is not borne out by the Sloane statistics. The proportion of icteric infants who did well, moderately and badly, was practically the same as of the other children in the institution not suffering from icterus. Icterus occurs with equal frequency in both sexes. There are usually no other symptoms than icterus, and the condition is practically never serious, though a prolonged course may occasion some concern. With the premature and poorly nourished it is the general condition and not the icterus that is dangerous. Very rarely a severe and fatal form of icterus is seen affecting successively several infants in a family. Death takes place in a few days without sufficient pathological evidence to explain the cause.

In jaundiced infants who have died from accident or other causes the skin and almost all the internal organs are found icteric. There is staining of the internal coat of the arteries, the endocardium, the pericardium and the pericardial fluid. The subcutaneous connective tissue is yellow; the spleen and kidneys only in the severe cases. The liver is slightly discolored. The bile ducts are normal. There may be small hemorrhages, especially on the serous surfaces. The brain and cord are rarely, and the cerebrospinal fluid never, bile stained.

Few subjects have given rise to wider speculation than this form of icterus. It has been held that it is due to obstruction from thick bile in

the bile ducts, to extensive blood changes, and to various other causes.

The researches of Yllpö have shown that in the last month of fetal life there is an increased production of bile pigment. Even at birth the blood contains three or four times the amount that the maternal blood contains. After birth there is a very rapid increase in the pigment content of the blood which usually lasts from three days to a week; exceptionally for several weeks. At the end of a few days the blood may contain twenty times as much pigment as at birth. Usually after a few days the pigment in the blood diminishes, rapidly at first, then more slowly. The normal is not reached for several weeks. All infants show this increased amount of bile pigment. Those that subsequently develop icterus have at birth a greater bilirubin content in the blood and also produce more pigment subsequently. Icterus is noticeable when the blood contains, roughly, 125 mgm. of pigment to each 100 cc. of blood. The cause of the increased production of pigment is not entirely clear. There is not yet sufficient evidence that it is due to the destruction of the red blood cells. Only a slight amount of the pigment can be excreted by the kidney. It is most probable that the liver at this early stage of development is unable to remove the excess of pigment from the blood. This accumulates and when it reaches a certain concentration in the blood, causes appreciable icterus. With the cessation of the blood destruction and the increase in functional activity of the liver, the pigment is removed. The difference in the icteric and the non-icteric infant is one only of degree. It is quite proper in such circumstances that the condition should be spoken of as "physiological icterus."

Diagnosis of the Different Varieties of Icterus.—The diagnosis of physiological icterus is to be made from malformations of the bile-ducts, and interstitial hepatitis. In early sepsis it is doubtful if the infection produces the icterus. It is more likely that the two conditions are associated. In the later sepsis jaundice may be due to an hepatic lesion, usually multiple abscesses. In malformations of the bile-ducts the icterus is usually more intense and appears almost immediately after birth; bile is absent from the stools; the icterus is persistent, and the symptoms go progressively from bad to worse, always ending fatally. In interstitial hepatitis the icterus develops at about the same time as, but is generally more marked than, in the physiological variety. Both liver and spleen are usually enlarged. The stools may be light colored, but still give a faint bile reaction.

Physiological icterus requires no treatment.

CHAPTER IV

THE ACUTE INFECTIONS OF THE NEWLY BORN

It is possible for the newly-born infant to suffer from almost any of the common infectious diseases. Smallpox probably has been most frequently observed. Rarely pertussis, influenza, measles, typhoid fever, malaria, and pneumonia have occurred in the first days of life. As the mothers in many instances were suffering from the diseases during or just prior to delivery, the infants appear to have been infected before birth through the circulation of the mother. In other cases, especially in pneumonia, influenza, and gastro-enteritis, infection may take place soon after birth. The symptoms of these diseases in the newly born differ very little from those occurring in any other young infant. In addition to the diseases mentioned, there are other forms of infection which belong especially—some of them exclusively—to the newly born.

THE ACUTE PYOGENIC DISEASES

Under this head are grouped various infections of the newly born, due to the entrance of the common pyogenic bacteria. They have been designated as *puerperal fever of the child,* also as *pyemia* or *septicemia,* or simply as *sepsis of the newly born.* A variety of pathological and clinical conditions are met with. In some cases there is only a localized external inflammation, often terminating in abscess formation; sometimes one or more of the internal organs is affected; occasionally a general blood infection—a true septicemia—is seen without any noteworthy local lesion; finally, there are the cases attended by the production of multiple abscesses in the viscera, joints, or cellular tissue —a true pyemia. Formerly infections of this class were very common, especially in large lying-in hospitals; but, owing to the general adoption of the methods of aseptic midwifery, they have steadily diminished.

Etiology.—The source of infection of the child may be the vaginal secretion of the mother or, in very rare cases, the mother's milk. Although it has been shown that in a great proportion of the cases the milk of a woman suffering from mastitis or from septicemia contains pyogenic germs, still the taking of these into the stomach is not likely to infect the infant. More frequently the child is infected by the nurse in the process of dressing the cord, bathing, or cleansing the mouth or eyes, possibly after having attended to the needs of a septic mother or another

child. Infection may be carried by the physician, by instruments, or by the dressings of the cord. Infection may occur through any wound or abrasion of the skin.

Infection through the umbilicus may take place either before or after the separation of the cord. The infection may take place through the umbilicus, yet this may give no external evidence of disease, although the umbilical vessels inside the body may contain pus. From this focus of infection may arise peritonitis, meningitis, or other inflammations. Entering through the mouth, bacteria may lead to infectious processes in the throat, the stomach or intestines, and rapidly produce death; or the alimentary tract may be the focus from which infection of distant parts may arise.

The microörganisms chiefly concerned in these infections are the common pyogenic bacteria, staphylococcus pyogenes aureus and the streptococcus. The next in importance is the gonococcus, the rôle of which, especially in cases accompanied by joint suppuration, has only recently been appreciated. Pneumococcus infections occasionally complicate the others mentioned. While streptococcus infections are in general more serious than those due to the staphylococcus, some of the most severe ones met with belong to the latter class.

Clinical Varieties.—*Omphalitis.*—In this variety there is inflammation of the umbilicus, and cellulitis of the abdominal wall in the immediate neighborhood. This results in the formation of an umbilical phlegmon. It may terminate in resolution, in abscess, or in gangrene. The usual termination is in abscess. These abscesses may be small and superficial, or they may be more deeply seated between the abdominal muscles and the peritoneum. Omphalitis usually begins in the second or third week of life, before the umbilicus has cicatrized. The process may result in erysipelatous inflammation and it may spread to the peritoneum.

Inflammation of the Umbilical Vessels.—This is one of the most frequent primary processes in pyemic infection. The umbilical arteries are more frequently involved than the vein. According to Runge, inflammation of the vessels is always preceded by inflammation of the connective tissue which surrounds them, as the poison is taken up by the lymphatics and not by the blood-vessels. Omphalitis is frequently present, but in some cases the umbilicus shows nothing abnormal.

In arteritis the vessels may be involved to any degree: sometimes only a short distance from the abdominal wall, sometimes quite to the liver. They contain pus, and often septic thrombi. Saccular dilatation is frequently present at several points. Pus sometimes exudes from the umbilical stump on pressure. The other lesions accompanying arteritis are those of pyemic infection, more or less widely distributed.

There are frequently present peritonitis, suppuration of the joints, erysipelas, multiple abscesses of the cellular tissue, sometimes suppurative parotitis. Atelectasis is common. Pneumonia was found in twenty-two of Runge's fifty-five cases.

In cases of phlebitis, the umbilical vein is usually involved for its entire length from the abdominal wall to the liver. This may lead to an acute interstitial hepatitis going on to suppuration, or to phlebitis of the portal vein and some of its branches. In either case there is more or less parenchymatous hepatitis, and often multiple abscesses of the liver, most of the patients being jaundiced. Peritonitis also is a frequent complication.

Peritonitis.—This is one of the most frequent pathological processes in pyemic infection, and is very often the cause of death. It is generally associated with umbilical arteritis, and often with erysipelas. In a considerable number of cases it is the most important lesion found. It may be localized or general. Localized peritonitis is generally in the neighborhood of the umbilicus or of the liver. It may result in adhesions, or in the formation of peritoneal abscesses. More frequently the peritonitis is general and resembles the septic peritonitis of adults. There is a great outpouring of fibrin coating the intestines and other viscera and the inner surface of the abdominal wall, causing adhesions between the abdominal contents. Collections of sero-pus are found in the pelvis and in various pockets formed by the adhesions. Sometimes blood is present in the exudation.

The special symptoms which indicate peritonitis are vomiting, abdominal tenderness and distention, and protrusion of the umbilicus. The abdominal enlargement is chiefly from gas, but may be partly from fluid. There are present thoracic respiration, dorsal decubitus, flexion of the thighs and fixation of all the muscles, the child lying perfectly quiet. The temperature is usually but not necessarily high. Marked leucocytosis is generally present.

Pneumonia.—The most common form seen is pleuropneumonia. There is an abundant exudate of grayish-yellow fibrin covering the lung. Occasionally collections of pus are found in the sacs formed by the adhesions. Serous effusions are rare. The pulmonary lesion consists usually in a bronchopneumonia, with consolidation of larger or smaller areas in the lungs—more often in the upper than in the lower lobes. It is not uncommon for minute abscesses to be found in the lung at various points. There is a purulent bronchitis of the larger and smaller tubes.

The symptoms are obscure and often indefinite. The only characteristic ones are cyanosis and rapid respiration, with recession of the chest walls on inspiration. The physical signs are inconstant and uncertain.

Pneumonia often can not be diagnosticated during life. In most of the fatal cases of pyogenic infection, whatever its type, there is found some involvement of the lungs. The changes are most extensive in cases in which the serous membranes are involved.

Pericarditis is rare and usually associated with pleurisy. Endocarditis is very rare. Hirst has, however, reported a case.

Meningitis.—When meningitis is present it is often associated with peritonitis or with pleurisy. The lesions are those of acute purulent meningitis with a copious exudation, sometimes associated with meningeal hemorrhages, or with acute encephalitis and the production of multiple minute abscesses in the cortex. The local symptoms are often not marked, and are sometimes very obscure. The most characteristic are stupor, dilated pupils, opisthotonus, bulging fontanel, general rigidity, convulsions, and occasionally localized paralyses. The temperature is generally high. A positive diagnosis can generally be made by lumbar puncture, by which means also the exciting cause of the meningitis can usually be determined.

Gastro-enteritis.—Diarrhea is a frequent symptom in all septic cases, constipation being rarely present. In many instances vomiting is a prominent symptom. In a small proportion of cases the most important local lesions are in the intestines, generally in the nature of a superficial catarrhal inflammation.

Stomatitis.—Infections of the oral mucous membranes are not infrequent but sometimes very severe. They may be due to the streptococcus, staphylococcus aureus or the gonococcus. An occasional complication of oral infections is abscess of the parotid.

Osteomyelitis.—Allard has reported a series of cases in which, after the general and local symptoms of pyogenic infection had existed for some time, suppuration occurred over various bones, especially the humerus, tibia, metatarsal bones, sacrum, etc. Trephining revealed the lesions of osteomyelitis. The abscesses usually made their appearance between the fourth and the sixth week. The most rapid case terminated fatally on the fourteenth day, and none lasted more than two and a half months.

Joint Suppuration.—In certain pyemic cases, and in some in which there are no other symptoms, acute suppuration in the joints occurs. This may come on very acutely in the first or second week, or more slowly as late as the second or third month. In the acute cases it is exceptional to have but one joint involved; frequently there are four or five. The small joints are rather oftener affected than the large ones, but almost any articulation in the body may be involved. With multiple joint suppuration there are present the general symptoms of pyemia —high temperature, marked prostration, wasting, and usually secondary

visceral inflammations develop. In those which occur late, or which develop more slowly, fewer joints are involved, often but a single one, the febrile symptoms are less marked or wanting. In our own experience, the organism most frequently found in these cases is the gonococcus; next to this in importance is the streptococcus and occasionally the pneumococcus is found. The joint lesion is usually a superficial one, the bones generally escaping. The gonococcus cases probably occur most frequently as a complication of ophthalmia; but we have seen several in which ophthalmia was not present and where the point of entry could not be determined.

Many of the abscesses supposed to be in the joints are shown at operation to be at the epiphyses; from this source the joints may be involved secondarily. A point to be remembered in the diagnosis of these joint inflammations is their resemblance to the epiphysitis of hereditary syphilis and other symptoms of that disease should be looked for. The confusion is increased by the fact that in syphilitic cases abscesses may follow as a consequence of a secondary infection.

Abscesses in the Cellular Tissue.—These are quite frequent, and may occur with suppuration in the joints or the internal organs, or they may exist as the only lesion. They are nearly always multiple and may be found in almost any location. They vary in size from one containing a few drops to half an ounce of pus. They are due to the introduction of pyogenic germs, usually staphylococci. Their course is benign, and they require no treatment except incision and cleanliness. When there is a disposition to their continued formation, the skin should be washed with an antiseptic solution and vaccines should be administered.

Erysipelas.—This is seen especially during the first two weeks of life and usually starts from the umbilicus or some abrasion of the skin, most frequently about the genitals or the scalp. When originating at the umbilicus it is generally complicated by other lesions, such as peritonitis and umbilical phlebitis. If it starts from any other part of the body it may be uncomplicated. Erysipelas beginning at the umbilicus gives rise to an area of induration and a circumscribed erythema. At first it may resemble a simple cellulitis; but the steadily increasing area of elevated induration and redness soon indicates the nature of the inflammation. From whatever point starting, the erysipelatous inflammation, owing to the feeble resistance of the tissues, in most cases spreads widely. The entire abdomen, chest, and back may be involved, and it may even spread to the extremities. Nearly the whole trunk may be affected in four or five days. It usually involves only the skin and superficial cellular tissue; but it may involve the deeper areolar planes and terminate in diffuse suppuration, or even in gangrene.

The constitutional symptoms are severe: great prostration, continu-

ously high temperature—102° to 105° F.—rapid wasting, and frequently vomiting, diarrhea, or convulsions are present. The disease is always serious, and usually fatal. It is often complicated by bronchopneumonia. General edema of the affected parts may persist for a few weeks after the inflammation subsides.

Distribution of the Lesions.—The frequency of the different visceral lesions in eighty-seven autopsies published by Bednar was as follows: Peritonitis in twenty-nine, pneumonia in fifteen, pleurisy in ten, meningitis in nine, meningeal hemorrhage in eight, encephalitis in eight, cerebral hemorrhage in four, enterocolitis in five, pericarditis in four. In thirty-one cases there was umbilical arteritis, and in nine cases umbilical phlebitis. There was one case each of pulmonary hemorrhage, pleural hemorrhage, acute hydrocephalus, acute bronchitis, and suppuration in the cellular tissue. Runge's later observations of thirty-six cases showed umbilical arteritis in thirty, umbilical phlebitis in three, and normal umbilicus in three. He found pneumonia in twenty-two of fifty-five cases. Other lesions frequently associated are atelectasis, swelling and softening of the spleen, cloudy swelling of the liver and kidneys, occasionally with foci of suppuration in these organs.

General Symptoms.—These may begin at any time during the first ten days—very rarely after the twelfth day. Fever is an exceedingly variable symptom—it may be very high; it may be almost absent; occasionally there is subnormal temperature. The course of the temperature is very irregular. Wasting is constant and quite rapid. It depends upon the inability to take and digest food, upon the intestinal complications, and upon infection. In quite a number of cases wasting is almost the only symptom. Icterus is common; in many of the worst cases it is intense. It is met with where the liver is the seat of an acute parenchymatous or acute suppurative inflammation, and in many other cases where it depends apparently upon the blood changes. Hemorrhages are common, and may be the direct cause of death. They may come from the umbilicus, the intestine, or almost any mucous membrane. They are sometimes subcutaneous, causing a general hemorrhagic eruption. Nervous symptoms are generally present, and are sometimes marked. They are restlessness, rolling of the head, a constant whining cry, twitchings of the muscles of the extremities or face, stiffening of the body, more rarely general convulsions. Late in the disease, dulness and stupor are present. The pulse is rapid and weak and the respirations are often irregular, even when there is no cerebral complication. Diarrhea is frequent; the stools are green, brown, sometimes black from the presence of blood, and are often very foul. Vomiting is less common. In addition to these there are symptoms due to the various forms of local inflammation—peritonitis, meningitis, pneumonia, erysipelas,

subcutaneous suppuration and gangrene, these all being found in varying degrees and in various combinations.

Prophylaxis.—Pyogenic infection of the child, like puerperal fever in the mother, may be considered a preventable disease. Its occurrence is usually due to a failure to carry out proper rules regarding cleanliness and asepsis in connection with delivery. The statistics of the Moscow Lying-in Asylum, published by Miller in 1888, show that previous to the general introduction of aseptic methods, from six to eight per cent of all infants born in the institution died from some variety of infection. In twenty-three hundred successive labors at the Sloane Hospital for Women, covering about eight years, not a single marked case occurred. From these figures it will be evident that in the vast majority of cases the occurrence of a case of infection of a serious nature is the fault of the physician or nurse in attendance.

The umbilicus should be cleansed and treated like any other fresh wound. Dry dressing should invariably be employed, and sterilized gauze or salicylated cotton in preference to household linen. If suppuration occurs at the time the cord separates, the parts should be cleansed daily with a bichlorid solution, and a wet dressing of the same applied. The ligatures and everything which comes in contact with the umbilical wound should be sterilized. Careful attention should be given to the mouth, genitals, and all the mucocutaneous surfaces, to prevent excoriations and intertrigo. Finally, every septic case occurring in an institution should be immediately isolated. A nurse in charge of a septic mother should not have the care of the infant.

Prognosis.—Pyogenic infections in the newly born, even in their mildest forms, are serious, and in their most severe forms almost always fatal. Very few cases recover in which erysipelas or any important visceral inflammation is present. The resistance of these patients is so feeble that the tendency of every inflammation is to spread, until they die from exhaustion. Only patients with localized inflammations, such as those of joints, skin, etc., are likely to get well.

Treatment.—This practically resolves itself into the treatment of individual symptoms as they arise. Wherever suppuration occurs, external abscesses should be evacuated and treated antiseptically. For the local inflammations of the lungs, peritoneum, and brain, little or nothing can be done in the way of direct treatment. Such inflammations are to be prevented, but can seldom be cured. The general indications are to look closely to the child's general nutrition by careful attention to all details of nursing and feeding, using stimulants whenever required by the condition of the circulation. For a local application in erysipelas, nothing in our experience has proven better than ichthyol ointment, ten to twenty-five per cent strength. It should be applied daily, spread upon muslin,

which is then covered by gutta-percha tissue to prevent drying. Vaccines have been much employed in erysipelas; our own experience, however, coincides with that of most observers that there is very little evidence that they have any value.

OPHTHALMIA

Ophthalmia of the newly born is to be classed among the pyogenic diseases. It usually consists in a purulent conjunctivitis. In the more severe cases there may be ulceration of the cornea, and even perforation into the anterior chamber of the eye.

The highly infectious nature of this ophthalmia is established. In the most severe cases the microörganism generally found has been the gonococcus; but in the milder forms the gonococcus may be absent, and any of the common pyogenic germs may be found. In the gonococcus cases the infection occurs during labor, from the secretions of the mother, from the examining fingers of the physician, or from instruments; or after birth from infected cloths and other materials which come in contact with the eye. Healthy lochia produce only a catarrhal inflammation. The infection occurring after birth may take place at any time. That due to gonococcus infection from the mother is generally manifested on the third day, and is often virulent from the outset.

The symptoms are, swelling of the lids, chemosis, copious purulent discharge, sometimes hemorrhages from the lids, ulceration, and there may even be sloughing of the cornea. The course of the disease depends upon the cause and upon the treatment employed. In the cases not due to the gonococcus the course is generally benign, and with ordinary cleanliness usually ends in recovery without any permanent damage to the sight. The gonococcus cases, unless energetically treated from the outset, are very frequently followed by permanent loss of vision. The best statistics upon the causes of blindness in adults show that from twenty-five to thirty per cent of such cases are due to ophthalmia in the newly born. This disease is occasionally complicated by other symptoms of gonococcus infection of a pyemic nature. Many cases followed by acute articular symptoms have been observed.

Prophylaxis is of the utmost importance. Credé's statistics show that in 1874 the frequency of ophthalmia in his lying-in hospital was 13.6 per cent. In the three years ending 1883, among 1,160 newly-born children, only one or two cases occurred. The method of prophylaxis which he adopted consists in dropping into the eyes of every child, immediately after birth, one or two drops of a two-per-cent solution of nitrate of silver. The general adoption of Credé's method, or of some similar means of disinfection, has resulted in a very great diminution in

the frequency of ophthalmia throughout the world. These prophylactic means should be obligatory in all institutions, and should be used in all cases in private practice wherever there is any possible suspicion of the existence of gonorrhea. In all other cases the eyes should be carefully cleansed with a ten-per-cent solution of argyrol. The use before delivery of an antiseptic vaginal douche is theoretically indicated, but practically it has been found to be inadequate for the prevention of the disease.

Treatment.—Everything which comes in contact with the eyes should be carefully disinfected. All cloths, cotton, etc., used for cleansing should be immediately burned. The strictest antiseptic precautions should be insisted on to prevent the spread of the infection by nurses. In institutions containing infants, severe cases of ophthalmia should always be isolated. The most important thing is to keep the eyes clean. In severe cases they must be cleansed every twenty minutes, night and day. It may be done by irrigation, or by using an eye-dropper with a bulbous tip, inserted alternately at the inner and the outer angle of the eye, and the fluid injected with force sufficient to empty thoroughly the conjunctival sac. Either a saturated solution of boric acid, or a 1-5,000 solution of bichlorid, may be used in this way. Once or twice in twenty-four hours two or three drops of a ten-per-cent solution of argyrol should be used in each eye after cleansing with sterile water. Next to these measures is the use of cold. It may be applied as ice compresses which are changed every minute or two from a block of ice to the eye. These may be continued one-fourth of the time in the milder cases; in the severe ones almost constantly. When the cornea is involved the pupil should be dilated by atropin. If only one eye is affected the sound one should be protected by covering it with a compress kept wet with an antiseptic solution.

TETANUS

Tetanus is an acute infectious disease characterized by tonic muscular spasm, which increases in severity by paroxysms occurring at longer or shorter intervals. It may be limited to the muscles of the jaw (trismus), or may affect all the muscles of the trunk, extremities, and neck.

The germ of tetanus usually gains access to the body of the infant through the umbilical wound. It exists in the soil, and the disease prevails endemically in certain localities. It is common in certain parts of Long Island and New Jersey. Among the negroes in some parts of the South it has for many years occurred with great frequency. It is stated that on one of the islands of the Hebrides every fourth or fifth child dies of tetanus. In a single house in Copenhagen eighteen cases

were observed. Tetanus presents no essential lesions. It is rare except where dirt and filth prevail; but these alone are not sufficient to produce the disease. It is rare in the tenements of New York.

Symptoms.—These, as a rule, begin on the fifth or sixth day, or at the time of the separation of the cord. The first symptoms may not appear until the tenth or twelfth day, but rarely later than this. Generally the first thing noticed is difficulty in nursing, which on examination is found to be due to rigidity of the jaws (trismus). Nursing may be impossible on this account. The muscles of the jaw feel hard, the lips pout, and all the muscles of the face seem firm. Soon a slight stiffening of the body occurs, the child straightening the back as he lies upon the lap and continuing rigid for a moment or two. In the interval he is at first completely relaxed. These paroxysms soon increase in frequency until they may come on every few minutes, being excited by any movement of the body. The relaxation is then only partial, and the neck and extremities and sometimes nearly the whole body may become rigid and stiff as a piece of wood. The arms are extended, the thumbs adducted, and the hands clenched. The thighs and legs are extended, and no motion is possible at the hip or knee. The jaws can be separated slightly or not at all. The firm contractions of the facial muscles give a peculiar expression to the features. There is a low, whining cry. Swallowing is difficult, sometimes impossible. The pulse is rapid and soon becomes weak. The temperature at first is normal, but in the most acute cases rises rapidly to 104° or even 106° F.; in the milder cases it does not go above 101° F.

Death may be due to exhaustion, to fixation of the respiratory muscles, or to spasm of the larynx. In the less severe cases all the symptoms are milder, and there may be intervals in which the rigidity is scarcely noticeable, so that respiration and deglutition may be carried on for some time. In cases which terminate in recovery the temperature is but slightly elevated. The tonic contractions gradually become less severe, and the paroxysms less frequent. The children usually suffer for several weeks from the general symptoms of malnutrition, which are proportionate to the severity of the attack. Of eighty-eight fatal cases which are reported by Stadtfeldt all but five died between the ages of six and ten days. The duration of the disease in the fatal cases is seldom more than forty-eight hours, often less than twenty-four hours; in those terminating in recovery, between one and three weeks.

Prognosis.—Few diseases of infancy are more fatal than tetanus. Where it prevails endemically it is regarded by the laity as so uniformly fatal that usually no physician is called. Scattered through medical literature are quite a large number of isolated cases in which recovery has occurred. At the present time the proportion of fatal cases is probably

between ninety and ninety-five per cent. Sporadic cases more frequently recover than those occurring in districts where the disease is endemic. The later the development of the symptoms, the slower their course, and the lower the temperature, the more likely is the case to recover.

Prophylaxis.—A proper understanding of the nature of the disease has brought with it the means of rational prevention. The first essential is obstetrical cleanliness, which must include scissors, hands, dressings, ligatures—in short, everything which comes in contact with the umbilical wound. In districts where tetanus is endemic, thorough aseptic treatment of the umbilicus should be insisted upon, both at the first dressing and later, particularly at the time of the separation of the cord.

Treatment.—All drugs whose physiological action is that of motor depressants of the spinal cord have a certain amount of value in tetanus. The most important ones are chloral and the bromids. Nearly all the reported cures have been by one of these drugs or a combination of them. The mistake usually made is in using too small doses. Enough to produce the physiological effects of the drug must be given. The initial dose should not be large, but it should be repeated until the full effects are obtained. Chloral, however, has been the drug most generally relied upon. An hourly dose of one or two grains is usually required. If no effect is visible in ten or twelve hours the dose may be further increased, as the patient is in much greater danger from the disease than he can possibly be from the drug. Chloral may be given by the mouth or by the rectum, but must always be well diluted. The single case of recovery which we have seen was one treated by the bromid of potassium. This infant took eight grains every two hours for three days, afterward smaller doses. The child must at all times be kept as quiet as possible, without unnecessary handling or bathing. If nursing or feeding by the mouth is impossible, because the jaws cannot be separated, the child may be fed by a tube passed through the nose. This is greatly to be preferred to rectal alimentation. Drugs may be administered in the same way.

The Antitoxin Treatment.—This is of especial value in prophylaxis. To be efficient as a curative measure it must be used early, for after the disease has developed it is very doubtful whether much can be accomplished by its use; but as it is harmless, it should be employed and given both intraspinally and intravenously.

EPIDEMIC HEMOGLOBINURIA (*Winckel's Disease*)

The essential features of this disease are hemoglobinuria with icterus and cyanosis, this combination giving the skin a deeply bronzed hue

(*maladie bronzée*). It is a rare disease, but has generally occurred epidemically in institutions. It is usually fatal. It is, without doubt, infectious, but its cause has not been discovered. Although generally called by the name of Winckel, who in 1879 made a report upon an epidemic of twenty-three cases, the disease was quite well described by Charrin in 1873, with a report of fourteen cases, and observed by Bigelow, in Boston, in 1875. All the cases included in Winckel's report occurred in one institution, affecting one-fourth of the children born during the period.

There is cyanosis, with a more or less intense icterus of the skin and internal organs. The umbilical vessels are usually normal. The kidneys are swollen, show small hemorrhages into their substance, and under the microscope the straight tubes are seen to be filled with crystals of hemoglobin, but contain no blood-cells. The bladder frequently contains brownish, smoky urine. The spleen is swollen and filled with blood pigment, which is diffused throughout the cells of the pulp, and free in the blood-vessels. Punctate hemorrhages are seen in most of the other viscera.

The symptoms usually begin from the fourth to the eighth day after birth, and are fulminating in character, seldom lasting more than two days. There are rapid pulse and respiration, general restlessness, prostration, cyanosis, and general icterus, which may be intense. The temperature is normal or slightly elevated. There is rapid asthenia, often terminating in coma or convulsions. The urine is passed frequently, in small quantities. It is of a smoky color, and contains hemoglobin in considerable quantity, renal epithelium, and sometimes granular casts and blood-cells, but does not contain bile pigment. Albumin is sometimes present, but not in large quantity.

Treatment is of little avail, since all severe cases are fatal.

FATTY DEGENERATION OF THE NEWLY BORN (*Buhl's Disease*)

A disease has been described by the author whose name it bears, the essential nature and causation of which are unknown. It occurs as isolated cases, and is characterized by inflammatory changes leading to fatty degeneration in the viscera, especially the heart, liver, and kidneys; it seldom lasts more than two weeks, and is almost invariably fatal. Many of the lesions are similar to the ordinary post mortem changes, and when found they should not be interpreted as pathological unless the autopsy is made within twelve hours after death.

The clinical features of this disease, as described, resemble those of pyogenic infection; and since the observations were made before modern

methods of bacteriological study, it is highly probable that Buhl's disease is merely a form of pyogenic infection in the newly born.

PEMPHIGUS NEONATORUM—BULLOUS IMPETIGO

Pemphigus is a term which designates a lesion rather than a disease. By it is meant an eruption of bullae occurring usually upon a red base, the contents being in most cases clear serum. A condition somewhat resembling pemphigus sometimes follows the use in the newly born of too hot baths. Again, bullae are seen as one of the lesions of congenital syphilis; they are then usually present at birth or appear soon after. They are most frequently seen upon the palms and soles. Infants so affected are generally in wretched condition, and soon die.

The only condition to which the term pemphigus neonatorum should be applied is quite different from both the preceding, and it has nothing in common with the pemphigus of later life. A better name is bullous impetigo, for its identity with impetigo contagiosa seen in older patients is now generally admitted. The disease is infectious, somewhat contagious, and occasionally occurs in small epidemics in institutions. Its spread in communities has been traced to midwives. The only important difference between this disease and the common impetigo contagiosa seen in older children, is its severity and its association with visceral infections. Most patients with bullous impetigo are delicate, neglected, and living in dirty surroundings; but not all are. We have seen it in robust infants who had received fairly good care.

The greater number of cases studied thus far have shown the presence in the blebs of the staphylococcus pyogenes aureus; less frequently the streptococcus has been the cause. The staphylococcus aureus was found in several typical cases occurring in our own hospital service. In one of these which came to autopsy, a general staphylococcus septicemia was present.

The clinical picture presented by pemphigus neonatorum is so striking that it can scarcely be mistaken. The symptoms begin in most cases between the fourth and tenth day of life. The bullae first appearing are scattered and often not larger than one-fourth or one-half inch in diameter. They may be seen upon any part of the body, but are especially frequent about the face, hands, and other exposed parts. They rupture or dry and form crusts without suppuration. The small bullae may gradually increase in size or several may coalesce until they cover an area two or three inches in diameter. As the disease progresses, new bullae may appear over almost any part of the body. The skin is at first slightly reddened, then an exudation of serum occurs beneath the epi-

dermis which loosens and slides upon the true skin. After rupture of the large bullae, the epidermis at the margin forms a thin filmy border or hangs in shreds easily detached. The base of the large vesicles is a moist bright-red surface. When many have formed the appearance closely resembles that seen after an extensive burn.

The course of the local symptoms is at first slow; then the bullae may spread with great rapidity and death occur in from twenty-four to forty-eight hours. In less severe cases the course is more prolonged, the blebs are smaller, and recovery may take place.

The constitutional symptoms are at first wanting, but increase with the number and extent of the bullae. There may be a slight rise of temperature or it may be subnormal. There is progressive weakness and

FIG. 8.—PEMPHIGUS NEONATORUM. Symptoms began on 13th day; death on 16th day of asthenia; temperature subnormal. The dark areas in the picture are entirely denuded of epidermis; they were formed by the coalescence of large bullae.

great depression, much like that following a burn, and death occurs from exhaustion or from some visceral inflammation such as pneumonia or meningitis.

A disease very closely allied to pemphigus neonatorum in its etiology and clinical symptoms is *dermatitis exfoliativa* (Ritter). This also is due to infection with staphylococci which are found not only in the skin but often in the blood and viscera. The cutaneous lesions when typical may readily be differentiated from pemphigus, but there are many instances in which the lesions of both conditions may be present at the same time. A further similarity is found in the fact that in institutional epidemics both forms of disease may occur side by side, pemphigus in some infants, dermatitis exfoliativa in others. There is at first a redness and slight swelling of the skin which usually occurs first around the mouth, spreading upon the face, and then appears upon the extremities and trunk. The skin seems as if macerated and eventually exfoliates in large masses, leaving exposed the red corium from which some serous exudation takes place but there is no accumula-

tion of fluid beneath the epidermis before the separation of the overlying skin. The area denuded may be very great, sometimes fully half the body being thus exposed. Death often results in two or three days. In other cases, it is delayed for a week or ten days. In some, recovery occurs. The general symptoms are similar to those seen in pemphigus.

It is important to distinguish pemphigus neonatorum from congenital syphilis. In syphilitic cases, the liver and spleen are usually markedly enlarged, and other characteristic changes may be present in the nails, mucous membranes, or elsewhere.

Treatment is of little avail in the most severe cases, when the bullae cover a considerable part of the surface of the body. The bullae should be opened and drained, and the surfaces dressed with gauze covered with a two-per-cent ointment of white precipitate. There is little danger of mercurial poisoning. When dressings are changed the skin should be sponged with a bichlorid solution, 1-5,000 strength, or a one-per-cent solution of ichthyol or permanganate of potash. On account of the contagious nature of the disease cases occurring in institutions should be isolated.

CHAPTER V

HEMORRHAGES

Hemorrhages are quite frequent during the first days of life, and are important not only from the fact that they are often the cause of death, but, when the brain is the seat, from their remote effects. There are several conditions in the newly born which predispose to bleeding—the extreme delicacy of the blood-vessels, and the great changes taking place in the blood itself and in the circulation in the transition from intra-uterine to extra-uterine life. Hemorrhages may complicate many of the diseases of the early days of life, such as syphilis or sepsis, or they may exist alone.

The cases may be divided into two groups: (1) Traumatic or Accidental Hemorrhages, which depend upon causes connected with delivery; (2) Spontaneous Hemorrhages, or The Hemorrhagic Disease of the Newly Born.

TRAUMATIC OR ACCIDENTAL HEMORRHAGES

These are mainly due to pressure in natural labor, or to means employed in artificial delivery, but some of them may possibly result from injuries received before birth. They are more frequent in large children,

in difficult labors, and where from any cause the body of the child has been subjected to undue pressure.

Hematoma of the Sternomastoid.—Hematoma of the sternomastoid muscle leads to the formation of a tumor in the belly of the muscle. It is a rather rare condition, usually noticed in the second or third week of life, and it disappears spontaneously, rarely causing any permanent deformity. The tumor varies from three quarters of an inch to one inch and a half in length, being about the size and shape of a pigeon's egg. It is movable, almost cartilaginous to the touch, and sometimes slightly tender. The situation of the tumor is usually about the center of the muscle. There is no discoloration of the skin.

In about two-thirds of the cases it occurs after breech presentations. It is much more frequent upon the right than upon the left side. In twenty-seven cases collected by Henoch the right side was involved in twenty-one and the left in only six cases. The explanation of this difference is to be found in the obstetrical position. Rarely, both sides may be involved. The head is usually slightly inclined toward the shoulder of the affected side and rotated toward the opposite side. The swelling slowly diminishes in size, and in most cases by the end of the third month has nearly or quite disappeared. Occasionally a slight torticollis remains for a longer time, but in the majority of cases the recovery is perfect. Hematoma of the sternomastoid is due to the twisting of the head during parturition. It is not an evidence of the employment of any improper force in delivery. The twisting of the head produces laceration of some of the blood-vessels of the muscle, and in some cases there is doubtless rupture of some of the fibers of the muscle itself. Following this there occurs a certain amount of inflammation of the muscle and its sheath. The tumor is due partly to blood-extravasation and partly to inflammatory products. In one or two recent cases in which the sheath of the muscle has been opened it has been found filled with blood.

The condition requires no treatment. Operative interference is positively contra-indicated.

Cephalhematoma.—This is a tumor containing blood, situated upon the head, usually over one parietal bone, and tending to spontaneous disappearance by absorption. The source of the blood is the rupture of the small vessels of the pericranium.

Etiology.—Cephalhematoma is sometimes due to a distinct traumatism like the application of forceps or to some other injury during labor. In the majority of cases, however, there is no evidence of such injury. Besides the conditions predisposing to all hemorrhages, there is the increased pressure in the blood-vessels of the head during delivery, especially when labor is prolonged or difficult; there may be changes in

the bone, such as an imperfect development of the external table; and, finally, there may be changes in the blood itself. Cephalhematoma is a comparatively rare condition; it was present according to the statistics of the Sloane Hospital for Women, in 20 of 1,300 consecutive births, or 1.6 per cent. The condition is more common after first or difficult labors, and in vertex presentations; occurring twice as often in males as in females, probably from the greater size of the head.

Lesions.—In the 20 Sloane cases, the situation was over the right parietal bone in 12; over the left in 2; over both parietals in 4; over the occipital in 2. The location of the tumor seems to have a very close relation to the position of the head in the pelvis. In 8 of the right-sided cases the head was in the left occipito-anterior position. Of the cases with occipital tumors, both were breech presentations. Of the 16 cases with a single tumor the labor was natural in 10, tedious in 4, and in 2 forceps were used. Of the 4 double cases, 2 were forceps deliveries.

Fig. 9.—Triple Cephalhematoma. Infant seven days old.

In rare cases triple tumors are met with, one over each parietal and one over the occipital bone (Fig. 9). The attachment of the periosteum along the sutures usually limits the tumor to the surface of one bone. It never extends across the sutures or over the fontanel. In cases where there is a more definite injury, such as that from forceps, the tumor may be present over any one of the cranial bones, but more frequently over the parietal. The seat of the hemorrhage is between the periosteum and the cranium. The scalp shows punctate hemorrhages and sometimes infiltration with blood. In recent cases the blood is fluid; later it is coagulated. The amount of extravasated blood is usually from half an ounce to an ounce. The cases following natural delivery are generally uncomplicated. The traumatic cases may be complicated by extravasations between the bone and the dura (internal cephalhematoma), or by meningeal or cerebral hemorrhages. If there is a wound, infection may be followed by purulent meningitis and even by cerebral abscess.

Symptoms.—The tumor is usually noticed from the first to the fourth day after birth, appearing as a slight prominence in one of the positions mentioned. Gradually increasing in size, it attains its

maximum at the end of a few days, and then slowly diminishes. In size and shape the usual tumor may be compared to the bowl of a tablespoon. In marked cases it may be one-third the size of the child's head. To the touch it is soft, elastic, fluctuating, and irreducible. It does not increase with the cry or cough. There is no extra heat and no signs of inflammation. Usually the tumor does not pulsate, although in rare instances pulsating cephalhematomata have been seen. Very soon the tumor is surrounded by a marginal ridge. At first this is apparently from coagulation of blood, but later it may be bony. The prominent ridge with the soft center gives a sensation somewhat like that of a depressed fracture. Sometimes on pressure there is obtained a sort of parchment-crackling. This is generally found as the swelling is subsiding, and is sometimes clearly due to the formation of minute bony plates upon the inner surface of the periosteum. It may be found when there is nothing but thin coagula to explain it. In certain cases following severe traumatism, cephalhematoma may be complicated with wounds of the scalp, fracture of the skull, and even lacerations of the dura mater or the brain. In such cases the tumor may become inflamed, but in the spontaneous cases this is extremely rare. The usual signs of abscess develop, which may open externally or burrow. Fortunately this termination is seldom seen.

As a rule, without any interference the uncomplicated cases go on to recovery. The complete disappearance of the tumor may be expected in from six weeks to three months, depending on its size; but a hard, uneven elevation may remain at its site for a longer time. The cases due to severe traumatism are more serious, the gravity depending not upon the cephalhematoma but upon the complicating lesions.

Diagnosis.—Cephalhematoma may be confounded with encephalocele. This, however, occurs along the line of the sutures or at the fontanels, is partially reducible, pressure causes cerebral symptoms, and frequently the tumor increases with respiratory movements. Caput succedaneum often appears in the same place as a cephalhematoma and at the same time, but this is an edematous, not a fluctuating tumor, and begins to disappear by the second or third day. From a depressed fracture of the skull, it is differentiated by the fact that in cephalhematoma there is a tumor and not a depression; the prominent margin which is raised above the contour of the skull is not osseous and the skull can be felt at the bottom of the center of the tumor.

Treatment.—The treatment in the uncomplicated cases is simply protective, all such cases tending to spontaneous recovery. No local or general treatment to promote absorption is required. The child should be so placed and so handled that no injury may be done to the affected part. Compresses are unnecessary. If complications exist, such as in-

jury to the bones, dura, or brain, they are to be treated in accordance with general surgical principles. Operative interference is called for only when suppuration has occurred, or when there are brain symptoms which point to the existence of internal as well as external cephalhematoma.

Visceral Hemorrhages.—While these are most frequent in large children and following difficult labors, they may occur in small children and where the labor has been easy and normal—their occurrence here being due to the feeble resistance of the blood-vessels. From one hundred and thirty autopsies upon still-born children or those dying soon after birth, Spencer concludes that intracranial hemorrhages are more frequent in head-forceps than in breech cases, and more frequent in breech than in natural vertex deliveries. Other visceral hemorrhages are much more frequent in breech cases.

Not all visceral hemorrhages are to be classed as traumatic. They are often seen with the spontaneous hemorrhages from the skin or mucous membranes. When, however, they are single, they seem to be of traumatic rather than of pathological origin.

The most important of the visceral hemorrhages are intracranial. These are discussed in the chapter devoted to Birth Paralyses. Rarely there may be large hemorrhages into the lung. Here the blood fills the air vesicles and the small bronchi, and coagula may be found even in the larger bronchi. A large part of a lobe or an entire lobe may be involved. On section the condition resembles atelectasis, and it may give the physical signs of consolidation.

The abdominal viscera suffer more than those of the thorax because less protected against pressure. Small hemorrhages are not uncommon upon the surface of any of the viscera covered by peritoneum. Intraperitoneal hemorrhages are rare, but may be very extensive, amounting to six or eight ounces. Sometimes no ruptured vessel can be found. The hemorrhage may be primarily in the peritoneal cavity, or it may result from rupture of one of the viscera, especially the suprarenal capsule. It may be large enough to produce death from loss of blood.

Small surface hemorrhages of the liver are not infrequent. Occasionally one of considerable size occurs separating the peritoneal covering and forming a tumor generally upon the superior surface. Such laceration may be produced during labor, and a slow accumulation of blood may take place beneath the capsule, death resulting from rupture into the peritoneal cavity. Laceration of the capsule of the liver in a still-born infant has been reported. Of the large hemorrhages, those into the suprarenal capsules are perhaps the most frequent. The capsule may be distended to nearly the size of an orange, the kidney being surrounded by a mass of blood-clots. Blood may be extravasated into

the retroperitoneal connective tissue and rupture may take place into the peritoneal cavity.

Except in the intracranial variety, visceral hemorrhages cause few symptoms, and in the great majority of cases the diagnosis is not made. Intrapulmonary hemorrhages have given rise to the signs of consolidation of the lung and even to hemoptysis. The abdominal hemorrhages are the most obscure. There may be a general abdominal distention with the usual symptoms of loss of blood, or there may be a circumscribed swelling. In many cases nothing is noticed until rupture of a subperitoneal hemorrhage takes place into the general peritoneal cavity, when there may be sudden collapse and death.

The visceral hemorrhages are not amenable to treatment. The prognosis depends upon the size and position of the hemorrhage. In the cases of abdominal hemorrhage the diagnosis is extremely obscure and is rarely made during life.

SPONTANEOUS HEMORRHAGES—THE HEMORRHAGIC DISEASE OF THE NEWLY BORN

A disposition to bleeding is seen with many diseases of the first few days of life, especially those of an infectious character like syphilis and pyemia. With most of these, however, the hemorrhages are small and the condition may be compared to the hemorrhagic tendency seen in certain forms of infection of later life, such as measles, smallpox, and malignant endocarditis. There is, however, a class of cases in which the hemorrhages are not associated with any other known process, and in which the escape of blood from the small blood-vessels is the chief or essential symptom. In these cases the bleeding is much more extensive than in the others mentioned. These hemorrhages are characterized by the fact that they are spontaneous in origin, having no connection with delivery, they are multiple in location, they tend to cease spontaneously after quite a limited time, but they are often greatly influenced by treatment. They are most often from the mucous membranes of the stomach and intestines, or from the umbilicus or beneath the skin, but they may be from almost any mucous surface or into any organ of the body.

Etiology.—These hemorrhages are not common, and are met with more often in institutions than in private practice. In 5,225 births in the Boston Lying-in Asylum, Townsend reports 32 cases of hemorrhage, or 0.6 per cent. In the Lying-in Asylum of Prague, Ritter observed 190 cases in 13,000 births, or 1.4 per cent. In the Foundling Asylum of Prague, Epstein reports hemorrhages in 8 per cent of 740 infants.

The condition is not a manifestation of hemophilia. Only 12 of 576 bleeders whose histories were collected by Grandidier had a history of hemorrhage at the time of the falling off of the cord, and symptoms very rarely appeared before the end of the first year. Hemorrhages in the newly born are only slightly more frequent in males, while in hemophilia they predominate 13 to 1. The hemorrhagic disease of the newly born is self-limited, and runs a definite course to recovery or death. The tendency to bleed does not extend beyond a few weeks, and often lasts but a few days. Circumcision has been done within a few days after the cessation of the hemorrhages without any unusual bleeding. In a case under our observation with the most extensive subcutaneous hemorrhages we have ever seen, all tendency to bleed had ceased before the separation of the cord, although there had previously been bleeding at the navel. The bleeding occurs with about equal frequency in feeble and in well-nourished infants. Syphilis is associated in but a small proportion of the cases. On the other hand of 132 cases of congenital syphilis observed by Mracek, only 14 per cent suffered from hemorrhages.

An association with sepsis has sometimes been noted. Of the 61 cases observed by Epstein not less than 29, and of the 190 cases of Ritter, 24 were associated with sepsis. During one year of our service at the Nursery and Child's Hospital there were 8 marked cases of hemorrhage in about 225 deliveries. While more cases of sepsis occurred among the children during that year than usual, it was striking that not one of these hemorrhagic cases gave any evidence of sepsis, and that none of the septic cases had bleeding. Yet the circumstances in which these hemorrhages sometimes occur point strongly to an infectious origin. The results, often remarkable, following the injection of human blood serum indicate that the essential cause, in the largest number of cases, is a lack of some substance in the blood essential to coagulation. Sufficient studies have not yet been made to establish the precise nature of these blood changes. The results of treatment would seem to show that the cause of these hemorrhages is not always the same.

While the hemorrhages are not traumatic, bleeding is exceedingly prone to occur in the skin over pressure points such as the back, the elbows, the occiput, and the sacrum. It is also common from the mucous membranes which are the seat of pathological processes, especially from the eyes, the nose, and the genitals.

Lesions.—In very many of the cases the autopsy shows nothing except the hemorrhages in the various situations and the blanching of the organs due to the loss of blood. The hemorrhages of the brain are usually meningeal and diffuse. They are considered more at length in the chapter upon Birth Paralyses. The pulmonary hemorrhages are usu-

ally small and unimportant, and large hemorrhages into the pleura or pericardium are very rare. The stomach and intestines may contain considerable blood variously disorganized in the different parts of the canal, and there may be ecchymoses of the mucous membrane. In addition, ulcers may be found in the stomach and duodenum. In twenty-four autopsies upon cases with hemorrhage from the stomach and intestines collected by Dusser, ulcers were found in the stomach in nine cases, and in the intestines in four. These ulcers are multiple, small, and usually superficial, but may extend to the muscular coat and may even perforate. The intestinal ulcers are found only in the duodenum and resemble those of the stomach. The cause of these ulcers is somewhat obscure; some of them are undoubtedly dependent upon inflammatory changes, probably of infectious origin; others have been compared to the peptic ulcers of later life, and are attributed to thrombi in the blood-vessels of the mucous membrane. These ulcers are found in but a small proportion of the cases in which bleeding occurs from the alimentary tract, and they may be wanting even when it has been very profuse. Small extravasations may be seen upon the surface or in the substance of any of the abdominal organs. The changes found in the blood have not been uniform.

Symptoms.—The onset is most frequently in the first week of life; very rarely after the twelfth day. The hemorrhages are usually multiple. Their location in Ritter's 190 cases was as follows: Umbilicus, 138 (umbilicus alone, 97); intestines, 39; mouth, 28; stomach, 20; conjunctivae, 20; ears, 9. In Townsend's 50 cases: Intestines, 20; stomach, 14; mouth, 14; nose, 12; umbilicus, 18 (umbilicus alone, 3); subcutaneous ecchymoses, 21; abrasion of skin, 1; meninges, 4; cephalhematoma, 3; abdomen, 2; pleura, lungs, and thymus, 1 each.

In many cases nothing is noticed until the hemorrhage begins. The first bleeding noticed may be from the stomach, intestines, or any of the mucous surfaces, beneath the skin, or from the umbilicus. The amount of blood lost in most cases is not great, but there is a continuous oozing. The total hemorrhage may be only a few drams or it may reach several ounces. The general condition is one of considerable prostration, often from the outset. In all cases there is rapid loss of weight. The temperature may be high, low, or subnormal. A marked elevation of temperature may depend not upon the hemorrhage but upon associated conditions. In a large number of the cases there is diarrhea.

The duration of the disease in cases which recover is usually but one or two days. In fatal cases it is rarely more than three days, and often less than one. Death may result from the gradual failure of all the vital forces or from rapid loss of blood.

Umbilical Hemorrhage.—A slight oozing from the umbilicus not in-

frequently occurs when the ligature has been improperly applied. This is generally controlled by simple measures. Spontaneous hemorrhage is quite different. It occurs rather later than bleeding from the mucous membranes, usually occurring between the fourth and the seventh day. There may be bleeding into the cord as well as from its free extremity. A slight stain upon the dressing is usually the first note of warning, but in exceptional circumstances a gush of blood is the first symptom. The hemorrhage may be temporarily arrested by various means, but it shows a strong tendency to recur in spite of everything which is done. The usual duration is two or three days. It has been known, however, to persist for twelve or fourteen days, and it may be fatal in less than twenty-four hours from the time it is noticed.

Hemorrhage from the Stomach and Intestines.—Bleeding occurs much less frequently from the stomach than from the intestines. The latter is called *melena.* Gastro-enteric hemorrhages begin, in the great majority of cases, during the first three days of life. The blood vomited is usually in dark-brown masses, and not very abundant; more rarely it is bright red. The quantity varies from one dram to half an ounce. Vomiting is liable to be excited by nursing. The blood discharged from the bowels is always dark colored, usually intimately mixed with the stool, very rarely in clots. If in doubt between blood and meconium, one should look for the corpuscles with the microscope. When this is not conclusive on account of the disorganization of the corpuscles, a chemical test for hemoglobin should be made. Concealed hemorrhage into the stomach may take place, which may even be sufficient to produce death, no blood being vomited or passed by the bowels. In such cases the autopsy may reveal quite a large quantity of blood both in the stomach and intestines.

Hemorrhage from the Mouth.—The quantity of blood is rarely large; but it is here that it is often first seen. Its source may be the mucous membrane of the mouth, pharynx, esophagus, stomach, or bronchi. It may be associated with ulceration of the hard palate, with thrush, or with fissures of the lips.

Hemorrhages from the nose are infrequent, and are more often due to syphilis than to other causes. These are rarely profuse, but are frequently repeated.

Subcutaneous Hemorrhages.—These often appear in places exposed to pressure, such as the sacrum, heels, occiput, or back, but may occur anywhere. In some cases these hemorrhages are very extensive, as in one recently under observation, where nearly one-third of the thorax was covered. When they occur alone or form the principal lesion, the prognosis is favorable.

Hematuria.—The urine is not only stained with blood, but some-

times contains clots. This hemorrhage may have its origin in the bladder, urethra, or kidney. Blood coming from the kidney is sometimes due to the irritation of uric-acid infarctions, and may have nothing to do with the general hemorrhagic disease.

Hemorrhage from the Conjunctiva.—The blood usually comes in drops from between the eyelids, chiefly from the tarsal surface. It is generally preceded by conjunctivitis.

Hemorrhage from the Female Genitals.—This not infrequently occurs without hemorrhages elsewhere, and under such circumstances is rarely serious. Cullingsworth collected thirty-two cases in children under six weeks of age—no case having resulted fatally. These are not to be regarded as cases of precocious menstruation.

Diagnosis.—This is generally easy, as the hemorrhages are usually multiple and some of them external. A slight hemorrhage from the intestine may be easily overlooked. Large hemorrhages into the internal organs also are obscure and not often recognized. Spurious hemorrhages from the stomach may occur, blood being vomited which has been swallowed during birth or nursing. The source of bleeding may also be the mouth, nose, or pharynx, and sometimes blood is swallowed in large quantities and afterward vomited. These cavities should therefore always be examined, since local treatment may be efficacious. Syphilis should be suspected when the bleeding is chiefly nasal.

Prognosis.—Before the introduction of treatment with human blood serum the hemorrhagic disease in the newly born had a very bad prognosis. Of 709 cases collected by Townsend, the mortality was 79 per cent. No case should be looked upon as hopeless, for perfect recovery has repeatedly taken place when it seemed impossible.

Treatment.—Local measures may be employed in all external hemorrhages with some prospect of benefit. The bleeding points may be touched with persulphate of iron or with chromic acid fused upon a probe, or fresh human blood or human serum may be applied locally. These measures may be employed alone or in combination with pressure.

Although recoveries have been reported following the use of a great variety of remedies, it is by no means established that the result was due to the drugs employed. Many of the milder cases recover without any special treatment. On the whole, the medicinal treatment is very unsatisfactory. Epinephrin is of doubtful benefit. Gelatin has had many advocates. It is used by subcutaneous injection. A 5-to-10-per-cent solution which has been twice sterilized is employed, from 25 to 50 c. c. being administered two or three times daily. Calcium lactate in some instances appears to exert a positive effect. It may be given in frequently repeated doses up to 20 or 30 grains a day.

The most efficient treatment is transfusion, first practiced by Carrel.

It should, if possible, be performed whenever the loss of blood has been great. From 90 to 150 c. c. may be given. This not only replaces blood lost but, in the vast majority of cases, stops further bleeding at once. Its action seems specific and the effects of transfusion are often truly marvelous. That the subcutaneous or intramuscular injection of human blood serum would control these hemorrhages was first shown by J. E. Welch. Almost equally efficacious is the injection of human blood in the same manner. Usually 30 to 40 c. c. of blood or blood serum is injected at one time, and this should be repeated every few hours, if bleeding continues. For transfusion, only the blood of a parent should be used without previous hemolytic tests; for subcutaneous use, blood from any healthy person will answer as well. The subcutaneous injection of horse serum has a certain value in these cases and should be employed when it is impractical to obtain human blood serum. It is, however, distinctly inferior. In some instances thrombin, prepared according to the method of Howell, has caused a cessation of the hemorrhage. A small proportion of patients, however, are not improved by the measures mentioned, and in spite of them bleeding may continue. These suggest a different etiology of which we have as yet no clue. The general treatment should have reference to maintaining the nutrition by careful feeding, judicious stimulation, and attention to the circulation, the body temperature, and the general condition of the child.

CHAPTER VI

BIRTH PARALYSES

Birth paralyses are chiefly due either to pressure upon the child by the parts of the mother or to artificial means employed in delivery. They may be cerebral, spinal, or peripheral.

Cerebral paralyses are in almost every instance due to meningeal hemorrhage, and accompanied by a certain amount of injury to the brain substance. Very infrequently they depend upon cerebral hemorrhage, laceration of the brain, or pressure from a depressed fracture.

Spinal paralyses are extremely rare, and only a few examples are on record. They are due to laceration of, or hemorrhage into, the cord or its membranes. These lesions produce paraplegia, the exact distribution of which depends upon the point at which the cord is injured.

Peripheral paralyses usually affect the face or the upper extremity. Paralysis of the face is due in most cases to the application of forceps. Paralysis of the upper extremity is most frequently of the "upper-

arm type," and is known as the Duchenne-Erb paralysis. It usually follows extraction in breech presentations. Peripheral paralysis of the lower extremity is almost unknown.

CEREBRAL PARALYSIS

Cerebral paralysis is often used synonymously with meningeal hemorrhage. This lesion is not infrequent, and is of great importance not only from its immediate effects, but because upon it depend many of the cerebral paralyses seen in later life. According to Cruveilhier, at least one-third of the deaths of infants which occur during parturition are due to this cause.

Etiology.—The same predisposing causes exist in the cases of meningeal hemorrhages as in others occurring at this time. A small number of cases are associated with syphilis; others with pyogenic infection. In a few cases there is a history of an injury—usually a fall or blow upon the abdomen—during the last months of pregnancy. Meningeal hemorrhage may occur as one of the lesions in the hemorrhagic disease of the newly born. The most important causes, however, are connected with parturition. These hemorrhages are essentially mechanical, and are favored by everything which increases or prolongs pressure upon the head. The conditions with which they are associated are tedious labor, breech presentations with difficulty in extracting the head, instrumental deliveries, and premature births. The majority occur in first-born children. In many of the cases there is also a hemorrhage outside the skull.

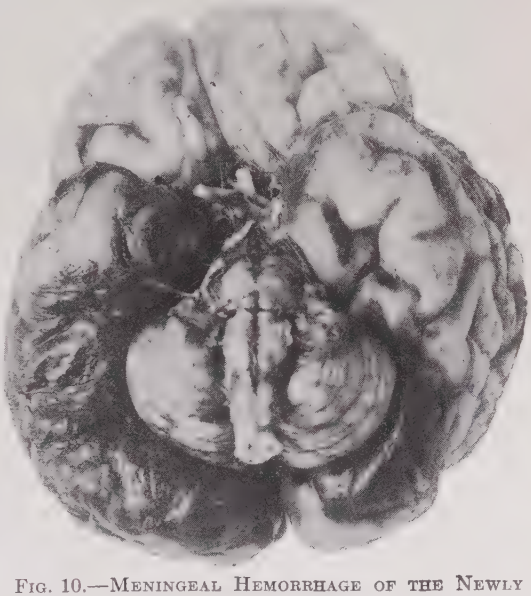


Fig. 10.—Meningeal Hemorrhage of the Newly Born. (Extravasation above the Tentorium.)

Lesions.—The hemorrhages may be large or small. If small, they are frequently multiple and are found scattered over the convexity. In such circumstances they are usually beneath the arachnoid. Edema of the brain is often associated with them. It is doubtful if

very small hemorrhages that may cause little more than a discoloration of the meninges are sufficient to account for death. They are found so frequently when there have been no symptoms referable to the brain that it is a question if they are not quite a common sequel of labor. Larger hemorrhages may be at the base or at the convexity and either in the anterior or posterior part of the skull. When upon the convexity, the blood usually comes from the veins ascending from the middle cerebral region to the lateral aspects of the superior longitudinal sinus. These are lacerated by the over-lapping of the parietal bones. Convexity hemorrhages are rarely limited to one hemisphere, although the one side may be much more affected. It is usual for the blood to gravitate toward the base and become diffused. Nearly the entire surface of the brain may be covered. Hemorrhages are frequently found over the cerebellum and the occipital lobes of the cerebrum; these are usually due to rupture of the tentorium. While this may allow of some extravasation of blood above the tentorium, the entire extravasation is often beneath it. Rupture of the tentorium is usually due to marked lateral compression of the head, but may occur when the pressure is anteroposterior. It is apparent that hemorrhages may result very rarely from marked venous congestion. In this way is explained the hemorrhage which is occasionally found in the lateral ventricles alone. This comes from rupture of the straight sinus or of the great vein of Galen. Hemorrhages between the dura and the skull may be said never to occur except when associated with fracture. If the child is still-born, or, if death has occurred on the first or second day, the blood is partly fluid and partly coagulated; later it is entirely coagulated and may have undergone partial absorption. The amount of extravasated blood varies between one dram and two ounces, the average amount being about one-half ounce. The blood extends into the fissures between the convolutions and sometimes into the ventricles along the choroid plexus, although this is rare. In large hemorrhages the brain substance is softened and in places may be quite disintegrated; but with small extravasations these changes are very slight and hard to demonstrate to the naked eye. In cases which survive for two or three weeks there is usually a certain amount of meningitis. The later changes—those of arrested development of the cortex and cerebral sclerosis—will be considered in the chapter devoted to Cerebral Paralyses in the section on Diseases of the Nervous System. Hemorrhages into the membranes of the upper part of the cord are found in a large proportion of the fatal cases. Associated hemorrhages of the lungs and other organs are not uncommon.

Symptoms.—If the hemorrhage is large, the child is usually still-born, although the fetal movements may have been active up to the

commencement of labor. When the hemorrhage is not so large as to be immediately fatal, the child may show no symptoms except dulness or stupor, with feeble or irregular respiration, death following within the first twenty-four hours. A large proportion of the infants are born asphyxiated, and frequently they are resuscitated only after considerable effort. They nurse feebly or not at all. Convulsions are common in cases which last for four or five days, and more with hemorrhages at the convexity than with those at the base. Opisthotonus is often present, also general rigidity of the extremities, clenching of the hands, and increased knee-jerks. Rarely there is complete relaxation of all the muscles. Sometimes there are automatic movements. The respiration is usually disturbed; in most cases it is slow and irregular. The pulse is feeble and usually slow. The pupils are more frequently contracted than dilated, and there may be oscillation of the eyeballs. There may be a slight exophthalmus. In large hemorrhages there is marked bulging of the fontanel, and often separation of the sutures. If the hemorrhage covers one hemisphere, there is complete hemiplegia of the opposite side. Small localized cortical hemorrhages may cause paralysis of the face, arm, or leg, according to the position of the lesion, or localized convulsions. In large hemorrhages at the base convulsions are rare, and death occurs early, usually in the first two days. In extensive cortical hemorrhages convulsions and rigidity of the extremities are frequent, and life may be prolonged indefinitely. There is usually no fever, but exceptionally the temperature may be high.

The majority of the fatal cases die within the first four days. In those lasting a longer time the symptoms are tonic spasm of the trunk or of one or more of the extremities, with localized paralysis—monoplegia, diplegia, or hemiplegia, according to the lesion—and localized or general convulsions often continuing for two or three weeks and gradually subsiding. In the mildest cases nothing abnormal may be noticed until the child is old enough to walk or talk. In those more severe there may be gradual and continuous improvement of the early symptoms, and the case may go on to apparent recovery, but usually there is some permanent damage to the brain.

The main diagnostic symptoms in recent cases are: bulging fontanel, slow pulse, stupor, rigidity, increased reflexes, convulsions, and paralysis, especially when localized, and opisthotonus. These vary with the extent and situation of the lesion. Lumbar puncture has very doubtful value.

Prognosis.—A large hemorrhage at the base quickly causes death; if it is located at the convexity, although the child may survive, there is always serious damage to the brain. Even from small hemorrhages some permanent injury usually results, though the extent of this may not be evident for years.

Treatment.—This is mainly prophylactic, the chief indication being to shorten tedious labors by the early use of the forceps. When the hemorrhage has been attributed to the forceps, the damage has probably been the result of the long-continued pressure before they were used. Nothing can be done after delivery to limit the amount of the hemorrhage, except to keep the child as quiet as possible. The removal of the clot by surgical operation has been successfully accomplished by Cushing and others. With more accurate diagnosis there seems to be no reason why a certain number may not be saved. For the best results operation should be done as soon as possible. One great difficulty is that of early and accurate diagnosis. Paralysis whether localized or general is of greater value in diagnosis than are convulsions. The latter, however, are especially important when localized or continuous and threatening life. The operative risk, while considerable, is not to be measured against the permanent mental deficiency usually resulting in most of these children when nothing is done. Cases with similar symptoms are sometimes seen in which there is no extravasation of blood found at operation, but only intense congestion with an excessive serous exudate. In them also relief may follow operation. The hopeless outlook for such cases when not relieved, justifies the taking of great risks.

FACIAL PARALYSIS

The usual cause of facial paralysis is the use of the forceps, but this does not explain all the cases. The etiology of those in which the forceps have not been used is still somewhat obscure. In peripheral facial palsy the nerve is pressed upon, either near its exit from the stylomastoid foramen, or where it crosses the ramus of the jaw, at which point the parotid gland gives it but little protection in the newly born. If the lesion is in front of this point, any one of the terminal branches may be affected; most frequently it is the temporofacial branch. As only one blade of the forceps commonly touches the face in this region, the paralysis is, as a rule, unilateral.

Roulland has reported several cases not due to the forceps. In these the pressure is believed to have been produced by the promontory of the sacrum at the superior strait, or by the ischium at the inferior strait, as paralysis followed when the head was long arrested at one of these points. It was not seen with face or breech presentations. When facial paralysis is of central origin it depends generally upon a meningeal hemorrhage, and the arm and leg of the same side as the face are involved. It is, however, possible for a very small cortical hemorrhage to produce paralysis of the face only.

In repose, the only symptom noticed may be that the eye remains open upon the affected side, owing to paralysis of the orbicularis palpebrarum. When the muscles are called into action, as in crying, the whole side of the face is seen to be affected. The paralyzed side is smooth, full, and often appears to be somewhat swollen. The mouth is drawn to the side not affected. In this paralysis, the tongue, of course, is not implicated. It is therefore rare that nursing is seriously interfered with.[1] If the paralysis is of central origin, only the lower half of the face is involved, while in peripheral paralysis, as the trunk of the nerve is injured, the upper half of the face, including the orbicularis palpebrarum, is also affected.

The paralysis is generally noticed on the first or second day of life, and does not increase in severity. Its course and termination depend upon the extent of the injury done to the nerve. Some idea of this may often be gained by the amount of injury to the soft parts, although this is not an infallible guide. In cases not due to the forceps, the paralysis is slight and disappears in a few days; the great majority of the forceps cases follow the same favorable course, the paralysis gradually disappearing without treatment in about two weeks. In more serious cases it may last for months, or it may be permanent. The reaction of degeneration is present in these severe cases, and there may even be perceptible atrophy of the muscles. This symptom is fortunately extremely rare.

Treatment.—Nothing should be done for the first ten days except to protect the eye and keep it clean. If improvement has begun by the end of this time, the probabilities are that the case will require no treatment. If no improvement has taken place by the end of the third or fourth week, electricity should be used regularly and systematically. If the muscles respond to it, the faradic current may be employed; if not, galvanism should be used. The electrical treatment should be continued for several months, or until recovery has taken place.

BRACHIAL BIRTH PALSY

This, sometimes called "obstetrical paralysis" or "Duchenne-Erb paralysis" is fortunately not a common condition. It is almost always unilateral, though occasionally both arms are involved. It may result from spontaneous delivery but is vastly more frequent following operative interference in difficult labor. In the majority of cases it is directly due to manipulation, though it may occur in the practice

[1] In this connection it is to be remembered that the principal part in nursing is done by the tongue, and not by the lips.

of the most skillful. Pressure from the application of forceps, while a possibility, is an infrequent cause, though long regarded as the most important one. The injury may be produced by any manipulation that forcibly draws the head and neck away from the shoulder. This puts the brachial plexus upon the stretch. If the force is slight, only stretching of the nerves is caused; if more extreme, laceration of the nerves is produced from above downward. The suprascapular nerve is by its position the one most exposed to injury and is the one that is first and most severely torn. The fifth cervical next is affected, then the sixth, the seventh and perhaps the eighth and the first dorsal. While the injury is almost always to the plexus alone it is probable that in some cases one or more of the roots in the cervical region may be torn from the cord. The amount of spontaneous improvement depends upon the extent of the lesion. When only overstretching has been produced, a complete recovery may take place. The same may be true when the laceration of the nerves has been slight and the ends remain in apposition. When more extensive injury has taken place complete recovery cannot be expected. Hemorrhage has occurred and there has been laceration of the fascia as well as the nerves. The result is usually the production of a cicatricial mass that interrupts the continuity of the nerves and prevents their regeneration. The nerve impulses are thus blocked.

FIG. 11.—ERB'S PARALYSIS, LEFT ARM.
Infant two months old.

The paralysis in severe cases is noticed soon after birth owing to the fact that the infant cannot use his arm. In less severe cases the paralysis may escape detection for several weeks.

The most common form of peripheral paralysis is that known as the "upper-arm type." The muscles paralyzed are the deltoid, biceps, brachialis anticus, supinator longus, and sometimes the supra- and infraspinatus. All these muscles may be involved, or only part of them, and in varying degrees. The arm hangs lifeless by the side; it is rotated inward, the forearm pronated, the palm looking outward (Fig. 11). The forearm and hand are not affected, except in cases where the whole plexus

has been lacerated. In severe cases there may be anesthesia of the outer surface of the arm, in the region supplied by the circumflex and external cutaneous nerves. This is rarely marked, and in its slighter degrees it is very difficult to determine. It is characteristic of this paralysis that the triceps is not affected, so that power to extend the forearm remains, although it cannot be flexed. A nodular mass in the region of the plexus may be felt. This is the result of the hemorrhage and the inflammatory reaction. Atrophy of the paralyzed muscles occurs after a few weeks, but the muscles are so small and so covered with fat that it is rarely noticeable before the second year. It is most conspicuous in the deltoid. In all severe cases the reaction of degeneration is present. In some of the cases of long standing there occurs a shortening of the tendon of the subscapularis muscle, often associated with subluxation of the humerus. The paralysis may be complicated with fracture of the clavicle, the neck of the scapula, or the shaft of the humerus, or with epiphyseal separation of its head. Injury confined to one nerve is very uncommon. We have seen two cases in which there was temporary paralysis of only the muscles supplied by the musculo-spinal nerve. The explanation of such cases is obscure.

The prognosis depends upon the severity of the injury. Some cases recover spontaneously in two or three months, improvement being observed within a few weeks, first in the biceps and last in the deltoid. Recovery after many months may take place even in cases apparently severe. Gradual improvement may continue to the end of the second year. The condition is, however, a very serious one. There is usually some permanent paralysis left and it may be so marked as to render the arm almost useless. Permanent paralysis is most frequently of the deltoid.

The electrical reactions are of some value in prognosis. If the muscles respond to faradism, rapid improvement can generally be prophesied. If the reaction of degeneration is present, improvement will be slow and the paralysis is likely to be permanent.

The diagnosis is usually not difficult, since the great majority of cases are of the "upper-arm type" with classical symptoms. Peripheral palsy of the arm can scarcely be confounded with that of cerebral origin. If the lesion is central it is one of the rarest occurrences for the arm alone to be involved; either the leg or face, or both, are generally likewise affected. If the case does not come under observation until the child is a year old, it may be difficult, or without a good history it may be impossible to distinguish peripheral paralysis from that due to poliomyelitis. The particular group of muscles involved in Erb's paralysis is the chief diagnostic point.

In recent cases the disability resulting from the tenderness or pain of

syphilitic epiphysitis may simulate paralysis, but there is lacking the characteristic position of the arm, and a careful examination discloses the fact that the paralysis is only apparent. This may affect both sides. Fracture of the clavicle or epiphyseal separation of the head of the humerus may also be mistaken for paralysis. In cases of long standing, paralysis of the deltoid may resemble dislocation of the humerus. The reaction of degeneration differentiates paralysis from surgical injuries with similar deformities.

Treatment.—As soon as the paralysis is discovered the injured arm should be put at rest by means of a sling, with the shoulder elevated in order to bring the ends of the nerves in apposition. At the end of two or three weeks gentle massage may be employed. In cases going on to permanent recovery improvement is rapid. At the end of two months it is generally possible to tell to what extent recovery will take place. If very little has been gained by that time, and if a surgeon expert in nerve surgery can be consulted, operation should be considered, for at this time less nerve degeneration will have taken place than at a later date and the regeneration of the nerves will require much less time. The operation consists in dissecting out and suturing the nerve trunks whose continuity has been broken by the injury. A. S. Taylor, New York, from an extended experience, has reported marked improvement in some otherwise hopeless cases by this operation. Though useful in mild cases, but little is to be expected from manipulation and electricity in severe cases without operation.

CHAPTER VII

TUMORS OF THE UMBILICUS, MASTITIS, ETC.

Granuloma.—This is nothing more than a mass of exuberant granulations at the umbilical stump. The mass is generally about the size of a pea– sometimes larger—bleeds readily, and has a thin, purulent discharge. It is promptly cured by the application of any simple astringent; powdered alum is probably the best. In case this is not successful, the granulations may be touched with nitrate of silver or snipped off with scissors.

Adenoma, Mucous Polypus, or Diverticulum Tumor—Umbilical Fistula. The first three terms are used synonymously to describe an umbilical tumor covered with a mucous membrane which is similar in structure to that of the small intestine. It is usually associated with an umbilical fistula. This tumor is formed by a prolapse at the navel of the mucous membrane of Meckel's diverticulum. This diverticulum is the remains

of the omphalomesenteric duct. When it is present in infants, it is found in various stages of development. Most frequently there is a blind pouch a few inches long given off from the lower part of the ileum. In other cases it may remain patent quite to the umbilicus, causing a fecal fistula (Fig. 12, A). As the intestine below it is generally normal, this fistula may persist for months or even years, giving rise to no symptoms except a slight fecal discharge from the umbilicus. In certain cases intestinal worms have been discharged through it. It may close spontaneously or be closed by operation.

A prolapse of the mucous membrane lining the diverticulum produces an umbilical tumor with a fistula at its summit (Fig. 12, B). This is the most common form. A cross-section shows under the microscope the

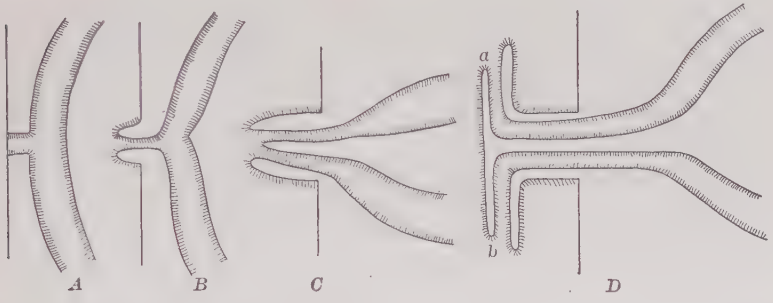


FIG. 12.—UMBILICAL FISTULA AND TUMORS PRODUCED BY PROLAPSE OF MECKEL'S DIVERTICULUM. (Barth.)

structure of the intestinal mucous membrane both as an external covering and lining of the fistulous tract. The prolapse may involve not only the mucous membrane but the entire intestinal wall. There then exists a conical tumor with a fistula which has but one external opening, but at a short distance from the surface it bifurcates, one branch leading upward and one downward (Fig. 12, C). A continuation of the prolapse gives a broad pedunculated tumor (Fig. 12, D), which may reach the size of an orange. Its covering is the same as in the other forms. It may contain several coils of intestine. In this form there are usually two fistulous openings (*a, b*) which communicate with the intestine.

In all of these cases the tumor is smooth, irreducible, of a rosy pink color, and from its surface there oozes a mucous discharge. Microscopical examination shows the external covering to be the same in structure as the intestinal mucous membrane. These tumors are generally small, varying in size from a pea to a small cherry, but they may be very much larger. A fecal fistula usually, but not invariably, coexists. In the condition represented in Fig. 12, B, it is easy to see how an obliteration of the fistula may occur. The small tumors are readily cured by the liga-

ture. The larger ones are usually associated with other serious malformations of the intestines, which make the outlook bad in almost every instance.

UMBILICAL HERNIA

Hernia into the umbilical cord is a rare congenital condition of a serious nature. It is due to some fetal defect, and varies in size from a small protrusion to complete eventration in which nearly all the abdominal organs are outside the body. Many cases in which only intestinal coils are contained in the sac, though the tumor is quite large, are amenable to surgical treatment, which should be instituted at once. In the very large ones the prognosis is bad.

The common umbilical hernia is quite a different condition, and while a source of much annoyance it is rarely serious. It is much more common in females than in males, and occurs especially in those who are poorly nourished and rachitic. The tumor is usually from one-fourth to one-half an inch in diameter; it may, however, be very large, and may even become strangulated, when a surgical operation may become necessary. The ordinary cases, however, require only mechanical treatment. The most important thing is prevention. For this purpose it is necessary, after the cord has separated, to place a firm pad over the navel and to use a snug abdominal band for the first two or three months. After this period it is uncommon for hernia to develop. In cases coming under observation after the third or fourth month, the pad and abdominal bandage are inadequate, and other means must be employed to retain the hernia. The best of these consists in the use of two adhesive strips applied obliquely over the abdomen, crossing at the umbilicus, the skin along the median line being folded inward so as to overlap the tumor, this forming the retention pad. A simple method of retention is to place over the tumor a coin or button covered with kid and hold it in position by a strip of adhesive plaster ten or twelve inches long. One should be cautious about using the small conical pads frequently employed, as these tend to dilate the opening rather than to close it. If the skin is made absolutely clean and zinc-oxid plaster used, excoriations are rare. The dressing should be changed every week or ten days and worn for several months. After the first year all mechanical treatment is unsatisfactory. For the very small tumors it is really unnecessary to use any form of apparatus, since these cases ordinarily show little or no tendency to increase in size, and the retention apparatus causes more annoyance than the hernia. These small herniae sometimes disappear spontaneously during childhood, and rarely need be considered in children over seven years of age. Operation is seldom necessary.

MASTITIS

According to Guillot, a certain amount of secretion in the breasts of the newly born is physiological. It is certainly very common. It is most abundant between the eighth and fifteenth days, but may continue in small quantities as late as the third month. It is seen with equal frequency in both sexes. The quantity of the secretion amounts in most cases only to a few drops; in some, however, as much as a dram has been obtained. Chemical analysis has shown this secretion to be essentially the same as the adult milk—containing fat, sugar, protein, and salts. In gross appearance it resembles colostrum. The researches of Sinéty have shown that the mammary gland of the newly born contains cul-de-sacs lined with secreting cells, resembling those of the adult. During the period of secretion the gland is slightly reddened, its vessels turgid, and all the signs of functional activity are present. This condition in itself is of no practical importance, and in most cases, if left alone, the secretion ceases spontaneously after a week or ten days. It sometimes happens, however, that the presence of this secretion tempts the nurse or attendant to rub or squeeze the breast. Such manipulation occasionally leads to serious results by exciting a mastitis which may terminate in abscess. Mastitis is not a very rare condition, and although the inflammation is not usually severe, it may be serious and even fatal. The predisposing cause is the congestion which accompanies functional activity, usually in the second week. The exciting cause is most often some form of traumatism—undue pressure, the squeezing of the breasts, or rough handling by the nurse. Through abrasions or fissures thus produced, microörganisms find a ready entrance with the same result as in the adult. It seems possible that the germs may enter through the lactiferous ducts without any abrasion of the skin. Want of cleanliness is always a favorable condition for such infection.

The symptoms of mastitis usually begin during the second week of life. There is redness, swelling, and the usual signs of inflammation, which may terminate in resolution or in suppuration. The process may be limited to the mammary region, or a diffuse phlegmonous inflammation may be set up, and the case terminate fatally. In the female it is possible for the cicatrization which follows such an inflammation to interfere with the subsequent development of the gland. The general symptoms are restlessness, loss of sleep, disinclination to nurse, and loss of weight. In cases of diffuse phlegmonous inflammation the general symptoms are those of pyogenic infection.

The parts should be kept scrupulously clean, and on no account should squeezing of the breasts be permitted. They should be protected

by a cotton pad. If acute inflammation develops, it should be treated as a surgical affection.

INTESTINAL OBSTRUCTION

The most frequent causes of intestinal obstruction in the newly born are malformations of the intestine; rarely it may be due to pressure from tumors, or from a persistent omphalomesenteric duct or artery. The various pathological conditions present in intestinal malformations are considered in the chapter on Diseases of the Intestines. The most common seat of obstruction is at the anus, the bowel being normally formed throughout, lacking only the external orifice. The next most frequent condition is obstruction in the rectum, which may be due either to a membranous septum in the gut, or to obliteration of the tube for some distance. These rectal obstructions are readily recognized. By the examining finger or a bougie the lower limit of the obstruction can be made out, but there is no means by which the upper limit can be determined except by opening the abdomen. When the obstruction is above the rectum, localization is more difficult; but the most frequent seat is the duodenum. Of 38 cases collected by Gaertner, the seat of obstruction was the duodenum in 19 cases, the jejunum in 3, the ileum in 11, the colon in 6, the ileum and colon in 1. There is often obstruction at more than one point.

The symptoms vary with the seat and the degree of the obstruction. In atresia of the anus or rectum there is at first simply an absence of all discharges from the bowel. Later there is abdominal distention from dilatation of the sigmoid flexure and colon. After several days vomiting begins. If there is atresia of the duodenum or any part of the small intestine, vomiting begins early—usually by the second day of life—and it is persistent. Nothing is passed from the bowels after the first dark discharge of the contents of the colon, which is chiefly mucus. There is rapid asthenia, and death from inanition usually occurs in four or five days. The higher the obstruction the shorter the duration of life. If the condition is one of stenosis only, the symptoms are similar to those described but less severe, and life may be prolonged for several weeks, or even months. The constipation in these cases is not absolute. When the cause of obstruction is external pressure, the symptoms do not always begin immediately after birth. We once saw a child in whom nothing abnormal was noticed for the first three weeks, but at the end of that time there developed all the signs of acute intestinal obstruction. Laparotomy revealed a loop of intestine constricted by a tiny cord, which was probably the remains of the omphalomesenteric duct.

Cases of imperforate anus and membranous septum in the rectum are readily relieved by proper surgical treatment. In the other varieties of obstruction, whether in the rectum, in the colon, or in the small intestine, although life may be prolonged by the formation of an artificial anus, the ultimate result is almost invariably fatal, death usually occurring from marasmus during the early weeks of life.

DIAPHRAGMATIC HERNIA

This is due to a congenital deficiency in the diaphragm, which is usually on the left side. Of 118 cases collected by Livingston, 83 were on the left side, 18 on the right, 4 were central, 2 were double, in 1 the diaphragm was absent. With small openings only a single coil of intestine, with large ones a considerable part of the abdominal contents, may

FIG. 13, A.—DIAPHRAGMATIC HERNIA OF THE RIGHT SIDE, POSTERIOR VIEW. Child sixteen months old; died of pneumonia at three and a half years.

FIG. 13, B.—THE SAME, IMMEDIATELY AFTER ADMINISTRATION OF BISMUTH IN SUSPENSION. Stomach in the right thoracic cavity.

be found in the thorax. This causes displacement of the heart, usually to the right side, prevents the full expansion of the left lung, and if the deformity occurs early in intra-uterine life the lung may remain rudimentary. If a large deficiency exists, infants may live but a few hours; with smaller ones, life may be prolonged indefinitely.

The symptoms noticed soon after birth are usually cyanosis, rapid respiration, a sunken abdomen, an overdistended chest, and dyspnea. Children often live but a few hours. In those who survive a longer time

dyspnea is generally the most prominent symptom. It may be constant, it may occur in severe paroxysms, or there may be attacks of cyanosis often of great severity, these being produced by an accumulation of gas in the stomach or the thoracic part of the intestine. Other symptoms may at times suggest intestinal obstruction. The physical signs vary much from time to time. Sometimes those of pneumothorax are present; at others there is so much dulness with the feeble respiratory sounds, as to suggest fluid. The signs are usually upon the left side, with displacement of the heart to the right. A positive diagnosis can often be made by means of the X-ray after the administration of bismuth. (See Figs. 13, A, and 13, B.) The condition is not amenable to treatment.

CONGENITAL STRIDOR

This term has been given to a rather rare form of dyspnea seen in very young infants, beginning usually in the first days of life. Respiration is noisy and inspiration is accompanied by a marked croaking, or crowing sound, and with recession of the soft parts of the chest wall, which, especially at times of excitement, may be very great, yet there is no cyanosis and no subjective distress. In spite of the apparent difficulty of respiration the child seems comfortable. Expiration is usually easy and voice and cry are normal. The stridor diminishes when the child is very quiet but usually does not quite disappear even in sleep.

The symptoms begin in most cases immediately after birth or during the first week or ten days of life. They may increase for three or four weeks, then remain about stationary until the sixth or eighth month; after which with the growth of the larynx the dyspnea and stridor steadily diminish. By the end of the second year it is usually gone or heard only on occasion.

For our knowledge of this affection we are especially indebted to the observations of Thomson, of Edinburgh, who believes that the condition is primarily functional and due to a want of proper co-ordination of the respiratory muscles. Secondarily there is produced a folding of the epiglottis upon itself along the median line, so that its lateral borders approximate each other. In many of the cases reported, however, the change in the larynx seems to be rather a malformation especially of the epiglottis, which greatly narrows the superior opening of the larynx. Congenital stridor is favored by the soft collapsible character of the structures of the larynx in young infants and the strong suction force of inspiration.

The prognosis in most of these cases is good, the chief dangers being from intercurrent disease or from bronchopneumonia. Considerable

deformity of the thorax may be produced (pigeon breast) which may persist to later childhood.

The diagnostic features of congenital stridor are the noisy respiration with marked inspiratory dyspnea and crowing, with the absence of distress or subjective symptoms of any kind. It seems to be more frequent in delicate children. Conditions with which it may be confounded are papilloma of the larynx, laryngismus stridulus, catarrhal croup, and laryngeal spasm associated with adenoids. The first three of these are excluded by the history and by the absence of changes in the voice; the last one by the fact that the child is not a mouth breather, that the dyspnea is not increased by closing the mouth.

Congenital stridor is not amenable to special treatment. Should the dyspnea reach an alarming degree tracheotomy may be performed. The indications are to maintain the child's general nutrition and to protect him, so far as possible, from diseases of the upper respiratory tract.

SCLEREMA

Sclerema is a condition characterized by hardening of the skin and subcutaneous tissues. It may occur in circumscribed areas or extend over nearly the entire body. It affects infants who are very feeble and usually terminates fatally. Although sclerema is chiefly seen in the first days of life it is not limited to the newly born, but may occur at any time during the first few months. It is not to be confounded with edema of the newly born, with which condition it is, however, sometimes associated. From published reports it appears to be of not very infrequent occurrence in Europe, chiefly in large foundling asylums. In America, sclerema is a rare disease. In the newly born, sclerema affects those who are premature or very feeble, sometimes those who are syphilitic. Later it may follow any condition leading to extreme exhaustion, especially the different forms of diarrheal disease.

The first thing to attract attention is usually the induration of the skin. It is often seen first in the calves or the thighs, sometimes first in the cheeks, but soon extends over the greater part of the body. It is especially marked in the cheeks, buttocks, and back, and regions where adipose tissue is abundant. It may affect the body uniformly or in circumscribed areas. The skin may be smooth or it may appear somewhat lobulated. The color is normal or slightly bluish, often tinged with yellow. The lips are blue, and the capillary circulation so feeble that after pressure upon the nails the blood returns slowly or not at all. The limbs are stiff and board-like. The skin is cold to the touch, and often the thermometer in the axilla will not rise above 90° F. In

one recorded case the axillary temperature was only 71° F. The general feeling of the body has been well likened to that of a half-frozen cadaver. The tongue and the mucous membrane of the mouth are cold; no radial pulse can be felt; the respiration is slow, irregular, embarrassed, and at times the movements of the thorax are scarcely perceptible. The cry is a feeble whine, scarcely audible. The duration of the disease is usually from three to four days. Death occurs slowly and quietly. If recovery takes place there is gradual improvement in the circulation and nutrition, and, later, a disappearance of the areas of induration.

The causes of sclerema are general, the most important factors being loss of fluids, great feebleness with lowering of the body temperature, and, in consequence, hardening of the subcutaneous fat. There are no essential lesions in this disease. Atelectasis is often present, and may have something more than an accidental association, as incomplete aëration of the blood is no doubt a factor in the production of the symptoms. Microscopical examination in typical cases has shown the skin to be normal.

The prognosis is very bad, because of the grave conditions of which it is the expression, but it is not invariably fatal. In its milder forms, where treatment is begun early, recovery may take place. The diagnosis is to be made from edema by the fact that there is no pitting upon pressure, by the rigidity of the body, and by the great reduction in the temperature. The most important thing in treatment is artificial heat; nothing but the incubator is efficient. In addition to this, care should be taken to promote the general nutrition by careful feeding and by all other means possible.

INANITION FEVER

The term *inanition fever* is not altogether a satisfactory one; but, until these cases are better understood, it is adopted because it emphasizes the very close connection which exists between the rise of temperature and the condition of inanition or starvation. Under this heading are included cases seen during the first five days of life—generally from the second to the fourth day—in which there is an elevation of temperature, apparently due to the fact that the infant gets very little, frequently nothing at all, from the breast at which he is being suckled. It is further characteristic of these cases that the temperature falls when the child is put upon a full breast, or when artificial feeding is begun, or even when water is administered, if freely given. Some have ascribed the symptoms to uric-acid infarction of the kidneys.

So far as our knowledge goes, the first to call attention to this condition was McLane (New York), who in 1890 reported to one of the

medical societies an extraordinary case of hyperpyrexia in a newly-born child. The infant was found on the sixth day with a temperature of 106° F., near which point it had remained for three days. The child was being suckled at a breast which was found to be absolutely dry. A wet-nurse was procured, the temperature fell to normal in a few hours, and the child, which when first seen was apparently in a hopeless condition, was soon perfectly well.

Since that time very extensive observations, extending to upward of three thousand cases, have been made at the Sloane and the Nursery and Child's Hospitals, which have established the fact that a rise of temperature to 102° or even 104° F. is quite common in newly-born infants during the first few days. This fever is accompanied by no evidences of local disease, and ceases in nursing infants with the establishment of the free secretion of milk. The fall in temperature is often rapid, dropping to the normal in a few hours after having continued for three or four days, and in a large number of cases it does not rise again.

The following case is a fairly typical one of the more severe form: The patient was the second child, the first having died at the age of ten days, from no disease, it was said, but simply from exhaustion. At birth the infant, a boy, weighed eight and a quarter pounds and was apparently vigorous. During the first forty-eight hours his loss in weight was five and a half ounces and his condition good. He was seen on the evening of the third day. In the preceding twenty-four hours he had lost eight ounces in weight, and the temperature had gradually risen, until at the time of our visit it was 102.8° F. The body was limp, the child making no resistance to examination. He cried with a feeble whine; the restlessness of the early part of the day having given place to complete apathy. The lips and skin were very dry, the fontanel sunken, the pulse weak. As the father, a physician, expressed it, "he had been wilting through the day like a flower in the sun." Although put to the breast regularly, the child had apparently obtained very little. It was, in fact, impossible to express any milk from the mother's breasts. Water was freely given and a wet-nurse secured in a few hours. The first milk was taken from the wet-nurse at 11 P.M., and the temperature, which fell gradually during the night, was normal the next morning and did not rise again. (See chart, Fig. 14.) During the succeeding four days the child gained eighteen ounces in weight, and at the end of a week was as well as an average infant of his age.

The symptoms are so uniform and so characteristic that they make for these cases of fever a class by themselves. The frequency with which this is seen is shown by the following statistics: Among 200 infants taken successively at the Nursery and Child's Hospital, 20 had fever during the first five days, reaching 101° F. or over, which was not

explained by ordinary causes and followed the course above described. In 500 successive children born at the Sloane Hospital, there were 135 with a similar fever. It was seen in vigorous infants as well as in those who were delicate. The usual duration of the fever was three days, the temperature generally touching the highest point upon the third or fourth day of life. In about two-thirds of the cases the temperature did not rise above 102° F.; in 9 it was 104° F. or over, the highest recorded being 106° F. The fall was generally quite abrupt, although not always so.

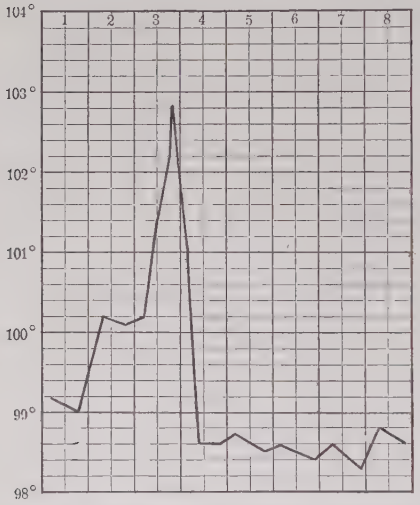


FIG. 14.—TEMPERATURE CHART. INANITION FEVER.

Daily weighings, which were made in these cases, showed that the infants continued to lose weight while the fever continued, and that the loss almost invariably exceeded by several ounces that of the children who had no fever. The maximum loss noted was twenty-eight ounces. In quite a large number of cases it exceeded twenty ounces. As a rule the infants began to gain in weight when the temperature remained at the normal point, but not until then.

The symptoms presented by these infants were a hot, dry skin, marked restlessness, dry lips, and a disposition to suck vigorously anything within reach. With very high temperature there were considerable prostration and weakened pulse. In the less severe cases there were only crying and restlessness. The rapidity with which the symptoms disappeared when the children were wet-nursed or properly fed, was very striking.

It is important that this fever should be recognized, because it gives at times the first warning of a condition which may prove fatal. The extra loss of ten or fifteen ounces in the first week is a serious handicap to newly-born infants, the effect of which may last for several weeks. The temperature of every child should be taken during the first week. All the usual local causes of fever are first to be excluded by a physical examination. This fever can hardly be confounded with that due to pyogenic infection, which rarely begins before the fifth or sixth day.

The treatment is simple, viz., to give water regularly every two hours, in quantities up to an ounce at a time if required by the thirst of the child. This should be done in every case where the temperature reaches 101° F. When the temperature does not at once begin to fall, the infant

should be put upon another breast or artificial feeding should be begun. Examination of the breasts from which the child has been nursing will usually reveal the fact that the secretion of milk is very scanty and often entirely absent.

Such a fever we have occasionally seen in older infants, usually in those who are nursing dry breasts or where fluid food and water have been withheld because of some gastric disturbance. It yields as promptly to treatment as does the same condition in the newly born.

SECTION II

NUTRITION

CHAPTER I

Nutrition in its broadest sense is the most important branch of pediatrics. In no other field and at no other time of life does prophylaxis give such results as in the conditions of nutrition in infancy. The largest part of the immense mortality of the first year is traceable directly to disorders of nutrition. The importance of correct ideas regarding this subject can hardly be overestimated. The problem is not simply to save life during the perilous first year, but to adopt those means which shall tend to healthy growth and normal development. The child must be fed so as to avoid not only the immediate dangers of acute indigestion, diarrhea, and marasmus, but the more remote ones of chronic indigestion, rickets, scurvy, and general malnutrition, since these conditions are the most important predisposing causes of acute disease in early life.

One of the difficulties has always been that temporary success may mean ultimate failure. If the injurious effects of improper feeding were immediately manifest there would be very much less of it than exists at the present time. Many things are valuable as temporary foods, which when used permanently are injurious. No better illustration of this is seen than in the too exclusive use of the carbohydrate foods. Infants fed upon many of the proprietary foods often grow very fat, and for the time appear to be properly nourished. The effect of the absence from the diet of some of those elements which are of vital importance may not be evident for months. The physiological laws regarding the requirements of the growing organism cannot be ignored without serious consequences, which will sooner or later be evident. Correct ideas of infant feeding are based upon a knowledge of these laws. An accurate understanding of fundamental principles is essential to success and the vast majority of failures may be ascribed to ignorance or disregard of them.

THE FOOD CONSTITUENTS AND THE PURPOSES THEY SUBSERVE IN NUTRITION

In infancy and childhood, as in adult life, the elements of the food are five in number: protein, fat, carbohydrates, mineral salts and water.[1] The forms in which they must be furnished to the child, and the relative quantities in which they are demanded, are different from those required by the adult. One reason for this difference is the delicate structure of the organs of digestion in infancy, and their inability to assimilate certain forms of food. Again, provision must be made not only for the natural waste of the body, but for its rapid growth, nearly trebling in size, as it does, during the first twelve months.

Amount of Food Required.—The attempt has been made to determine accurately the amount of food which an infant should receive during the first year. The food of infants who were thriving satisfactorily has been measured, and many metabolism experiments have been carried out for the purpose of definitely settling this question. While all these observations have shed much light upon the subject we are not yet able to reduce to a mathematical formula the amount of food which shall be given to keep an infant in health and enable him to develop normally. As yet, the results of intelligent clinical observation of infants form our best guide as to food requirements. Individual infants, though they may all be equally healthy, differ very much in this respect, depending upon their weight, their size and also much upon their physical activity. One that is particularly active or restless requires more food than does one who is very quiet and lethargic. The size of the body, or the surface area, is doubtless of much more importance in estimating food requirements than the weight, but the latter is so much more easily determined that it has come into general use in estimating the amount of food to be given *per diem*. It is a matter of general agreement that the requirements of the infant, relatively to the weight, are greatest during the first months of life and become gradually less, so that by the end of the first year they are only about three-fourths as great as during the first month. Heubner placed the child's needs at 100 calories per kilo (45 per pound) during the first quarter year and at 70 per kilo (30 per pound) during the last quarter year. These figures have been much used as an arbitrary standard, and indeed they do furnish an excellent starting point for the feeding of an individual child, but they can hardly do more. The subsequent variations in the amount of food must be decided by the child's demon-

[1] There are other substances whose presence in the food is vitally necessary for life, such, for instance, as the vitamins. They exist in most of the common articles of food. Their chemical composition is uncertain. Their absence produces definite symptoms.

strated needs and his digestive capacity; but wide variations from these averages, whether above or below them, are usually found to be either inadequate or disturbing. Again, these amounts are designed for healthy infants with good digestion. Sick children, or those suffering from digestive disturbances, must be fed according to the capacity of their digestion. The expression in calories of the energy value of the food does not imply that this is to be regarded as a method of feeding. It is only a method of stating the amount of food which a child is receiving, in a more accurate and scientific way than others that have been employed, e.g., the number of ounces given daily, which really represents only the volume of the food, or tells rather the amount of water in which the food is given.

The calculation of the total food in terms of energy units is chiefly of assistance in enabling one to recognize readily whether an infant is receiving too much or too little food.

In determining the calories of the food it is calculated that:

1 gram of	fat	yields	9.3	calories
1 " "	carbohydrate	"	4.1	"
1 " "	protein	"	4.1	"

Protein.—Protein is essential to life, since it is the only kind of food which is capable of replacing the continuous nitrogenous waste of the cells of the body upon which health depends. Protein is also indispensable for growth. In the adult only the requirements of repair are to be supplied. In the child a much larger amount is demanded to provide for growth. Without the aid either of the fats or the carbohydrates, protein may sustain life for a considerable time; but in so doing a great excess of such food is required. When fats and carbohydrates are added to the food much less protein is required to replace the nitrogenous waste.

Of all the forms in which protein food may be furnished to the body, in proportion to its nitrogen content, milk taxes the digestive organs least. Furthermore, there is no other form of protein in which those amino-acids which have been shown to be essential for growth are so abundantly supplied as in milk. These facts are of great importance and indicate the superiority of milk as a food for infants, particularly during the first year. The protein of woman's milk is very readily digested. Regarding the protein of cow's milk there is no doubt that the view formerly held that it was difficult of digestion was erroneous. On the contrary, under most conditions it is digested and absorbed with facility. During most of the first year, milk furnishes all the protein that is needed for proper nutrition. But as cow's milk protein is low in certain important amino-acids, a larger amount of it must be given than the

protein contained in woman's milk, or growth will suffer. During the second year meat, eggs, etc., may add to the protein of the diet.

The digestion of protein is begun in the stomach but is principally carried on in the intestines. The albumoses and peptones produced by gastric and pancreatic digestion are broken up as the result of the action of the erepsin of the intestinal juice into polypeptids and finally into amino-acids. It is as amino-acids that nearly all of the nitrogen is absorbed. In almost all circumstances, the nitrogen of the protein is well absorbed. The tendency to retain nitrogen is one of the striking attributes of the infant. He retains this if it is in any way possible and may continue to do so even when losing greatly in weight. This may be taken as an indication of the great efforts that the body makes to further growth.

The nitrogen which is not retained is largely excreted by the urine. The nitrogen of the feces is relatively small in amount, is influenced somewhat by the kind of food and is in considerable part derived from the intestinal secretions which themselves contain a certain amount of protein.

In artificial feeding it has been maintained that a large excess of nitrogenous products must be disposed of by digestion and elimination and that this taxes the organs of digestion and excretion. It may be said that there is at the present time no proof that milk protein even in considerable excess is dangerous to the welfare of the infant.

The prolonged use of a diet in which the protein is insufficient in amount or defective in character produces a certain definite group of symptoms which are not always referred to their proper cause. In infants the most striking are slower growth, anemia, poor circulation, feeble muscular power, disinclination to exertion, and various functional nervous disturbances. Such children are often very fat. Vegetable proteins do not seem able permanently to take the place of animal proteins in the food of young infants for the reason that most of them are deficient in some of the essential amino-acids. Since in milk and in fact in almost all the foods of the infant a very constant relation exists between the protein and the salts, it is somewhat difficult to separate symptoms due to low protein from those due to low salts; the two are often combined.

The ingestion of casein in large amount produces in infants, large, dry, light colored stools, often of an alkaline reaction. They also contain a high proportion of mineral salts. With these stools there is usually constipation. While this effect in health is one not to be desired, it is decidedly advantageous in diarrhea to combat the fermentation produced by carbohydrates and fats. For this reason, as will be seen later, protein in large amount is a valuable therapeutic remedy for many intestinal conditions during infancy and childhood.

Fats.—Fats are a most important source of energy to the body, their caloric value being a little more than twice as great as that of either the carbohydrates or the protein. They save nitrogenous waste and increase the body weight. The large amount of fat stored up in the subcutaneous tissues in infancy is one of the best evidences of health.

The amount of fat received by a breast-fed infant is relatively much greater than that given to adults in a normal diet. A well-nourished, nursing infant weighing fifteen pounds actually receives about one-half as much fat as is allowed in a ration for an adult doing moderate work, who weighs ten times as much. There can be no doubt that fat is beneficial for infants and that those who can take a reasonable amount of fat thrive better than those who can not. It is also plain that the one of the ingredients of cow's milk most difficult for the infant to digest is the fat.

Fats may, for a considerable time, be largely replaced by the carbohydrates; but nutrition suffers if this substitution is complete or long continued. Fats are acted upon very slightly in the stomach, although they greatly retard the emptying of the stomach. Their digestion in the intestine is, under normal conditions, very complete, and only a small percentage of the fat passes through the intestine unchanged. Under normal conditions, from 80 to 90 per cent of the fat ingested is absorbed either as fatty acids or as soaps. No neutral fat can be absorbed.

When the diet contains fat and protein in considerable quantity and is low in carbohydrate, stools are formed consisting largely of calcium and magnesium soaps, and the loss of these substances may even be so great that a negative balance of these minerals results. In certain circumstances, fats in the intestine may be decomposed and acids formed, but this rarely occurs unless carbohydrates in excess are also given. As a result of this fermentation, irritating products—chiefly the lower fatty acids—are formed, and these readily provoke diarrhea. In the diarrheal stools there may be sufficient potassium and sodium loss to bring about a negative balance of these minerals. The influence of the fat, therefore, upon the mineral balance is an important one.

Carbohydrates.—Although, like the fats, these can not replace the nitrogenous waste of the body, they are important aids in sparing the protein, and in this respect they are even more valuable than the fats. The carbohydrates are partly converted into fat, and may thus increase the body weight. They are capable of replacing the fat-waste of the body.

Carbohydrates are the most abundant of the solid elements of the food, although they form a smaller percentage of the entire quantity of food in infancy than in adult life. The soluble carbohydrates which are used as foods for infants are: milk sugar, cane sugar and mixtures of maltose and dextrin. Maltose in a pure form is not used on account of its cost and because it has no advantages. Mixtures containing maltose

have distinct advantages in some circumstances. Since all sugars are finally converted into glucose, they are, to a certain extent, interchangeable. Milk sugar has an advantage in not fermenting with the common varieties of yeast present in the stomach as do both maltose and cane sugar. Except for this, there is not much to choose between milk sugar and cane sugar. Gain in weight is satisfactory with either, and they are equally safe. They have the same disadvantages and dangers in that they readily undergo fermentation in the intestine by the action of bacteria. As a result of this fermentation, lower fatty acids are formed not only from the sugar but also from the fats which are present in the food, with the result which has been described above under the fermentation due to excessive quantities of fat.

The ability of the young infant to digest starches is relatively feeble, although this power does exist to some degree from birth; but the greater part of the carbohydrates required should be furnished in the form of sugar. To infants of four months and over, starches may at times advantageously be added to the diet, and after seven or eight months the quantity may be considerably increased. But in whatever form or quantity used thorough cooking is necessary.

The advantages of the carbohydrates as foods depend upon the ease with which they are digested and absorbed. They are at a great disadvantage on account of the readiness with which all of them, and especially the sugars, undergo fermentation in different parts of the alimentary tract. The mixtures of maltose and dextrin, for some unexplained reason, are often safer to give to children who have suffered from diarrhea. While they themselves have a tendency to cause rather loose, brownish stools, they do not so readily undergo excessive fermentation and may sometimes be given with safety when other sugars, especially lactose, would cause serious disturbances.

A diet consisting too exclusively of carbohydrates often leads to a rapid increase in weight, but it is not accompanied by a proportionate increase in strength. Infants so fed have but little resistance, and many of them become rachitic. The easy digestion of foods consisting chiefly of soluble carbohydrates, such as sweetened condensed milk and the proprietary infant foods, and the rapidity with which children so fed gain in weight, lead to a great misapprehension in regard to their value as foods. The ultimate results of such one-sided feeding, if long continued, are almost invariably disastrous.

In building up the cells of the body the protein is first in importance, but in the production of energy the fats and the carbohydrates have a greater value. In a proper diet all of these elements are represented.

Mineral Salts.—The great importance of the mineral salts in the nutrition of infants and children has only recently been appreciated.

These salts are important not only for growth, but for all the physical and chemical processes which are carried on in the body. If they are not furnished in sufficient amount in the food, or if conditions exist in which their absorption, retention and utilization are interfered with, all the functions of the body are disturbed and life may be jeopardized. Except in the case of infants fed upon the proprietary foods, salts are very seldom lacking in the food. Those who receive woman's milk usually receive an adequate supply; and those who are fed on cow's milk receive not only the salts required, but a very considerable excess of them, often two or three times the requirements of the child. This excess apparently does no harm, as it is either not absorbed or is excreted by the intestines or kidneys. The mineral salts form from 10 to 35 per cent of the dried matter of the normal stool. For perfect nutrition not only must all the mineral salts be furnished in the food but the other elements of the food must not have an injurious effect upon their retention. The chief dangers to the retention of sodium and potassium arise from fermentation of carbohydrates and fats in the intestine. Disturbances in the metabolism of the salts are very frequent and are no doubt at the basis of many common nutritional disturbances of infancy.

Water.—The food of all young mammals consists of from eighty to ninety per cent of water. This is needed for the solution of certain parts of the food, such as the sugar, the salts, and some of the protein, and for the suspension of other protein and the emulsified fat. All the food is thus dissolved or very finely divided so as to be more readily acted upon by the delicate digestive organs of the infant. Water is needed also in large quantities for the rapid elimination of the waste of the body.

The amount of fluid required by the infant, in proportion to his size and weight, is much greater than that required by the adult. During early infancy an infant should receive daily an amount of fluid equal to about one-fifth his body weight. As it is practically impossible to give to a young infant any considerable part of this as water, this figure gives us an important guide as to the volume of the food to be given daily to an artificially-fed infant. The passage of a large amount of urine of low specific gravity is one of the physiological conditions of infancy and sufficient water must be furnished to the infant to make this possible. It is not therefore a matter of indifference whether we give the daily amount of food with twenty or with thirty-five ounces of water. After six months fluids can be given in the form of fruit juices, broth, etc., and, besides, the older infant will usually take water in proper amount without difficulty, so that the same relation of the volume of food to the body weight need not be maintained. Of the water received it is estimated that 59 per cent is eliminated by the kidneys; 33 per cent by the lungs, 6 per cent by the intestines, and that from 1 to 2 per cent is retained.

CHAPTER II

THE INFANT'S DIETARY

WOMAN'S MILK

Woman's milk is the ideal infant-food. A knowledge of its character, composition, and variations is indispensable, for upon this knowledge are based all our substitutes for woman's milk when this can not be obtained.

Woman's milk is a secretion of the mammary glands and not a mere transudation from the blood-vessels; although under abnormal conditions it may partake more of the character of a transudation than a secretion. A few drops may be squeezed from the breasts before parturition; generally speaking, however, it is only present after delivery. During the first two days the secretion is scanty. Usually upon the third or fourth day it becomes well established, although it may be delayed many days longer and yet become abundant. During the period of lactation, milk is constantly formed in the mammary glands, but the process is more active while the child is at the breast.

Physical Characters.—Woman's milk is of a bluish-white color and quite sweet to the taste. When freshly drawn its reaction is amphoteric to litmus, or slightly acid to phenolphthalein. The specific gravity varies between 1.026 and 1.036, the average being 1.031 at 60° F. On the addition of acetic acid only a slight coagulation is seen, this being in the form of small flocculi, and never in large masses as is the case in cow's milk. Microscopically, there are seen great numbers of fat-globules nearly uniform in size and some granular matter. Occasionally there are present epithelial cells from the milk-ducts or from the nipple.

Early Milk.—The secretion of the early days of lactation to which the term "colostrum" has been given, differs quite markedly from the later milk. It is of a deep-yellow color, which is chiefly due to the colostrum-corpuscles. It has a specific gravity of 1.030 to 1.035, a strongly alkaline reaction, and is coagulated into solid masses by heat, and sometimes the milk of the first days coagulates spontaneously. It is very rich in protein and in salts. Microscopically the fat-globules are of unequal size, and there are present large numbers of granular bodies known as colostrum-corpuscles. These are four or five times the size of the milk-globules, and they are probably leucocytes in which are contained numerous fat granules. They are much larger than ordinary leucocytes and are nucleated.

The colostrum-corpuscles are very abundant during the first few days,

but under normal conditions they are not found after the tenth or twelfth day.

Composition of Colostrum

	First and second days	Two to ten days
Fat	2.38	3.00
Sugar	3.38	7.50
Protein	8.60	2.25
Ash	0.37	0.30
Water	85.27	86.95
	100.00	100.00

The characteristic features of colostrum milk continue for a period varying from five to ten days; but it is not until about the end of the first month that the milk assumes its stable or "mature" character. The milk of the intermediate period is sometimes spoken of as "transition milk." It shows a marked but gradual fall in the protein and ash, and a moderate rise in the fat and sugar until the composition of mature milk is reached; after this time no constant or regular changes are seen in the proportion of the different constituents until near the close of lactation.

Daily Quantity.—Exact information upon this point is difficult to obtain. There are recorded, however, extended observations made with great care upon a number of cases. The eight cases quoted below [1] were

[1] Haehner's cases (Jahrb. f. Kinderh., xv, 23; xxi, 314). Case I. Female; birth-weight, 7 pounds 14 ounces (3,100 grams). First week, lost 1½ ounce (45 grams); after this gained steadily during the twenty-three weeks of observation; from second to ninth week, average weekly gain 8 ounces (241 grams); from tenth to eighteenth week, average gain 4½ ounces (138 grams); from nineteenth to twenty-third week, average gain 4 ounces (130 grams); weight at the end of twenty-third week, 14 pounds (6,690 grams).

Case II. Male; birth-weight 6½ pounds (2,950 grams). Loss, first week, 3 ounces (90 grams); after this gained steadily during the eleven weeks of observation; from second to eleventh week, average weekly gain 7½ ounces (214 grams); weight at end of eleventh week, 11 pounds 2 ounces (5,045 grams).

Case III. Female; birth-weight 3 pounds 9 ounces (1,620 grams). Gain, first week, 1½ ounce (45 grams); during the succeeding twenty-one weeks of observation, average weekly gain 5 ounces (141 grams); weight at the end of twenty-second week, 10 pounds 3 ounces (4,620 grams).

Laure's case (Thèse, Paris, 1889). Female; birth-weight 8 pounds 13 ounces (4,000 grams); loss, first week, 8 ounces (225 grams); after this gained steadily during the nine weeks of observation, on an average 9½ ounces (268 grams) weekly; at the end of ninth week, weight 13 pounds 3½ ounces (6,000 grams).

Ahlfeld's case (Deutsch. Ztschr. f. Prakt. Med., 1878). Birth-weight 7 pounds 14 ounces (3,100 grams). Observations continued from fourth to thirtieth week. During first ten weeks, average weekly gain 5¾ ounces (161 grams); from eleventh to twentieth week, 7½ ounces (214 grams); from twenty-first to thirtieth

all healthy infants, nursing exclusively and gaining steadily in weight.

From these observations, and others less extended, the average daily quantity of milk secreted under normal conditions of health may be assumed to be pretty nearly as follows:

	Approximately.
At the end of the first week	10 to 16 oz. (300 to 500 c.cm.)
During the second week	13 to 18 oz. (400 to 550 c.cm.)
During the third week	14 to 24 oz. (430 to 720 c.cm.)
During the fourth week	16 to 26 oz. (500 to 800 c.cm.)
From the fifth to the thirteenth week	20 to 34 oz. (600 to 1,030 c.cm.)
From the fourth to the sixth month	24 to 38 oz. (720 to 1,150 c.cm.)
From the sixth to the ninth month	30 to 40 oz. (900 to 1,220 c.cm.)

It will be noted that the amount increases very rapidly up to about the eighth week, and after this much more slowly. The amount of milk varies also with the demands of the child in a very striking way.[1] The

week, 6 ounces (168 grams); at the end of the thirtieth week, weight 18 pounds 9½ ounces (8,435 grams).

Feer (Jahrb. f. Kinderh., xlii, 195). Three cases.

In all these cases the amount of milk was determined by weighing the infant both before and after every nursing during the entire period of observation. The following table gives in a condensed form the daily quantity of milk in these cases:

Time.	Haehner's 1st Case.	Haehner's 2d Case.	Haehner's 3d Case.	Laure's Case.	Ahlfeld's Case.	Feer's 3 Cases. Average.
	Grams.	Grams.	Grams.	Grams.	Grams.	Grams.
1st day	20	75	20	....	....	....
2d day	176	135	45	....	....	....
3d day	265	325	70	125	....	....
4th day	420	295	99	222	....	....
5th day	360	290	124	400	....	256
6th day	374	340	136	475	....	(average
7th day	423	350	156	500	....	1st week)
Average 2d week	497	423	229	556	....	....
Average 3d week	550	468	314	730	....	....
Average 4th week	594	531	379	810	576	610
Average 5th week	663	561	447	944	655	667
Average 6th week	740	661	472	978	791	753
Average 7th week	880	681	525	1,038	811	802
Average 8th week	835	730	568	1,024	845	815
Average 9th week	766	665	584	1,085	810	820
Average 10th to 13th week	796	....	600	....	869	795
Average 14th to 17th week	807	....	673	....	983	845
Average 18th to 23d week	870	....	709	....	1,029	919
Average 24th to 30th week	....	....	....	....	1,145	1,002

[1] There are a number of recorded instances in which the amount of milk secreted has been quite extraordinary—in some cases as much as four quarts daily. Lactation in exceptional instances also is unduly prolonged. We know of one well authenticated American case in which it continued for seven years. Among the Japanese it is frequent for it to continue up to three or four years. Among the Hottentots and other savage races lactation may be prolonged until the sixth or seventh year.

quantities mentioned can not be taken as an absolute guide to the amount of food to be given to bottle-fed infants. Breast milk contains an average of twelve per cent solids: while the modification of cow's milk best suited to the early months seldom has more than from nine to eleven per cent solids. For this period, therefore, somewhat larger quantities are needed than of breast milk.

A comparison of the daily amount of milk taken with the weight of the child at the different periods, showed that both during the early and the later periods the larger children took not only more milk, but considerably more in proportion to their body weight than did the smaller ones. This harmonizes with the common observation that small children are much more likely to be overfed than large ones.

The average quantity taken at one nursing by five of the children previously mentioned was as follows:

	Approximately.
During the first week	$\frac{5}{8}$ to $1\frac{1}{2}$ oz. (18 to 45 c.cm.)
During the second week	1 to 3 oz. (30 to 90 c.cm.)
During the third week	$1\frac{1}{2}$ to 4 oz. (45 to 120 c.cm.)
During the fourth week	$1\frac{1}{2}$ to $4\frac{1}{2}$ oz. (45 to 140 c.cm.)
From the fifth to the seventh week	2 to 5 oz. (60 to 150 c.cm.)
From the eighth to the eleventh week	$2\frac{1}{2}$ to $5\frac{1}{2}$ oz. (75 to 160 c.cm.)
During the fourth month	3 to 6 oz. (90 to 180 c.cm.)
During the fifth month	$3\frac{1}{2}$ to $6\frac{1}{2}$ oz. (110 to 200 c.cm.)
During the sixth month	4 to 7 oz. (120 to 220 c.cm.)

Between the limits mentioned the greater number of cases will undoubtedly fall. The amount taken at one time is, however, modified by the frequency of nursing, and is therefore not so good a guide to the amount of food required, as is the quantity taken in twenty-four hours.

Composition.—According to the analyses of Pfeiffer, Koenig, Leeds, Harrington, Adriance, Courtney and Fales and others, the composition of human milk is as follows:

	Normal Average (Mature Milk)	Common Healthy Variations.
	Per cent	Per cent
Fat	3.50	3.00 to 5.00
Sugar	7.50	6.50 " 8.00
Protein	1.25	1.00 " 2.00
Ash	0.20	0.18 " 0.25
Water	87.55	89.32 " 84.75
	100.00	100.00 100.00

In the older analyses the percentage of protein was almost invariably made too high and the sugar too low. After the first month there are no regular changes in composition until near the end of lactation. This is a point to be borne in mind in the selection of wet-nurses.

Milk also contains certain natural ferments which, though little understood, are believed to have a function in digestion.

Protein.—The important forms of protein are casein and lactalbumin; several others, lactoglobulin, lactoprotein and nuclein are also described. The casein is in suspension by virtue of the presence of calcium phosphate in the milk, with which it is probably in combination. It coagulates only slightly with rennet, while acetic acid produces a loose flocculent precipitate. The lactalbumin resembles the serum-albumin of the blood. In woman's milk it is nearly twice as abundant as casein. Its proportion to casein is nearly twelve times as great as in cow's milk.

The total protein of normal mature milk is usually between 1.0 and 1.5 per cent. In abnormal specimens the variations are from 0.7 to 3.5 per cent. The total protein is highest in the colostrum period; it falls steadily to the latter part of the first month. After this time, during the mature period, the variations are slight, but it tends to fall slowly. Toward the end of lactation the proportion of protein falls quite rapidly.

Fat.—This exists in the form of minute globules, which are held in a state of permanent emulsion by the albuminous solution in which they are suspended. The fat of woman's milk is chiefly made up of the neutral fats—palmitin, myristin, stearin and olein; the last mentioned predominating. There are also small quantities of free fatty acids, but these are much less in amount than in cow's milk. The fat of woman's milk is relatively low in volatile fatty acids, compared with that of cow's milk. The proportion of fat is subject to even wider variations than is that of the protein, 3.5 per cent being taken as the normal average. In a series of thirty-four analyses the fat varied between 1.12 and 6.66 per cent. The highest percentage we have known was 10.91. In forty-three analyses by Leeds, the variations were between 2.11 and 6.89 per cent. The proportion is very little affected by the period of lactation.

Sugar.—The sugar is in solution. Its proportion is more nearly constant under all conditions than any other constituent of milk. The ordinary variations are usually within the limits of 6.5 and 8 per cent.

Ash.—The average proportion of inorganic salts is 0.20 per cent, or a little more than one-fourth that of cow's milk. The percentage composition of the ash as compared with that of cow's milk is given in a subsequent chapter.

With the exception of calcium phosphate nearly all the salts are in solution. The milk of the first few days is very rich in salts, chiefly owing to the large proportion of sodium and potassium chlorid; after the first month the normal variations[1] are slight and inconstant.

[1] From thirty-eight analyses by Courtney and Fales at the Babies' Hospital, the following average figures were obtained of the composition of the

The Examination of Milk.—The exact composition of human milk is to be determined only by a complete chemical analysis. Many variations in composition the physician may readily ascertain for himself by simple methods.

The *quantity* of milk secreted by the breasts may be estimated by the quantity which may be drawn by a breast-pump, although this is not a very reliable test. If the child nurses habitually thirty or forty minutes, the probabilities are very strong that the milk is scanty. If the breasts at nursing time are full, hard, and tense, the supply is probably abundant. If the breasts are soft and flabby, and appear to fill only while the child is nursing, it is almost certain that the quantity is small. The only really reliable test is weighing the infant just before and after nursing, upon an accurate pair of scales sufficiently sensitive to indicate half-ounces. This should be repeated several times in the day.

The *reaction* of woman's milk even when freshly drawn is rarely alkaline, being amphoteric to litmus, or slightly acid to phenolphthalein.

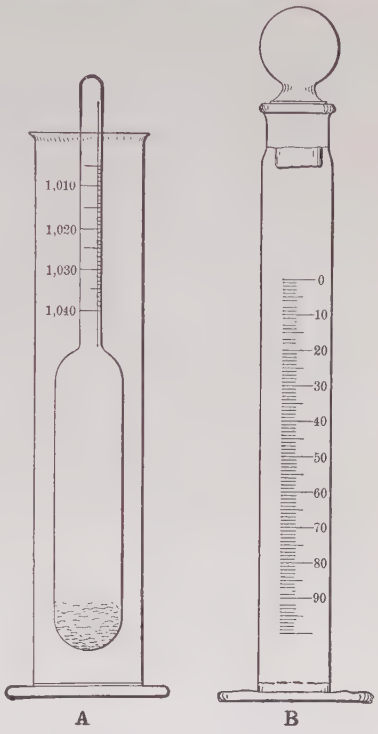


Fig. 15.—Apparatus for Examination of Woman's Milk. The authors' lactometer and cream-gauge.[1]

The *specific gravity* may be taken with any small hydrometer graduated from 1.010 to 1.040 (Fig. 15, A). The specific gravity is lowered by the fat, but increased by the other solids. An ordinary urinometer will answer every purpose, the only

ash of woman's milk at the different periods of lactation. (Amer. Jour. Diseases of Children, Oct., 1915.)

Period	No. of Analyses	Total Ash	CaO	MgO	P_2O_5	Na_2O	K_2O	Cl
Colostrum (1–12 days)	5	.3077	.0446	.0101	.0410	.0453	.0938	.0568
Transition (12–30 days)	6	.2407	.0409	.0057	.0404	.0255	.0709	.0580
Early mature (1–4 months)	9	.2056	.0486	.0082	.0342	.0154	.0539	.0351
Middle mature (4–9 months)	8	.2069	.0458	.0074	.0345	.0132	.0609	.0358
Late milk (10–20 months)	10	.1978	.0390	.0070	.0304	.0145	.0575	.0442

[1] The authors' apparatus may be obtained from Eimer & Amend, Eighteenth Street and Third Avenue, New York.

difficulty being the quantity which is required to float the instrument.

Microscopical Examination.—The microscope may reveal the presence of fat globules, colostrum corpuscles, blood, pus, epithelium, and granular matter. Colostrum corpuscles are abnormal after the twelfth day; pus and blood are always abnormal. The presence of any of these elements necessitates the suspension of nursing, at least temporarily. But little importance can be attached to the size and appearance of the fat globules as affecting the nutritive properties of the milk.

The Determination of Fat.—The simplest method is by the cream-gauge (Fig. 15, B). Its results are only approximate, but in most cases sufficiently accurate for clinical purposes. The tube is filled to the zero mark with fresh milk, which stands, corked, at room temperature for twenty-four hours, when the percentage of cream is read off. The ratio of this to the fat is approximately five to three; thus 5 per cent cream indicates 3 per cent fat, etc.

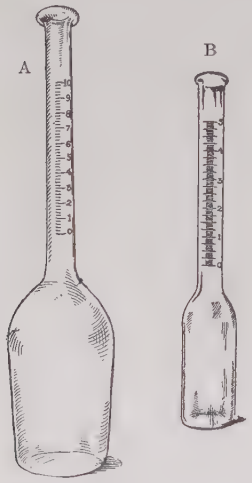


Fig. 16.—Tubes for Determining the Fat in Milk. A, Babcock's tube for cow's milk; B, Lewi's modification for woman's milk.

For an accurate determination the best ready method is the Babcock test, which requires 20 c.c. of milk, or the modification by Lewi[1] of the Leffman and Beam test for cow's milk. This requires special tubes.

Sugar.—The proportion of sugar varies so little that it may be ignored in clinical examinations.

Protein.—Clinical methods for the estimation of the protein are not very satisfactory.[2] We may also form some idea of the protein from a knowledge of the spe-

[1] Lewi's method is as follows:

(1) Place in the milk flask 2.92 c.c. of woman's milk measured in a special graduated pipette; (2) carefully rinse the pipette and add the same quantity of sulphuric acid C. P. of specific gravity 1.830. The acid should be added slowly, and mixed with the milk by gently rotating the flask. The color turns to a very dark brown from the oxidation of the sugar and protein; (3) now add 0.6 c.c. of a mixture of equal parts of fusel oil and strong hydrochloric acid; (4) add sufficient of a mixture of the same sulphuric acid and water, equal parts, to bring the level of the fluid well up into the neck of the flask; (5) centrifuge for three or four minutes. The percentage of fat is now read off, each one-tenth gradation in the neck of the flask representing 0.3 per cent of fat in the specimen of milk.

[2] The one giving the best results is that in which the protein is precipitated by a solution of phosphotungstic and hydrochloric acids in the Esbach tube, the

cific gravity and the percentage of fat, if we regard the sugar and salts as constant, or so nearly so as not to affect the specific gravity. We may thus determine whether it is greatly in excess or very low, which, after all, is the important thing. The specific gravity will then vary directly with the proportion of protein, and inversely with the proportion of fat, i. e., high protein, high specific gravity; high fat, low specific gravity. The application of this principle will be seen by reference to the accompanying table.

Woman's Milk

	Specific gravity—70° F.	Cream—24 hours.	Protein (estimated).
Average	1.031	7%	1.25%
Normal variations	1.028–1.029	8%–12%	Normal (rich milk)
Normal variations	1.032	5%–6%	Normal (fair milk)
Abn'l variations	Low (below 1.028)	High (above 10%)	Normal or slightly below
Abn'l variations	Low (below 1.028)	Low (below 5%)	Very low (very poor milk)
Abn'l variations	High (above 1.032)	High	Very high (very rich milk)
Abn'l variations	High (above 1.032)	Low	Normal (or nearly so)

A specimen taken for examination should be either the middle portion of the milk—i. e., after nursing two or three minutes—or, better, the entire quantity from one breast. The first milk is slightly richer in protein and much poorer in fat. The last drawn from the breasts is lower in protein and much higher in fat. The following analyses from Forster illustrate these differences:

	First Portion.	Second Portion.	Third Portion.
	Per cent	Per cent	Per cent
Fat	1.71	2.77	5.51
Protein	1.13	0.94	0.71

Conditions Affecting the Composition of Woman's Milk.—*The Age of the Nurse.*—This has no constant influence. Other things being equal, the milk of very young women, and also of those over thirty-five years of age, is likely to be lower in fat than that of women between twenty and thirty-five years.

Number of Pregnancies.—Adriance found that the average milk of 23 primiparae and 23 multiparae, both taken at the third month, showed the following differences: The milk of the primiparae averaged higher in fat and in protein, but a little lower in sugar.

Acute Illness.—In the majority of cases of acute illness of a minor character and of short duration there is no perceptible effect upon the

percentages being read off after standing twenty-four hours. For description see Boggs, Johns Hopkins' Hospital Bulletin, No. 187, October, 1906.

milk. In the acute febrile diseases of a severe type the quantity of milk is reduced, the fat is low, and the protein is apt to be high. In septic conditions bacteria may appear in the milk.

Menstruation.—The effect of this is exceedingly variable, depending much upon the individual and the case of menstruation. The most frequent changes noted are diminution in the quantity and a lower fat with the protein sometimes increased.

From observations upon 685 cases, Meyer noted disturbances in the child in over one-half the number. Our own experience accords rather with that of Pfeiffer and Schlichter, who consider it quite exceptional for the child to be visibly affected. Schlichter made observations upon infants during 233 menstrual days, noting the condition of the stools and digestion both before and after menstruation. In ninety per cent of the cases there was no perceptible influence. In only eight per cent were the stools bad, and in only three per cent was there disturbance of the stomach with vomiting. It is safe to say that the changes in milk accompanying menstruation are not uniform, and that in very many cases none of importance are produced.

Diet.—The composition of the milk is not so much influenced by diet as was formerly believed to be the case. The milk of an under-nourished woman is likely to be poor both in fat and protein. Sufficient food causes an increase in these substances. It is doubtful if the amount of fat can be further influenced by feeding either fat or carbohydrate, though by some authors it has been claimed that this could be done. Diet has a similar influence upon the quantity of protein. The amount of sugar is nearly constant under almost all circumstances. It is probably true that when the quantity of protein and fat are high they may be somewhat reduced by exercise and taking less food, but not to a marked extent unless under-nourishment had been present. All fluids tend to increase the quantity of milk.

The nursing woman should have a generous diet of simple food, and should drink largely of milk or gruels made with milk. The diet should be a varied one, not excessive in nitrogenous food nor in vegetables. Salads and highly seasoned dishes should be avoided, not so much because they upset the child, although this may happen, as because they are likely to disturb the digestion of the nurse. Nearly all the common vegetables and sweet fruits in season may be allowed in moderation. Strong tea and coffee should be prohibited, although weak tea or coffee may be allowed, each but once a day. Cocoa is not objectionable. In addition to her regular meals the nurse should have milk or gruel at bedtime. The diet should in all cases be adapted to her digestion. The bowels should move daily, by the use of laxatives if necessary. Great harm often results from overfeeding with its consequent indigestion.

Alcoholic Beverages.—With many women the use of malted liquors—ale, beer, etc.—increases the quantity of milk and the proportion of fat; but with many others their only effect is to fatten the nurse, often to a striking degree. The effect of spirits upon the quantity and composition of the milk is less marked, but along similar lines. Unless taken in large amounts by the mother, alcohol does not appear in her milk, and there is no sufficient evidence that in usual amounts it has any deleterious effect upon the milk; but the general use by nursing women of alcoholic beverages in any form is to be condemned. There are in most cases much better means of improving the milk than by ale, beer or whisky; they should be recommended by the physician to nursing women with the greatest discrimination. The danger of the formation of the alcoholic habit is too great to be passed over lightly.

Drugs.—The elimination of drugs through the milk is somewhat uncertain and variable; few of those popularly supposed to affect the child through the milk really do so. Given in full doses, belladonna regularly appears in the milk. Opium does not do so constantly; but when the milk is poor, enough may be excreted to produce serious symptoms. The iodids and bromids when long administered may be eliminated in sufficient quantity to produce their constitutional effects in the child. Mercury does not appear regularly, but only after prolonged use, and then in variable quantity. Most of the saline cathartics, arsenic, and the salicylates are occasionally found in the milk sometimes in quantities sufficient to produce symptoms in the nursing child.

Pregnancy.—The milk of a nursing woman who has become pregnant is generally scanty and poor in quality, especially in fat. The milk of a woman suffering from the toxemia of pregnancy is toxic to her infant. Fatal consequences have not infrequently followed putting an infant to the breast shortly after eclamptic attacks in the mother.

Bacteria.—Under normal conditions woman's milk may contain a few bacteria. They are chiefly cocci derived from the external milk ducts and are of no importance. In suppurative inflammation of the mammary gland, numerous bacteria may be found in the milk; also in some cases of puerperal sepsis. Tubercle bacilli have been demonstrated by Roger and Garnier in the milk of a woman with advanced tuberculosis, but ordinarily they are not present unless the gland is the seat of the disease.

The Elimination of Antitoxin and Other Protective Substances by the Milk.—The immunity of nursing infants to most of the contagious diseases has long been noted, but until recently little understood. Animal experiments have demonstrated the constant presence of diphtheria antitoxin in the milk of immunized animals. The Widal reaction has been

obtained with the milk of mothers suffering from typhoid and with the blood of their healthy nursing infants.

Nervous Impressions.—The effect of the nervous condition of a woman upon her milk secretion is very striking, and much more important than the diet. Both the quantity and the composition of the milk are markedly changed by many different nervous impressions. Fright, grief, passion, excessive sexual indulgence, or any great excitement may entirely arrest the secretion, or if not arrested the milk may be so altered in composition as to make the child acutely ill. Worry, anxiety, fatigue, intense or prolonged nervous strain may so alter the milk as to cause it to disagree with a child who had previously thrived well upon it, or they may greatly diminish and sometimes even arrest the secretion. It is the nervous condition of the mother more than anything else which determines her success or failure as a nurse. If a mother would nurse successfully, she must have plenty of rest and sleep, moderate exercise, keep her mind free from unnecessary worries, avoid social engagements, and lead a simple, regular, natural life. Unless she can and will do this successful nursing can hardly be expected.

The nature of the changes produced in milk by nervous disturbances in the mother are as yet little understood. Some infants are so profoundly affected as to suggest the development of toxic substances in the milk. The milk of the tired and worried mother is nearly always low in fat, while the protein is usually high, and possibly there are other changes as yet unknown.

COW'S MILK

Cow's milk being our main reliance in the artificial feeding of infants and the staple food of nearly all young children, it follows that everything relating to its production and handling is important. For fuller information than it is possible to give here the reader is referred to special works upon the subject.[1]

The essential conditions to be fulfilled in cow's milk which is to be used as a food for infants and young children are: (1) Freshness; (2) it should contain no preservatives; (3) it should be from healthy animals, free from tuberculous or other taint; (4) it should be clean; (5) it should not be skimmed or otherwise falsified; (6) it should con-

[1] Convenient works for a physician's use are Richmond's Dairy Chemistry; Aikman's Milk, Its Nature and Composition, Block, London; Russell's Outlines of Dairy Bacteriology; Belcher's Clean Milk, Hardy Publishing Co., New York; Pearsons' Jensen's Milk Hygiene; Milk and Its Relation to Public Health, Bulletin 56, U. S. Public Health and Marine-Hospital Service; The Milk Question, M. J. Rosenau.

tain no pathogenic organisms: (2) the number of other organisms should not be excessive. It is also desirable for purposes of infant feeding that the composition of the milk, particularly the percentage of fat, should be known, and that the milk should be as nearly uniform as possible from day to day and at different seasons of the year. Mixed or herd milk is therefore to be preferred to that from a single animal, since it is subject to fewer variations. The common varieties or "grade cows" should be chosen rather than highly bred animals, if for no other reason, because they are more hardy, less subject to disease, and less susceptible to other influences which might affect the milk.

As ordinarily handled, milk should if possible be used before it is twenty-four hours old; after this time changes occur very rapidly, and such milk can not in summer be used with safety for infants. Milk may be safe for a longer time provided special precautions are taken in producing and handling it, and special care in keeping it constantly at a temperature below 50° F.

Preservatives are sometimes added, particularly in hot weather, by unscrupulous dealers to retard the souring of milk, in order thereby to avoid the necessity and expense of proper icing. Formerly boric or salicylic acid was, and recently formaldehyd has been largely employed for this purpose.

Microörganisms in Milk.—Most of the common bacteria grow readily in milk, and the conditions under which it is produced and handled render it liable to contamination in many ways.

1. *Disease in the Cow.*—From disease of the udder streptococci or other pyogenic germs may enter the milk in such numbers as to excite acute gastro-enteritis in a child, but the particular danger under such circumstances is "septic sore throat." Within the last few years several severe epidemics of this dangerous disease have been reported. A number of these have been traced to herds that have included one or more animals with septic infection of the udder. Other diseases which may be communicated from the cow are tuberculosis, anthrax, and the foot-and-mouth disease. Veterinarians differ much in their estimates of the amount of tuberculosis among cattle, the estimates range from 3 to 25 per cent. It is the general opinion that it is on the increase, though this may only mean that the disease is now more often recognized. Of the cattle slaughtered in London, 25 per cent are stated to be tuberculous. Unless the process is advanced or the udder is the seat of disease, tubercle bacilli are usually absent from the milk. Nevertheless tubercle bacilli are frequently found in small numbers in ordinary market milk. In 107 unselected specimens of milk sold from cans in New York City, Hess found tubercle bacilli in 17, or 16 per cent. Rabinowitch and Kempner in 25 similar examinations in Berlin found tubercle bacilli in 7, or 28 per

cent. Macfadyen in London found, in 77 samples of milk, tubercle bacilli present in 17, or 22 per cent. These figures may probably be taken to represent average conditions in large cities. But the dangers from milk are not quite so great as would appear from these findings, for in many of the cases the number of bacilli is very small and only discovered by animal inoculations.

For reasons given elsewhere (*vide* Tuberculosis), we can not believe the danger of acquiring tuberculosis through milk as great as many have represented; yet milk must be regarded as one of the sources of tuberculous infection. The sale of milk from cows showing evidence of tuberculosis upon physical examination, and from those having tuberculosis of the udder should not be permitted; also the milk of every cow which reacts to the tuberculin test unless pasteurized.

2. *Specific Pathogenic Organisms Accidentally Gaining Access to Milk.*—The rôle of milk in the spread of infectious disease may be appreciated by the fact that in 1900 Kober was able to find records of 330 outbreaks which were traced to it. The disease most frequently communicated in this way is typhoid fever. In the reports of 195 epidemics collected, typhoid existed at the dairy in 148 instances; in 24 cases the employees acted as nurses to typhoid patients, and in 10 they continued at work, although themselves suffering from the disease.

Next to typhoid the disease most often spread through milk is scarlet fever. The sudden and simultaneous development of a considerable number of cases in a community should lead one to consider the milk supply as a possible cause. Of 99 epidemics of scarlet fever, there was disease at the farm or dairy in 68; in 17, employees were themselves affected, and in 10 they acted as nurses.

During 1911 and 1912 extensive epidemics of septic sore throat occurred in Boston, Chicago and Baltimore which were traced to streptococci spread through milk.

Besides the diseases mentioned, diphtheria, cholera, dysentery, and certain forms of diarrheal diseases may be spread by milk.

3. *Other Bacteria Found in Milk.*—These are chiefly derived from the dust of the stable, the hands and clothing of the milker, and from the dirt which falls from the udder and belly of the cow into the pail during milking; very many come from the cow's excreta. Freeman exposed a Petri gelatin-plate beneath a cow's udder for one minute during milking and obtained 4,450 colonies. The varieties of bacteria found in fresh milk are many and vary with locality. Toward the souring point the great majority are of two or three varieties only; fully 95 per cent at that time belong to the lactic-acid-producing group. They cause the ordinary souring of milk by acting upon the milk sugar. Colon bacilli are very common. Other bacteria act upon the milk protein,

inducing various putrefactive changes; and still others have a peptonizing power.

Many of the bacteria are harmless. Others, while not strictly pathogenic, yet when present in large numbers induce changes in milk that in susceptible infants may cause serious illness. The relation of bacterial contamination of milk to infantile diarrheas is considered in the introductory chapter upon Diarrheal Diseases.

The Number of Bacteria in Milk.—This depends upon three conditions: (1) Cleanliness in handling; (2) temperature; (3) age of the milk. Hence the bacterial count becomes of value in furnishing information as to these matters, although of less importance in regard to the production of disease than the nature of the organisms present. The influence of temperature alone upon the multiplication of bacteria in milk is well shown by the following experiment: Four samples of the same milk were kept at different temperatures for twenty-four hours and equal quantities were then plated; No. I was kept at 60° F. and showed 134,340 colonies; No. II was kept at 55° F. and showed 67,170; No. III was kept at 50° F. and showed 1,362; No. IV was kept at 45° F. and showed 448.

The number of bacteria in bottled milk from good single dairies usually ranges from 10,000 to 50,000 per c.cm., according to the season. Milk from mixed dairies delivered in cans usually ranges from 100,000 to 1,000,000, though much higher figures are often reached in very hot weather.

The number of bacteria in cream is nearly always greater than in milk. Freeman's experiments with gravity cream showed that the bacteria were 300 times as numerous in the cream as in the milk left behind, the bacteria being apparently carried up with the fat globules. This emphasizes the necessity of the greatest care with reference to the use of cream.

A Bacteriological Standard for Pure Milk.—It is impossible and undesirable to fix a numerical bacteriological standard for pure milk. One milk commission requires that the milk shall not have more than 10,000 bacteria in each cubic centimeter; another fixes the limit at 30,000. A milk commission should insist that milk be produced from healthy animals and under the most hygienic conditions, that it be handled only by healthy persons, and that every possible precaution be taken to exclude pathogenic germs. It is possible to lay too much stress upon the mere number of bacteria. There is no evidence that when the conditions mentioned have been fulfilled the results in infant feeding are better with a milk containing 5,000 bacteria or less, than with one containing 20,000. Nor is there any proof that milk containing 20,000 or 30,000 bacteria per c.cm. is for this reason alone injurious. A low bacterial count may

be taken as presumptive evidence that the milk is produced under hygienic conditions and carefully handled, and in such circumstances the entrance of pathogenic germs is improbable. Such a milk is therefore preferable. While it is quite possible to produce milk which is practically sterile, the expense entailed is so great as to make the commercial production of such milk impracticable.

The Means of Excluding Pathogenic Bacteria, and of Checking the Spread of Contagious Diseases through Milk.—Rules are readily deducible from a study of the records of how milk has usually been infected.

1. No person suffering from, or in contact with, a contagious disease should enter a dairy building or come in contact with the milk or milk-utensils. Children, domestic animals, and, so far as possible, flies should be excluded from rooms where milk is handled.

2. Milk should not be handled in or near dwellings, privies, or stables; cans and pails should be washed only at the dairy, and after ordinary cleansing they should be boiled or sterilized with live steam.

3. Dairies should be subject to regular city or state inspection. Milk from cows showing physical evidence of tuberculosis should be excluded; also that from animals which are in any way sick or are suffering from disease of the udder. Milk from apparently healthy animals which respond to the tuberculin test should not be used for food in a raw state.

4. During epidemics of scarlet or typhoid fever or septic sore throat no raw milk should be used; and all cases of such diseases occurring in the families of those who produce or handle the milk should be immediately reported and isolated by the authorities. Most of the rules mentioned are enforced by milk commissions which supervise the production of "certified milk."

Composition of Cow's Milk.—Except in the percentage of fat, the composition of mixed or herd milk does not vary greatly with the different breeds. The fat is lowest in the Holsteins, and highest in the Jerseys.

Composition of Cow's Milk [1]

	Jerseys	Holsteins	Average Good Herd Milk.
Fat	5.61	3.46	4.00
Sugar	5.15	4.84	4.75
Protein	3.91	3.39	3.50
Ash	0.74	0.74	0.75
Total Solids	15.41	12.43	13.00
Water	84.59	87.57	87.00
Total	100.00	100.00	100.00

[1] In the table the figures for Jersey and Holstein herds are the averages given by the New York State Experiment Station. The requirements in New York and most of the States are, fat, at least 3 per cent; total solids, 12 per cent.

In a poor milk the only important difference to be considered is that the fat is from 0.5 to 1 per cent lower than the averages given. In Jersey milk the chief difference is that the fat is 1 to 1.5 per cent higher than the averages; there is also an increase, though a less important one, in the other solids. As to the relative advantages of the different breeds for infant feeding, the difference does not seem great, provided all are equally healthy. Jerseys and all highly bred animals are more prone to disease and minor disturbances than the hardier common breeds.

The Examination of Cow's Milk.—The application of heat often causes coagulation in milk which is near the souring point, and also in colostrum milk. Both are unfit for use. The normal *reaction* of cow's milk is amphoteric or slightly acid; if alkaline, it is pretty certain that something has been added to it.

The *specific gravity* is from 1.028 to 1.033. If the cream has been removed, the specific gravity is raised; if water has been added, the specific gravity is lowered.

The best of all ready methods of determining the fat content is the Babcock test. The cream-gauge may be used as for woman's milk, the 100 c.c. size to be preferred; but it is not to be relied upon unless the milk is put into the cylinder soon after it is drawn and cooled rapidly by being placed in ice-water. Under these conditions, if the reading is made after eight to twelve hours, the percentage of cream to that of fat is about three to one.

A *microscopical examination* of the cream and the sediment may give valuable information. Not much can be learned from a study of the fat globules, but among them may be found colostrum corpuscles, which are usually present for nearly a week after calving. The sediment after centrifuging should be examined to ascertain the number and character of the cells present and should be stained for bacteria. The character of the cells can best be determined by the use of a differential blood stain. A few leucocytes are almost invariably found in normal milk; epithelial cells and lymphocytes may be present in quite large numbers without impairing the quality of the milk. However, eosinophile cells and polymorphonuclear neutrophiles, when in large numbers or in clumps, invariably indicate disease in the cow and the milk should be rejected. Such milk is often ropy in consistency owing to the presence of mucus and usually contains many long chains of streptococci. When used for infants it may excite severe digestive disturbances, usually diarrhea. Red blood cells in milk may be due to traumatism, to inflammation or to the fact that milk is taken too soon after calving. Whenever polymorphonuclear leucocytes, blood or streptococci are at all numerous, the milk should not be used and a thorough inspection of

the herd should be made. The only sure way of demonstrating the presence of tubercle bacilli in milk is by animal inoculation.

The casein [1] of cow's milk is readily coagulated by rennet and by acids. The curd formed by the gastric juice is tough and firm and is more slowly dissolved by the action of the digestive fluids. The casein of woman's milk is not regularly coagulated by rennet, and only slightly and with difficulty by acids. The curd formed by the gastric juice is loose and flocculent, and is readily and completely dissolved.

The inorganic salts in cow's milk are about three and a half times as abundant as in woman's milk; but as will be seen in the following table, the proportion in which the principal salts are present is very nearly the same, the only notable exception being in the phosphoric acid.

	Cow's [2]	Woman's [3]
CaO	22.8%	23.3%
MgO	2.8%	3.7%
P_2O_5	27.4%	16.6%
K_2O	24.7%	28.3%
Na_2O	10.9%	7.2%
Cl	15.5%	16.5%

The large amount in cow's milk is mainly derived from the casein. Even when diluted twice the chief salts of cow's milk are still in excess of those in woman's milk. In all dilutions of cow's milk the total salts may be calculated as one-fifth the protein. The ratio thus is about the same as salts to protein in woman's milk. The larger amount of total salts in cow's milk apparently has no injurious effect upon the digestion of healthy infants; most of the excess is not absorbed or retained. Thus while an infant fed on woman's milk absorbs about 80 to 85 per cent of the salts in the milk and retains from 40 to 50 per cent, one fed on cow's milk absorbs only about 60 per cent and retains about 15 per cent.

The ash of milk, however, does not accurately represent its mineral constituents. About 8 per cent of the phosphoric acid of the ash, according to Richmond, is derived from the casein; sulphuric acid, though traces are found in milk, is not to be regarded as one of its true mineral

[1] By Haliburton and some other chemists the term *caseinogen* is given to this protein as it exists in milk. When this is acted upon by rennet it splits up into two substances: One, the firm, insoluble coagulum to which only the term *casein* is applied; the other, a soluble protein which is known as *whey-protein;* this is present in but small amount. Those who use the term casein to designate the protein as it exists in milk, refer to the curd formed by the action of rennet in the stomach as *paracasein.*

[2] Average of four standard authorities.

[3] Average of sixteen analyses of mature milk, Courtney and Fales.

constituents. The most recent analyses show the presence of citric acid in both woman's and cow's milk.

The amount of iron in milk is extremely small. In woman's milk it is about 1.5 mgm. per liter or .00015 per cent (Bahrdt and Edelstein). In cow's milk it is only about one-third this—really a negligible quantity.

Bacteria.—Cow's milk always contains a large number of bacteria which increase in proportion to the age of the milk; woman's milk is seldom quite sterile but contains a few cocci from the milk ducts. To what degree the bacterial content of cow's milk affects its digestibility by healthy infants, it is impossible to make positive statements. There seems abundant clinical evidence that excessive bacterial contamination of the varieties commonly present in milk are detrimental even to healthy infants, and that to the delicate, the feeble and the diseased their effects are most injurious. So far as the production of disease is concerned, it is of course the nature, not the number of bacteria which is important. The beneficial effects seen of sterilizing or boiling the milk fed to feeble children are only in part to be ascribed to the destruction of bacteria.

Cream.—A great misapprehension exists as to its composition. It is often spoken of as if it were something entirely different from milk. It should be regarded rather as milk which contains an excess of fat. Cream was formerly obtained by skimming—the gravity process; at present, almost entirely by the use of a centrifugal machine known as a separator. The latter process has the advantage in point of time, as centrifugal cream can be put upon the market from twenty-four to thirty-six hours earlier than gravity cream.

The following table gives the composition of an average milk and of centrifugal cream of different densities removed from the same milk:

	Whole Milk.	Cream.				
		I.	II.	III.	IV.	V.
Fat	4.00	8.00	12.00	16.00	20.00	40.00
Sugar	4.75	4.60	4.50	4.15	3.90	3.00
Protein	3.50	3.40	3.30	3.20	3.05	2.20
Salts	0.75	0.70	0.65	0.60	0.55	0.45

The percentages of protein and sugar in the 8 and 12-per-cent cream are but little lower than in milk; in the very rich creams they are reduced by about one-third.

It is unfortunate that no general standard exists as to what shall be sold as cream. In New York State the law provides that cream shall contain at least 18 per cent fat. Very rich, centrifugal cream has from 35 to 40 per cent fat; the usual centrifugal cream has about 18 to 20

per cent. Gravity cream has generally from 16 to 20 per cent fat.

None of the methods for determining the fat in milk is applicable to cream, except the Babcock test. Such variation exists in the strength of cream that the physician who is prescribing it for infants should have tests frequently made.

Methods of Obtaining Milk Containing Various Proportions of Fat —Top-Milk, Skimmed Milk.—To secure a milk for infant feeding which is fresh and at the same time one which contains a larger proportion of fat than does whole milk, one may remove only a certain number of ounces from the top of a quart bottle. If cow's milk is put into bottles soon after it is drawn and rapidly cooled, the top-milk may be removed after four hours. Milk bottled at dairies and then transported should be allowed to stand after it is received for at least two hours before removing the top-milk. This may be done with a siphon, spoon, or a small special dipper; pouring off is not accurate.

Skimmed milk, or milk which contains a smaller proportion of fat than does whole milk, may be obtained from bottled milk by removing a certain number of ounces from the top of the quart bottle and using only the remainder.

It is unnecessary in practice to have a top-milk which contains more than 7 per cent fat; while it is desirable at times to obtain milk which is practically fat-free. It is also desirable to know the percentage of fat that is obtained when one uses various quantities from the top or bottom of a quart of milk. These values are only approximate, but if the top-milk is carefully removed, are sufficiently accurate for practical purposes. These may be obtained from average herd milk or very rich milk as follows:

From one quart 4 per cent milk			From 5 per cent milk
Upper 16 oz.	has	7% fat;	upper 20 oz.
" 20 "	"	6% " ;	" 24 "
" 24 "	"	5% " ;	all
All	"	4% " ;	rest after removing top 2 oz.
Rest after removing top 2 oz.	has	3% " ;	" " " " 3 "
" " " " 4 "	"	2% " ;	" " " " 5 "
" " " " 8 "	"	1% " ;	" " " " 8 "

Fat-free milk can be obtained only by the removal of the cream by a separator.

In general it is wise for one who has much to do with infant feeding to have his patients take milk from the same supply to secure uniformity in his results.

In or near large cities it is possible to obtain from milk laboratories cream or milk with any desired percentage of fat.

Milk Sterilization.—The term *sterilization* is widely and rather loosely used to signify the heating of milk for the destruction of germs.

It should, however, be borne in mind that none of the methods commonly employed renders milk sterile in the bacteriological sense of the word. What is accomplished is the destruction of such pathogenic germs as may be present, and from 95 to 99 per cent of the other bacteria, so as to retard for a considerable time the ordinary fermentative changes.

The advantages of sterilizing milk are obvious. When we consider the enormous number of bacteria present in cow's milk with the usual methods of handling, and that they are frequently the cause of disease, it is not strange that after its introduction by Soxhlet in 1886 the practice of heating milk used for infant feeding rapidly extended over the world. Following him, the earlier experiments in sterilization were made at 212° F., usually continued for an hour and a half, and this temperature is still largely employed on the Continent of Europe. Even this does not render milk safe for very long. Spores are not destroyed, and at ordinary room temperatures spore-bearing bacteria may soon develop in such numbers as to make the milk dangerous. Since some of these bacteria act upon the milk-protein and not upon the sugar, such milk may not be sour, and hence its danger may not be recognized.

There are some disadvantages in heating milk. The change in taste and the constipating effects of sterilized milk are very noticeable. Some of the lactose is converted into caramel, causing a slight change in color; the lactalbumin is partially coagulated, this beginning at 160° F. (70° C.), and the casein is rendered less coagulable by rennet; Rettger has shown that when milk is heated above 185° F. (85° C.) a volatile sulphid is liberated, conclusive evidence of some change in the protein; the organic phosphorus is changed into an inorganic phosphate; the citric acid is partially precipitated as calcium citrate, and some lime salts, which are usually soluble, are converted into insoluble compounds. Some changes also occur in the fat. Moreover, certain natural ferments in fresh milk are destroyed by heat.

Many of these changes are doubtless without any injurious effect upon nutrition. There is, however, one important clinical reason for believing that the nutritive properties of milk may be impaired by heating to 212° F.—viz., the occurrence of scurvy in infants who are fed solely upon such milk for a long time. Of 379 cases of infantile scurvy brought together in the Report of the American Pediatric Society, sterilized milk was the previous diet in 107. Many such cases have come under our own notice. Again and again cases of scurvy have been cured by simply changing from sterilized to raw milk.

Heating at Lower Temperatures—Pasteurizing Milk.—To obviate the disadvantages above referred to, the practice has come largely into use in America of employing much lower temperatures for milk sterilization.

At present 150° to 155° F. (65° to 68° C.) are the temperatures generally employed. These temperatures are maintained from twenty to thirty minutes. This is sufficient to kill the bacilli of tuberculosis, diphtheria, and typhoid fever, and from 98 to 99.8 per cent of all other bacteria in milk. Nearly all of the objectionable changes produced in sterilized milk are avoided when the temperature is raised only to 150° F. (65° C.), while it accomplishes the purpose for which milk is heated. The advantages of this form of heating are therefore obvious. But spores are not destroyed, and such milk requires special handling. It should be rapidly cooled, kept at a low temperature, and used within twenty-four hours after heating.

Commercial Pasteurization of Milk.—This was first accomplished by passing milk through hot pipes in which it was exposed to a temperature of 140° to 160° F. for a brief period, usually less than one minute. This has been found practically to be insufficient to destroy the pathogenic organisms in milk. At present the method followed is known as the "holding process." By this the milk is slowly passed through a succession of vats being held at a temperature of about 150° F. for thirty or forty minutes. It is afterwards cooled, then drawn into sterilized containers, bottled and labeled "pasteurized" milk. For this process expensive and complicated apparatus is necessary and even when done on a large scale it adds to the cost of the milk. The limited control which it is possible for a municipality to exercise over milk producers and distributors, the impossibility of securing adequate inspection of dairy-farms and creameries, a conviction that a large part of the typhoid seen in cities and towns is milk-borne, and the occurrence of extensive epidemics of septic sore throat from milk infection, have forced upon many boards of health the necessity of compelling pasteurization of all milk used for food in an uncooked state unless the same is from "certified dairies" supervised by competent milk commissions. This practice bids fair to become general. The objections to and disadvantages of pasteurized milk should also be considered. There is a temptation to an unscrupulous producer to neglect precautions necessary to keep milk clean, and at the same time to a dealer to deliver milk that otherwise would be unsalable because near the souring point. The necessity of keeping pasteurized milk cold and of using it within twenty-four hours if possible, must be taught the public. The label "pasteurized milk" too often conveys a false sense of security and leads to the neglect of the precautions mentioned. The date of pasteurization should be stamped upon the label. It should be known that, unless milk is kept cold and used soon it may, even though pasteurized, contain an immense number of bacteria although it does not turn sour. The closest kind of supervision should be exercised by authorities which

permit or require the general pasteurization of the milk of a community.

Pasteurization vs. Sterilization.—From what has already been said it would appear that the argument is altogether in favor of pasteurization. The lowest temperature and the shortest time that will surely destroy the objectionable bacteria in milk would seem to merit general adoption. Pasteurization, however, requires considerable care, intelligence, and special apparatus. When all these can be secured it should be employed as the method of choice.

Sterilization at 212° F. (100° C.) is much simpler; it can be done with many simple and inexpensive forms of apparatus or even without any special apparatus. Where no ice is available, it is safer in hot weather than pasteurization. Among the poor of our large cities, in summer, boiling is to be advised as the most satisfactory, and indeed the only efficient, method of sterilization. It should not be forgotten that the use of such milk as the sole diet for a long time is attended with a certain amount of risk, even though a small one; and one should always be on the watch for the soreness of the legs and the spongy gums that indicate the beginning of scurvy. Heating to 212° F. on two successive days is also to be recommended where milk must be kept for one or two weeks, as upon ocean journeys.

Methods of Heating Milk.—Milk for infant feeding should be sterilized at home preferably in small bottles, each one of which contains a sufficient quantity for one feeding. These bottles may be plugged with cotton or corks, or special stoppers may be used. Soxhlet's apparatus may be employed, or Arnold's, or any one of a half dozen others sold in the shops. All that is really necessary is to expose the bottles on all sides to live steam in a closed vessel. It can be done effectively in any tin vessel which has a closely fitting cover and a perforated bottom, and which can be placed over a pot of boiling water. Sterilization at 212° F. is usually continued for one hour. The bottles should then be cooled in water as quickly as possible and placed upon ice or in the coolest place available.

A simple apparatus for pasteurizing milk has been devised by Freeman in which the temperature is raised to 150° F. (65° C.) by hot water.[1] An essential step in pasteurizing milk is rapid cooling. After

[1] Milk may be effectively, but not very accurately, pasteurized in the home without any apparatus in the following manner: Place the bottles of milk in a covered pot containing enough tepid water to cover the bottles to the neck. Allow this to stand on the top of a stove until the water begins to simmer. Now remove the pot from the stove to a table and let it stand for twenty minutes covered. Then cool the bottles of milk by placing them first in water at room temperature and afterwards in ice water.

thirty minutes the bottles should be removed from the pasteurizer, placed in water at the room temperature and afterward in ice-water, where they should remain half an hour before being placed in the cold room or ice chest.

The sterilization of milk is useful, first, for the destruction of pathogenic germs; secondly, for the destruction of the bacteria causing fermentation, thus enabling one to feed with safety milk in which, though it may be forty-eight hours old, no important fermentative changes have occurred. As a therapeutic measure sterilized milk is useful in various forms of gastric or intestinal infection such as typhoid fever, dysentery, diarrhea, etc. It is a matter of clinical observation that sterilized milk is sometimes well borne when raw milk is not, particularly by very young infants.

Shall All Milk Used for Infant Feeding Be Sterilized?—In warm weather only the very cleanest milk can safely be used without heating. In winter, the heating of milk is not so necessary; but so long as milk is produced and handled as the bulk of milk is at present, not being delivered in large cities until it is considerably over twenty-four hours old, and not consumed until over forty-eight hours old, some form of heating should invariably be practiced, unless it is known to be produced and handled under the best conditions. In the country where milk is obtained fresh and used before it is twenty-four hours old, sterilizing is unnecessary if the cows are healthy and the milk properly handled.

It is quite possible to produce milk which does not need sterilization. There are special dairies supplying such certified milk to many of our large cities, and their number may be very greatly increased if the medical profession will use its influence in this direction. Our preference for infant feeding is a milk so clean and fresh that it may be safely given even in summer without heating, but this is at present available only for the small minority. Healthy infants with good digestion usually do well upon raw milk even though the number of bacteria is quite large; while delicate infants or those with digestive disturbances may be seriously affected by such milk.

The feeding of boiled or sterilized milk must be considered quite apart from the question of microörganisms. There are unquestionably some very young and some delicate infants with feeble digestion who thrive better upon heated than raw milk even though the number of bacteria in the latter may be very small. Experiments in feeding raw and boiled milk to young animals for comparative results have not been numerous, nor quite conclusive. Those which have been published in Europe are rather in favor of boiled milk. The latest experiments of Daniels and Stuessy in this country, however, indicate that animals do

not thrive for a long period upon boiled milk; although for the first two or three months those fed on boiled milk did distinctly better than those fed upon raw milk. Brennemann (Chicago) has made some experiments upon the human subject (a young adult) which show conclusively that boiling has a marked effect upon the coagulation of the casein in the stomach. Under certain conditions this seems to produce a distinctly beneficial effect upon digestion. However, this is far from proving that all milk used for infant feeding shall be so heated. The use of boiled milk for long periods is not to be recommended; although with young infants and for short periods it is frequently of the greatest service.

Frozen Milk.—During very cold weather milk is often unavoidably delivered in a partially or completely frozen condition and the question frequently is raised whether any important change is produced by freezing which affects its digestibility by young infants. So far as is known the changes brought about are purely physical ones. Only the water of the milk freezes, the fat undergoing separation in consequence. When such milk is warmed again, the fat globules may coalesce to form an oily layer of butter fat. While older children or robust infants are seldom affected by such milk, considerable disturbance may be produced in delicate or susceptible infants. Occasionally vomiting is excited, but more often there is diarrhea which may become severe. The higher the fat percentage in the milk fed, the more severe are the symptoms likely to be.

Peptonized Milk.—Milk is peptonized through the agency of a substance derived from the pancreas, usually that of the pig. This is known in the market as "extractum pancreatis," the active ferment being the trypsin. As this acts only in an alkaline medium, bicarbonate of soda should first be added to the milk. The purpose of peptonizing is to secure a partial digestion of the protein of milk before feeding.

Milk which has been peptonized ten minutes is not altered in taste; if, however, the process is continued for twenty minutes, a slightly bitter taste is noticed which increases with the duration of the process. Peptonizing may be arrested at any stage by raising the milk to the boiling point; but if the milk is to be fed at once this is not necessary.

Peptonized milk is useful only when the stomach is so sensitive as to be affected by the coagulation of milk, something which is rarely seen. The prolonged use of peptonized milk as the sole food is sometimes followed by scurvy.

Condensed Milk.—This is prepared by heating fresh cow's milk to 212° F. for twenty minutes for sterilization, and then evaporating *in vacuo,* so that one part of condensed milk represents about two and a half parts of the original milk. Sweetened condensed milk is preserved

in tin cans, with the addition of about seven ounces of cane sugar to a pint.

The composition of sweetened condensed milk is shown in the following table; also the results obtained when it is diluted:

	Condensed Milk.[1]	With 6 Parts of Water Added.	With 12 Parts of Water.	With 18 Parts of Water.
	Per cent.	Per cent.	Per cent.	Per cent.
Fat	9.61	1.37	0.73	0.50
Protein	8.01	1.14	0.61	0.42
Sugar { Cane, 42.91 / Milk, 12.03 }	54.94	7.89	4.75	2.90
Salts	1.78	0.25	0.13	0.09
Water	25.66	89.35	94.28	96.09

[1] Analysis of Borden's Eagle-Brand condensed milk.

The reasons both for the success and for the failure of sweetened condensed milk as an infant-food are apparent from a study of its composition. As a temporary food it is often useful, first because it is nearly sterile, but chiefly because the fat of cow's milk has been reduced by the usual dilution to a point at which an infant with a very weak digestion can bear it, while it furnishes an abundance of sugar; but it is low in protein. Infants fed upon condensed milk are often fat, but have, as a rule, feeble resistance when attacked by acute disease, especially of the intestinal tract. It is rare to see a child reared on condensed milk who does not show some evidence of rickets. The prolonged use of condensed milk is sometimes a cause of scurvy. Condensed milk is admissible for temporary use during attacks of indigestion, for infants with feeble digestion, especially in summer, for very young infants during the first two or three months, or among the very poor, when the cow's milk which is available is still more objectionable. It should not be used as a permanent food when good, fresh cow's milk can be obtained. In travelling it is often the most convenient as well as the safest food to use. It should usually be diluted ten to twelve times for an infant under one month, and from six to ten times for those who are older.

During recent years condensed milk without any addition of sugar is sold in the market; in many large cities this is delivered fresh daily in bulk; it is also sold in tin cans in a sterilized form. To distinguish it from other condensed milk it is called "evaporated milk." Its strength is about the same as that of the better known condensed milk, i. e., one part representing about two and a half parts of the original milk, without any addition of sugar or other preservative. Evaporated milk re-

quires the same modification as ordinary cow's milk. For routine use it should be diluted with from eight to twelve parts of water, and sugar added. When diluted with water the proportion of fat and protein will be approximately the same as in condensed milk given in the foregoing table. Additional carbohydrates may be introduced in whatever form may seem desirable, either as sugar (milk sugar, cane sugar, or maltose) or as starch (barley, oat or wheat flour). It is a sterile, cooked milk. Some children thrive upon it who cannot so well digest either raw milk of the same percentage composition or even freshly pasteurized milk. It should not be long continued as the sole food when good fresh milk can be obtained.

Dried Milk.—Dried milk sold under various names has more recently been put upon the market. It is prepared either from whole milk or from skimmed milk. The process of manufacture most extensively employed is that of spraying the milk upon hot revolving cylinders by which means the water is driven off almost instantaneously. A preparation of dried milk made from partially skimmed milk to which milk sugar has been added is sold under the name of "mammala." It contains 12 per cent of fat; 54 per cent of sugar; 24 per cent of protein, and 5 per cent of salts. A somewhat similar preparation but higher in fat is sold in England under the name of "glaxo." Dried milk is a sterile, white powder and in sealed cans keeps indefinitely. When eight parts by volume of water are added (one level teaspoonful to the ounce) it approximates in composition the original milk. It may be further modified if desired. Its application is similar to that of condensed milk over which it presents some obvious advantages in travelling; it is open to the same objections as a permanent food, and should not be advised when fresh milk can be obtained.

Buttermilk and Other Forms of Fermented Milk.—Various forms of fermented milk are in use which differ according to the milk used and the process followed. They resemble each other in that the fermentation is excited by some of the varieties of lactic acid organisms, in some cases with the addition of yeast, which ferment a portion of the milk sugar. The ordinary buttermilk of commerce is sometimes made from sweet, but usually from sour cream. If from the latter, it resembles the fermented milks in that it contains little or no fat but a certain amount of lactic acid, the result of fermentation. It differs from them in that the fermentation in buttermilk is due to a great variety of lactic acid organisms; besides, it contains many other forms of bacteria than those concerned in the process of fermentation. Buttermilk should be made with care or it may be grossly contaminated. It, therefore, varies greatly in taste and considerably in composition under different conditions. The following is an average of many analyses.

Buttermilk

Fat	0.50 per cent
Milk sugar	4.00 " "
Lactic acid	0.80 " "
Protein	3.60 " "
Inorganic salts	0.75 " "
Water	90.35 " "
	100.00

When used as an infant food it is sometimes sterilized and sometimes not. The sugar content is raised by the addition of milk sugar or cane sugar; sometimes also barley flour or other farinaceous food is added A formula much used is: buttermilk, one quart; barley flour, two even tablespoonfuls; water, four ounces; cook slowly, constantly stirring, for twenty minutes; then add two teaspoonfuls of cane sugar. The advantages of buttermilk as an infant food are chiefly due to its low fat content and to the small amount of lactic acid which it contains. Its cheapness is an important consideration and makes it available for the very poor.

Other fermented milks, sometimes called buttermilk, are known also as *lactic acid milk, lactobacilline, lactobacillary milk, lactone buttermilk*, etc. They are sometimes made from whole milk but chiefly from skimmed milk. This is usually first sterilized and then the ferment added in the form of a tablet, mixture or culture from some previously fermented milk. The ferment consists of different varieties of lactic acid organisms; the one most frequently employed is known as the Bulgaricus. The product resembles ordinary buttermilk in its composition except that it usually has a higher acidity. It is a purer product since the fermentation takes place from one or two selected varieties of organisms and not from a great number as in ordinary buttermilk. It differs according to the exact varieties or combinations used, also according to the temperature maintained and the duration of the fermentation. A temperature of 80° to 85° F. is usually employed and this is continued from six to twelve hours according to the degree of acidity desired. The milk is then bottled and put on ice, where a slight change continues, although the milk alters but little for several days. The taste is rather pleasant unless the acidity is too pronounced. It should not contain alcohol or acetic acid. These fermented milks are sometimes used in acute disease, but chiefly in chronic intestinal conditions after the first year. They are not adapted to continuous use in infant feeding.

Kumyss has been made by the Tartars for centuries from mare's milk. It is made in this country from cow's milk, sometimes skimmed, but usually from the whole milk. The fermentation is generally started with yeast and is continued in corked bottles usually for several days, with

frequent agitation. Kumyss contains carbon dioxid, lactic acid, alcohol and traces of butyric and acetic acids. The acidity and the taste depend upon the duration of the process.

Zoolak or *matzoon* is made from whole milk which is first sterilized and then has added to it a ferment which contains some form of yeast. It differs from kumyss chiefly in that the process is carried on in open vessels and the carbon dioxid allowed to escape. It is a thick smooth liquid and has a taste resembling that of sour cream.

Both kumyss and zoolak are better adapted for use with older children than with infants; they are chiefly valuable in cases of chronic intestinal indigestion. For infants they should be diluted with water and often given with a spoon since they are too thick to go through the ordinary nipple.

Protein Milk (*Eiweiss-Milch* of Finkelstein).—In this milk modification is secured a mixture low in sugar with a moderate fat and a high protein. It must be carefully prepared to secure a uniform product. The average composition when made as directed below[1] is fat 3.0 to 3.5 per cent; sugar 1.8 per cent; protein 3.75 per cent; salts 0.65 per cent. Its caloric value is about 15 to the ounce. The fat percentage varies considerably according to that of the fat of the milk used and the care exercised in its preparation. When less fat is desired partially skimmed milk may be substituted. The proportion of the ingredients other than the fat is pretty uniform. The total salts are a little lower than in whole milk; the proportion of insoluble salts, especially calcium, is, however, greater, while that of the soluble salts of sodium and potassium is somewhat less. Protein milk has a slightly sour, rather insipid taste, so that its administration to some infants is difficult. It is made

[1] To one quart of whole milk warmed to about 100° F. one-half ounce of liquid rennet or better one junket tablet dissolved in water is added, stirring for a moment only; after standing at room temperature for twenty or thirty minutes, or until it is firmly coagulated, it is poured upon two layers of gauze or cheesecloth and suspended for about one hour to drain off the whey. The curd is then washed twice with cold boiled water, after which the dry curd is rubbed through a very fine sieve with a vegetable masher, or some similar instrument, with the gradual addition of one pint of buttermilk. Enough boiled water is added to make one quart. When needed in quantity for hospital use, from five to ten quarts may readily be prepared at one time. After coagulation it is poured upon the cheesecloth and allowed to drain undisturbed for ten to fifteen minutes. The curd is then rolled from side to side by manipulating the cheesecloth and the whey removed in a few minutes. It should then be washed. Protein milk, made as above described, will contain the greater part of the fat, casein and insoluble salts of the original milk, also the salts, sugar and protein of the buttermilk, which makes up half its volume. The sugar, the albumin, the soluble salts and a little of the fat are removed with the whey and the wash.

more palatable by the addition of one grain of saccharine to the quart.

The advantages of protein milk depend upon: (1) its low sugar; (2) its relatively high fat and insoluble salts whose soaps favor the production of formed stools and check intestinal fermentation; (3) the high protein (nearly all casein), which, having been precipitated and then mechanically subdivided, is well borne by the stomach; (4) probably to some degree the lactic acid organisms contained in the buttermilk. The high percentage of casein is readily held in suspension. When properly made protein milk is smooth and homogeneous and readily passes through an ordinary rubber nipple. It can be warmed to the usual temperature before feeding, but if heated much above this point the curd separates. Protein milk is to be regarded as a therapeutic agent, not as an infant food for prolonged use. It has a wide field of usefulness in both acute and chronic disturbances of digestion with intolerance of carbohydrates, particularly those associated with diarrhea.

Junket or Curds and Whey.—Junket is made as follows: To one pint of fresh lukewarm cow's milk are added two teaspoonfuls of essence of pepsin, liquid rennet, or half a junket tablet. It is stirred for a moment and then allowed to stand at the room temperature until firmly coagulated. Junket is useful in the feeding of older children, but should not be given to infants.

Whey.—The milk is coagulated with rennet as above, the curd is then broken up, and the whey strained through muslin by suspension. The composition of whey varies somewhat, depending upon the way in which it is prepared. If it is desired to have as little fat as possible, skimmed or fat-free milk should be used, and the whey should be strained through fine muslin without pressure. If it is desired to retain some of the fat, whole milk may be used, cheesecloth as a strainer, and more pressure. The protein of whey is chiefly lactalbumin.

Whey is useful for infants with gastric symptoms when low fat is desired. Its high sugar and salt content usually contraindicate its use in cases with intestinal symptoms, especially if diarrhea is present.

Whey

	Average 46 Analyses (Koenig).	From Whole Milk (Adriance).	From Fat-free Milk (Adriance).
Protein	0.86	0.94	1.17
Fat	0.32	0.96	0.04
Sugar	4.79	5.49	5.36
Salts	0.65	0.48	0.52
Water	93.38	92.13	92.91
Total	100.00	100.00	100.00

Wine whey is made by simply adding sherry wine to whey prepared in the usual manner, in the proportion of one part to four of whey, possibly better by using the wine to coagulate the milk (Still). The wine (cooking sherry preferred) is added to the milk in the proportion mentioned and the mixture slowly brought to the boiling point. After standing off the fire for three or four minutes it is strained through two layers of coarse muslin, or cheesecloth. Sherry whey is useful as an emergency food for short periods in acute illness for children who will take very little food; it is seldom given alone, but alternating with some other food.

BEEF PREPARATIONS

The nutrient value of these preparations is to be measured by the amount of albumin they contain—their stimulant properties by the proportion of extractives.

Beef Juice.—Expressed beef juice is made as follows: A piece of round steak is slightly broiled, and the juice pressed out by a meat-press or a lemon-squeezer. Two or three ounces can ordinarily be obtained from one pound of steak. This is seasoned with salt and given cold or warm, but not heated sufficiently to coagulate the albumin in solution.

An excellent method of making beef juice without cooking is by taking one pound of finely chopped lean beef and eight ounces of water and allowing this to stand in a covered jar upon ice from six to twelve hours. The meat is then squeezed by twisting in coarse muslin. It is seasoned with salt and given as above.

Beef extracts are not to be considered in any sense as foods. Kemmerich has shown that animals receiving nothing else died of starvation, and sooner even than when everything was withheld. They contain no nitrogen in the form of protein, but only in combination with the soluble extractives. They are stimulants, but as such are seldom required.

Meat.—Rare scraped beef is easily digested by most young children. There are many conditions in which other forms of protein are not well borne, where children even as young as twelve months appear to digest this beef-pulp without difficulty. It should be made from very rare or raw steak, finely scraped and well salted. A tablespoonful may be given at one feeding to a child of eighteen months. In nutrient properties this far exceeds the beef preparations in the market. The alleged danger of tapeworm from the use of rare scraped beef or beef juice is in this country so slight that it may be disregarded.

Broths.—Animal broths may be made from mutton, veal, chicken, or beef. A good formula for general use is the following: One pound of lean meat, one pint of water; let stand for two hours, then cook over a

slow fire for two hours down to half a pint. After it has cooled, skim off the fat and strain through a cloth. The composition of a broth so made is given by Cheadle as follows:

Beef Broth

Protein	1.02
Extractives	1.82
Fat	0.00
Salts	0.88
Water	96.28
	100.00

From their composition it will be seen that broths contain very little nutritive material. They are stimulating and they furnish an excellent means of adding inorganic salts to the diet in the latter part of the first year. Vegetables and barley, rice or wheat flour may be cooked with the broth.

Albumin Water.—This is prepared as follows: The white of one fresh egg is mixed with a pint of cold water, a little salt, and a teaspoonful of brandy added. It should be given cold. The nutritive value of this preparation, it should be borne in mind, is very small.

CEREALS

Barley Water.—This may be made either from the grains or from the barley flour. When the grains are used, the following is the formula which we have been accustomed to employ: To two tablespoonfuls of pearl barley, add one quart of water and a pinch of salt, and boil continuously for six hours, keeping the quantity up to a quart by the addition of water; strain through coarse muslin. It is an advantage to soak the barley for a few hours before cooking. The water in which it is soaked is not used. When cold this preparation makes a rather thin jelly. Its composition by analysis is as follows:

Barley Water

Starch	1.63
Fat	0.05
Protein	0.09
Inorganic Salts	0.03
Water	98.20
	100.00

An almost identical product may be obtained in an easier way by using barley flour, one even tablespoonful to each twelve ounces of water,

and cooking for twenty minutes. A thicker jelly when desired can be made by using twice as much of the barley.

Rice, Wheat, or Oatmeal Water, etc.—These may be made in the same manner as the barley water, using the same proportions either of the flour or the grains. These are useful as additions to milk for healthy infants who have reached the age of five or six months; they may also be given in many cases of acute or chronic indigestion when milk must be omitted or given in small quantities. When there is a tendency to constipation oatmeal is preferred; when to looseness, barley, wheat, or rice water.

INFANT FOODS

It is not possible, nor even desirable, for a physician to know all about the infant foods with which the market is flooded. He should, however, know at least that they are not perfect substitutes for breast-milk, that as permanent foods they are greatly inferior to properly modified cow's milk, and that they are capable of doing and have done much positive harm. Scurvy has so frequently followed their prolonged use, when given without the addition of fresh milk, and sometimes even when they have been given with it, that there can be no escaping the conclusion that they were the active cause. Their general use is condemned with practical unanimity by authorities on infant feeding. Yet by industrious and skilful advertising they are forced upon public attention, and are extensively used by the laity and even by the medical profession. They are expensive. They add little or nothing to our resources in infant dietetics; in fact, they tend to retard rather than advance our knowledge of this subject.

There are, however, a few occasions when some of these preparations may be useful as temporary expedients or when nothing better can be obtained. They should be used only with a very definite knowledge of exactly what they do and what they do not contain. Their name is legion; but those most commonly employed in this country may be grouped as follows:

1. **The Milk Foods.**—Nestlé's food is perhaps the most widely known. The others closely resembling it in composition are the Anglo-Swiss, the Franco-Swiss, the American-Swiss, and Gerber's food. These foods are essentially sweetened condensed milk evaporated to dryness, with the addition of some form of flour which has been dextrinized; they all contain a considerable proportion of unchanged starch.

2. **The Liebig or Malted Foods.**—Mellin's food may be taken as a type of the class. Others which resemble it more or less closely are Liebig's, Horlick's malted milk, and cereal milk. Mellin's food is com-

posed principally (80 per cent) of soluble carbohydrates. They are derived from malted wheat and barley flour, and are composed chiefly of a mixture of dextrins, dextrose, and maltose.

3. **The Farinaceous Foods.**—These are imperial granum, Ridge's food, Hubbell's prepared wheat, and Robinson's patent barley. The first consists of wheat flour previously prepared by baking, by which a small proportion of the starch—from one to six per cent—has been converted into sugar. In chemical composition these four foods are very similar, consisting mainly of unchanged starch which forms from seventy-five to eighty per cent of their solid constituents.

4. **Miscellaneous Foods.**—Under this head may be mentioned Carnrick's soluble food and Eskay's food. The composition of these is given in the following table:

Composition of Infant-Foods [1]

	Nestlé's Food.	Mellin's Food.	Eskay's Food.	Malted Milk.	Ridge's Food.	Imperial granum.	Carnrick's food.
	Per cent.	Per cent.	Per cent.	Per cent	Per cent.	Per cent.	Per cent.
Fat	5.50	0.24	1.16	8.78	1.11	1.04	7.45
Protein	14.34	11.50	5.82	16.35	11.81	14.00	10.25
Cane sugar	25.00	.....	.....	.....	.....	.....	.....
Dextrose	.....	.....	53.46 [2]	.....	0.52	0.42	.....
Lactose (milk sugar)	6.57	.....		49.15 [3]	.....	.....	.....
Maltose	27.36	60.80	.....		.....	.....	.....
Dextrins		19.20	14.35	18.80	1.28	1.38	.....
Total Soluble carbohydrates	58.93	80.00	67.81	67.95	1.80	1.80	27.08
Insoluble carbohydrates (Starch)	15.39	.....	21.21	.....	76.21	73.54	37.37
Inorganic salts	2.03	3.59	1.30	3.86	0.49	0.39	4.42
Moisture	3.81	4.73	2.70	3.06	8.58	9.23	3.42

[1] With the exception of Nestlé's food and Carnrick's soluble food, these analyses were made for the authors by E. E. Smith, Ph.D., M.D., of samples purchased in the open market. [2] Chiefly lactose. [3] Largely maltose.

The essential feature of all infant foods is that they are composed principally of carbohydrates and are lacking in fat. Some of them contain a large proportion of unchanged starch. Furthermore, their protein, though often sufficient in amount, is chiefly vegetable, not animal protein. No one of them can be regarded in any sense as a proper substitute for breast-milk.

Some of these foods—Nestlé's and other milk foods, malted milk, cereal milk, and Carnrick's food, and even some of the farinaceous foods like imperial granum—are advertised as substitutes for breast-milk and recommended for use alone. Others, such as Mellin's, Liebig's, and Eskay's foods, are intended to be used with milk. The use of any of the

commercial foods alone is admissible only for short periods during derangements of digestion, when we wish to withhold for the time all milk fat. Their prolonged use almost invariably produces some grave disorder of nutrition, most frequently scurvy. Those foods which require in their preparation the addition of milk are open to less serious objections, but are not necessary or even desirable. They should never be used with condensed milk. When added to fresh milk they may furnish the additional carbohydrates required by an infant fed upon a diluted cow's milk. In such a case they take the place of milk sugar or cane sugar in the milk modification. There is no proof to sustain the claim that they increase the digestibility of cow's milk. Farinaceous foods may be used as an addition to milk after the sixth or seventh month and during the second year.

CHAPTER III

INFANT FEEDING

CHOICE OF METHODS OF FEEDING

The different methods of feeding which are available are:

1. Breast-feeding, either by the mother or by a wet-nurse.
2. Mixed feeding, or a combination of nursing and artificial feeding.
3. Artificial feeding exclusively.

In deciding by which one of these methods a child shall be fed, many circumstances must be taken into consideration: the vigor of the child, the health of the mother, and especially the surroundings, since these determine very largely the success or failure of any method employed.

Maternal Nursing.—This is the natural and the ideal method of infant feeding. Every mother should nurse her infant unless there are some very weighty reasons to the contrary. The physician should do all in his power to encourage maternal nursing and to promote its success. He should explain to the mother how important breast-milk is for the child; that fully four-fifths of the deaths under one year are in infants who are artificially fed. He should also make clear the conditions by which alone successful nursing can be accomplished; viz., proper diet, regular habits of sleep and exercise, and a simple life, in so far as possible free from causes of nervous excitement, fatigue, overwork, or worry. Social engagements should be avoided. Much can be done by patience and persistence even in the face of many discouraging circumstances. Nursing may be furthered by proper care of the nipples before delivery, and by attention to them during the early days of nursing to

prevent fissures and mastitis, which often interrupt successful nursing.

As a result of extensive propaganda the number of mothers of all classes of society who nurse their children in the United States has beyond question materially increased during the last ten years. This is a hopeful sign. Among the poor and ignorant where artificial feeding is not likely to be well done, all possible efforts should be made to increase maternal nursing as the most effective means of reducing infant mortality.

When Maternal Nursing Should not be Attempted.—(1) No mother who is the subject of tuberculosis in any form, whether latent or active, should nurse her infant; it can only hasten the progress of the disease in herself, while at the same time it exposes the infant to the danger of infection. (2) Nursing should seldom be allowed when serious complications have been connected with parturition, such as severe hemorrhage, puerperal convulsions, nephritis, or puerperal septicemia. After severe hemorrhage and even after sepsis, women may recover so as to nurse successfully. There is great danger to the child in nursing after eclampsia; even when put to the breast two or three days after the mother's last attack, fatal convulsions have followed. (3) If the mother is suffering from any serious chronic disease or is very delicate, since great harm may be done to her without any corresponding benefit to the child. With reference to the last-mentioned condition, an absolute opinion can not always be given at the outset. As a rule, mothers are more likely to succeed in nursing first or second children than subsequent ones. One should not be too ready to decide that there will be no milk, but should persist in stimulating the breasts by suckling the child. The milk may be delayed until the tenth or twelfth day, and yet come in such abundance that nursing may be successfully carried on for many months. In general the capacity for lactation diminishes with each successive pregnancy.

Artificial Feeding vs. Wet-Nursing. When maternal nursing is impossible or undesirable, the milk of another woman would seem to be the most natural and best substitute. While this is theoretically true, the practical obstacles are so many as to put wet-nursing out of the question as a general method of feeding. We have in America no peasant class like that of Europe to draw upon; and in the class which furnishes most of our wet-nurses the capacity to nurse has steadily diminished. The expense of a wet-nurse—thirty to forty dollars a month in New York—the danger of transmitting contagious disease, and the difficulty of obtaining proper care for her own infant, are all very serious objections to wet-nursing. The recent advances in artificial feeding have placed it now on quite a different footing from that which it formerly

occupied. While it is true that good breast-milk is unquestionably the best food, it is equally true that properly modified cow's milk is a far better food than the milk of many wet-nurses who are employed. These facts added to the constantly increasing difficulty of obtaining good wet-nurses have caused wet-nurses to be pretty generally discarded, even in our large cities, where formerly no other substitute for maternal nursing was considered.

There are, however, some conditions in which wet-nurses are necessary, even indispensable. Some infants, usually those who have been badly started, can not be made to thrive upon any form of artificial feeding. There are also premature infants and other very delicate ones whose powers of assimilation are so feeble that they are reared in any circumstances only with the greatest difficulty, but whose chances of life are much increased by a good wet-nurse. Again, in young infants who have been suffering for some time from chronic indigestion and failing nutrition, the symptoms of acute inanition sometimes develop with great rapidity and severity. From such a condition, apparently hopeless, infants may sometimes be rescued by the timely assistance of a good wet-nurse.

The difficulties in the way of successful infant feeding in hospitals, foundling asylums and other institutions for young infants are such that in them partial wet-nursing should be employed whenever possible, at least long enough to give the infant a good start.

Mixed Feeding.—Mixed feeding, or a combination of nursing and artificial feeding, may be employed whenever the supply of the nurse is insufficient. The use of one or two feedings a day from the bottle after the third or fourth month may do much to relieve the mother from the strain of nursing entirely, without disturbing the infant's progress. During the later months more feedings may be introduced for the purpose of gradual weaning.

BREAST-FEEDING

Care of the Breasts during Lactation.—For the safety of both mother and child it is essential that the most scrupulous attention be given to cleanliness. The nipples, and the breasts as well, should always be carefully washed after each nursing. Usually plain water is sufficient, or a weak boric-acid solution may be employed.

Nursing during the First Days of Life.—This is necessary, to accustom the child and the mother to the procedure, and to empty the breasts of the colostrum; it probably also promotes uterine contractions. All these results can be attained by putting the child to the breast on the first day once in six hours, on the second day once in four hours. The

child gets from the breast only from four to six ounces a day during the first two days. Did he require more nourishment before the milk-flow is fully established, we may be sure that Nature would not have been so late with her supply. The common practice of administering to an infant a few hours old all sorts of decoctions, with the idea that because he cries he is suffering from colic, can not be too strongly condemned. A certain amount of crying is desirable. In exceptional circumstances, when an infant is unusually large and strong and cries excessively, it may be necessary to give food even on the first day; but this is not to be the rule. A little warm water should first be given; from two to four teaspoonfuls at a time are sufficient. If this does not satisfy the child, regular feeding should be begun on the second day. Should the milk be delayed beyond the second day, the child should be put to the breast at regular intervals, but only for two or three minutes, and then given the bottle afterwards if still hungry. It is important not to cease in our efforts to induce a secretion for several days longer, and the best of all means is the stimulation of the child's sucking.

Nursing Habits.—Good habits of nursing and sleep are almost as easily formed as bad ones, provided one begins at the outset. Much of the wear and tear incident to the nursing period may be avoided if the child is trained to regular habits. Attention to these minor points often makes all the difference between successful and unsuccessful nursing. After the third day, seven nursings in the twenty-four hours are sufficient, and no more should be allowed. An infant at this age can usually be depended upon to take at least one long sleep of from four to six hours in the twenty-four. For the rest of the day the child should be awakened, if necessary, at the regular nursing time, and put to the breast; this plan being continued until ten o'clock at night. He should then be allowed to sleep as long as he will, and but one nursing given between this hour and six in the morning. In the course of two or three weeks a healthy infant can usually be trained to nurse and sleep with almost perfect regularity—frequently, when a month old, going six hours regularly at night without feeding. A trained nurse of our acquaintance states that out of thirty-three infants of which she had the care from birth, thirty-one were trained without difficulty in the manner stated. So far as the child is concerned, regular habits of feeding and sleep, and regular evacuations from the bowels, which nearly always go with them, are most important factors in infant hygiene.

Less frequent nursing and relieving the mother of night nursing after the child is three months old are of the greatest value, and by lessening the wear and tear of nursing will often enable her to continue lactation, when otherwise it would be brought to an abrupt termination. On no account should the child be allowed to sleep upon the mother's breast,

Schedule for Breast-Feeding

Age.	Number of Nursings in 24 Hours.	Interval During the Day.	Night Nursings Between 10 p.m. and 6 a.m.
		Hours.	
First day	4	6	1
Second day	6	4	1
Three days to three months	7	3	1
Three to six months	6	3	0
After six months	5	4	0

nor in the same bed with the mother. The temptation to frequent nursing is thus largely removed. No mere sentiment in regard to these matters should be allowed to interfere with the plain dictates of reason and experience.

Symptoms of Unsuccessful Nursing during the Early Weeks.—Attempts at maternal nursing so often result in failure, jeopardizing the health, and even endangering the life of the child, that it becomes a matter of the greatest importance to decide this question of nursing aright, and as early as possible. On the one hand, one should not hastily wean a child on account of symptoms which may have no connection with the food, nor should one advise weaning when the indigestion from which the infant is suffering is due to causes which are temporary and remediable. On the other hand, nursing should not be continued simply because a conscientious mother desires it, when every indication points to failure. If artificial feeding is to be employed the difficulties are fewer when it is begun early than after the digestive organs have been deranged by several weeks of poor nursing. These cases form a very considerable group and present peculiar difficulties in practice. While a decision is being reached as to the ability of the mother to nurse, there is required close observation and a careful study of all the conditions, and even then the physician is liable to make mistakes.

The body-weight gives valuable information. The child does not gain or continues to lose after the usual initial loss of the first three or four days. Observations on the weight at least twice a week are necessary, and in cases presenting special difficulties the weight should be taken daily.

At times there may be no vomiting, diarrhea, or even severe colic, yet the child may fret and worry continually, sleep but little, and show general discomfort. Such symptoms are sometimes due to indigestion but are more frequently due to hunger. In other cases definite symptoms of gastric indigestion may be present, usually vomiting or frequent regurgitation of small amounts of undigested milk, later mixed with mucus;

eructations of gas with or without vomiting may occur, and distention of the stomach with gas and gastric colic may follow.

More often the symptoms of indigestion are intestinal. Occasionally there is constipation, but as a rule the stools are frequent, thin and green, containing flaky masses of undigested milk, and, after a short time, mucus is present. At times there is much gas and the stools are sour and irritating. If constipation is present there is apt to be severe colic and sometimes abdominal distention. The almost uniform absence of any elevation of temperature in these cases points strongly against the existence of any infection, which is further indicated by the prompt recovery under appropriate treatment.

Before considering the case one of inadequate nursing, or simple indigestion in a nursing infant, one should be careful to exclude organic conditions, particularly, if vomiting is present, hypertrophic stenosis of the pylorus.

As the first step one should endeavor to gain some idea as to the quantity of milk secreted. During the first week, particularly from the second to the fourth day, the temperature may be elevated quite apart from septic or inflammatory conditions or even evidences of indigestion. This is particularly seen where the breasts secrete almost nothing (see Inanition Fever). Often when the milk is very scanty something may be learned from the manner in which the child takes the breast. When the milk is abundant, five or six minutes are often sufficient. If the milk is very scanty, an infant will frequently nurse half or three-quarters of an hour and then stop, more because he is exhausted than because he is satisfied. Sometimes, when the breasts are practically empty, the child will seize the nipple and nurse vigorously for a few moments, then drop it in apparent disgust and refuse to make any further efforts. The only satisfactory way of determining the quantity of milk secreted is to weigh the infant before and after nursing. This should be done at each nursing until all doubt is removed. If the milk is merely scanty, but not otherwise abnormal, the infant does not gain, but may show no symptoms of indigestion, such as vomiting, colic, or undigested stools, and he frets and cries from hunger only.

An excessively rich milk is usually found under the following conditions: The mother is in good health, has large breasts which are full and tense at nursing time. In most cases she is upon a very abundant diet, getting little or no exercise, and frequently taking some alcoholic beverage with the notion that because the child is not thriving the milk is poor. The child may be colicky, sleepless, and uncomfortable, may vomit, may have frequent stools containing much undigested food, and may be losing in weight. A similar condition is often seen when a wet-nurse makes a change from the simple life and habits of her own home

to the more luxurious life and diet of the family to which she goes. The milk then has usually a high specific gravity, is high in fat and usually high in protein.

A scanty milk of a poor quality is most often seen when the mother is delicate or anemic, or perhaps has had a difficult or complicated labor, and who besides is anxious and worried. It is often with the greatest difficulty that one can secure the necessary half ounce required for examination. The milk is usually low in total solids and very low in fat. The specific gravity may be only 1.024 to 1.027, and the fat only one per cent or less.

A disturbed or disordered milk secretion is sometimes seen when the milk is scanty, often when it is very abundant. Like the group of cases just mentioned, this is frequently met with when the mother's general health is below the normal, but particularly is it influenced by her nervous condition. It is the highly nervous, emotional, worried woman whose milk we are now considering. During the first week or two the secretion may be excessive and then rapidly diminish; or, though the milk continues abundant, the infant does not thrive. It is most frequently found on examination that the milk is low in fat (0.50 to 1 per cent), while it may be high in protein (1.75 to 3.50 per cent). The child's symptoms are usually those of intestinal indigestion—severe colic, flatulence, and frequent, green, undigested stools.

Management.—The cause of the symptoms being in the food and not in the child, the futility of all medicinal treatment will be at once apparent. He who expects to relieve the symptoms of indigestion by the use of digestive ferments, by giving something before the nursing to dilute the milk, or to check frequent intestinal discharges by opium or astringents, will be disappointed. Temporary benefit often follows a dose of castor oil, but unless the milk can be materially changed in composition no permanent improvement in the child is to be looked for. The question usually to be decided relates to the continuance of nursing. We have a choice of four courses: (1) To continue nursing, endeavoring to correct the milk through treatment of the mother; (2) partly to nurse and partly feed from the bottle; (3) to stop all nursing temporarily, pumping the breasts meanwhile to keep up the secretion while we attempt to improve its character; (4) to wean at once and entirely. In deciding which of these courses is to be adopted we must take into consideration the condition of the child, the severity and duration of his symptoms, the findings of the milk examination, and the condition of the mother.

While the analysis of the milk is of some value in determining the course to be pursued, and should, if possible, be made, it is of much less importance than the child's symptoms. We must be guided not by what the milk contains, but by how seriously it disagrees. The chemical ex-

amination may show the milk to be of normal average in the proportion of its different ingredients and yet the child be seriously upset by it; on the other hand, a child may be doing admirably upon a milk which shows proportions which differ very greatly from the normal average. The question always concerns the effect of the particular milk upon the particular child.

When the symptoms of indigestion are severe or have been prolonged it is usually a mistake to attempt to relieve the condition by simply substituting some other food for part of the nursings. This seldom leads to any material improvement in the symptoms, while it does confuse the result, since we can not now tell whether it is the breast or the bottle feeding which disagrees. A better plan is to stop nursing entirely for a time and try the bottle alone. If the symptoms are at once relieved the weaning should be permanent.

When symptoms point to a scanty milk, but of fair quality—i. e., infant not gaining but without any particular symptoms of indigestion—one is often able to overcome the difficulties and continue the nursing to advantage. Until a decided increase in the milk has occurred the child should have supplementary feedings from the bottle in sufficient number to insure his being properly nourished. This may be done by giving one or two entire feedings a day from the bottle or a smaller amount may be given immediately after each nursing. In this way the advantage of the stimulating effect of suckling upon the secretion of milk is secured.

In the treatment of the mother the first thing is to secure for her an undisturbed rest at night. If possible, she should be entirely relieved of the care of the infant at this time, and if feeding is necessary the bottle should be given. She should have a certain amount of fresh air every day, driving if possible, or walking as soon as she is able to take more active exercise. Gentle massage of the breasts is often useful in stimulating secretion. It should be done with care and with every precaution against infection, and may be repeated two or three times a day for ten minutes. The diet should be abundant, with a large allowance of milk and meat, especially beef. If there is anemia, iron should be given. Every means should be taken to improve her general nutrition, and allay her nervous symptoms for whatever benefits these improves the milk. If the conditions present are incident to the confinement or the convalescence, the prognosis is good; and in the course of a week or two very marked improvement may be evident, and lactation may be successfully continued. If, however, the conditions depend upon constitutional debility, the prognosis is much worse. Temporary improvement may take place, but it soon becomes evident that the nursing is a failure.

When the symptoms are found to be associated with an over-rich milk the prospects for continuing nursing are much better than when the milk is poor. Unless the infant's digestion is very feeble or has been seriously upset either with vomiting or diarrhea, one can usually so alter the milk by treating the mother as to make it possible to keep the baby at the breast. Alcohol should be prohibited; the diet, especially the amount of solid food, should be reduced, and the mother required to take daily exercise in the open air, particularly by walking. The intervals between nursings should be lengthened, always to three hours, and often to four. In some cases there is an advantage in diluting the milk by allowing the child to take water before putting him to the breast. The improvement following such a change in regimen is often immediate, and with increasing age and weight the child gradually becomes accustomed to and is able to digest the rich milk. If, however, the child's symptoms of indigestion are of an aggravated type, whether gastric or intestinal, it will be necessary, even though the weight is increasing normally, to stop nursing entirely for a time. The breasts should be pumped at regular intervals and the child placed upon some other food until the symptoms are relieved, and then brought back gradually to breast-feeding.

If the examination shows the milk to be of very poor quality (i. e., low in fat, low in total solids), whether scanty or abundant, the outlook is not good. It is seldom that the conditions affecting the mother, to which such a milk is due, can be removed.

When we see a fretful, colicky, sleepless infant with either no gain in weight or a loss of a few ounces a week, and with stools which never approach the normal, and these conditions have lasted for three or four weeks, we are justified in taking the child from the breast at once. When the symptoms are less pronounced, and especially when, in spite of all discomfort and indigestion, the infant is gaining in weight, even though not rapidly, further efforts may be made before weaning is ordered.

Summary.—Poor milk is usually low in fat and scanty in quantity, while the protein may be either high or low. Very rich milk is usually high both in fat and protein. Very poor milk can seldom be permanently improved unless the causes are very definite and of a temporary character. Over-rich milk can usually be improved if the true explanation for it can be reached. Results are to be judged not so much by the change in the composition of the milk as by improvement in the infant's symptoms. Since good feeding gives so much better results than poor nursing, if circumstances are such that artificial feeding can be properly done, it is advisable to stop nursing after a fair trial—e. g., of two to three weeks—has been made, rather than waste time in pro-

longed efforts to improve the breast-milk. On the other hand, under conditions in which feeding is likely to be very badly done, one should persist for a longer time in efforts to promote lactation. But in no circumstances should one hastily and without carefully considered reasons advise a woman not to try to nurse her baby.

Wet-Nursing.—In the selection of a wet-nurse, it is by no means so essential as has generally been supposed, that her child shall be of about the same age as the child she is to nurse, for, after the first two or three weeks, the changes in the composition of breast-milk are insignificant. It is always desirable that the wet-nurse shall have nursed her own infant long enough to demonstrate the fact that she has an abundance of good milk; hence, taking a wet-nurse at the end of the first or second week is always fraught with considerable uncertainty. It is the quality of the milk, not its age, which determines whether or not it will agree. For an infant over one month old, a good wet-nurse whose milk is anywhere between one and six months old will usually answer perfectly well; and even for premature infants such a milk may be used without hesitation, but it should at first be diluted.

A good nurse must, first of all, be a healthy woman, free from syphilitic or tuberculous taint. The evidence afforded by a careful physical examination of the nurse and her own child may be considered sufficient. The tuberculin skin test is of no value in deciding whether a nurse shall be accepted or rejected. We are not yet in a position to assert that a Wassermann test should be employed in every case before selecting a nurse. The nurse must have good mammary glandular development. The breasts should be full and hard three hours after nursing. They may be very large and yet supply very little milk, being then composed almost entirely of fat. On the other hand, some smaller breasts may be almost all glandular tissue and secrete an abundance of milk. The difference in the size of a breast before and after nursing is one of the best guides as to the amount of milk it is secreting. The nipples should be free from erosions or fissures, and long enough for the needs of the child. Preferably a wet-nurse should be of a phlegmatic temperament, and of a good moral character. This is desirable for personal reasons, although there is no evidence of moral qualities being transmitted through the milk. It is desirable that she should be between twenty and thirty years of age, although much more depends upon the individual than upon the age. An examination of the milk may be of some assistance in selecting a nurse; but the best evidence to be obtained of the character of a woman's milk is the condition of her own child, which should always be seen before she is accepted. It often happens that a woman who has had an abundant supply of milk for her own infant has very little for another infant for the first few

days in her new surroundings. It should not be too readily decided that she is incompetent as a nurse, for, under most circumstances, with proper treatment the regular flow of milk will be re-established.

Weaning.—Weaning should always be done gradually, when possible, for the sake of both mother and child. Sudden weaning is apt to be followed by an attack of acute indigestion in the infant. This, however, is not a necessary result, and usually depends upon the fact that the child is given too high percentages of cow's milk at the outset. Weaning in hot weather is usually to be avoided, but the harm from this is not nearly so great as sometimes results when lactation is unduly prolonged because of a prejudice against a change of food at this time. While there are many women of the lower classes who are able to nurse their children to advantage for the entire first year, the number of such among the upper classes is small. By the latter, nursing can rarely be continued beyond the ninth, and often not beyond the sixth month, without unduly draining the vitality of the mother and at the same time harming the child. Since the early months of breast feeding are the most important, every effort should be made to have the mother continue nursing for five or six months. There is seldom trouble in feeding a baby for the second half year who has done well upon the breast for the first half.

The late months of lactation, like the early months, require close watching. It is a common mistake to continue both maternal and wet-nursing too long, owing to a dislike of making a change when things are going tolerably. If it has not been done before for reasons previously considered, breast-feeding should be supplemented by other food by the ninth or tenth month in any case. The child's progress in weight is a good guide as to time of beginning. In the absence of evident signs of disease, a stationary weight for several weeks makes weaning advisable; a steady loss makes it imperative.

The accompanying weight-chart (Fig. 17) illustrates this point.

When a nursing infant has been accustomed from birth to take one feeding a day from the bottle—always a great convenience to a nursing mother—gradual weaning is generally an easy matter; otherwise it is sometimes an impossibility, the child refusing all food except the breast so long as this is given, and nothing but starvation inducing him to take food either from a bottle or a spoon.

Sudden weaning may be required at any time from the development in the mother of acute disease of a serious nature, such as typhoid fever or pneumonia, or grave chronic disease, such as tuberculosis or nephritis, from the intercurrence of pregnancy, or from disease of the mammary gland. Through many of the minor ills—mild attacks of bronchitis, pharyngitis, indigestion, and even malarial fever—mothers frequently

nurse their children without any seeming detriment to them or to themselves. In acute illness of short duration, if severe, it is usually better, unless we decide to wean altogether, to feed the child from the bottle and to maintain the flow of milk by the occasional use of the breast-pump three or four times a day rather than to allow it to dry up. The previous flow can often be re-established after an interruption of a week or two, and sometimes after a much longer time.

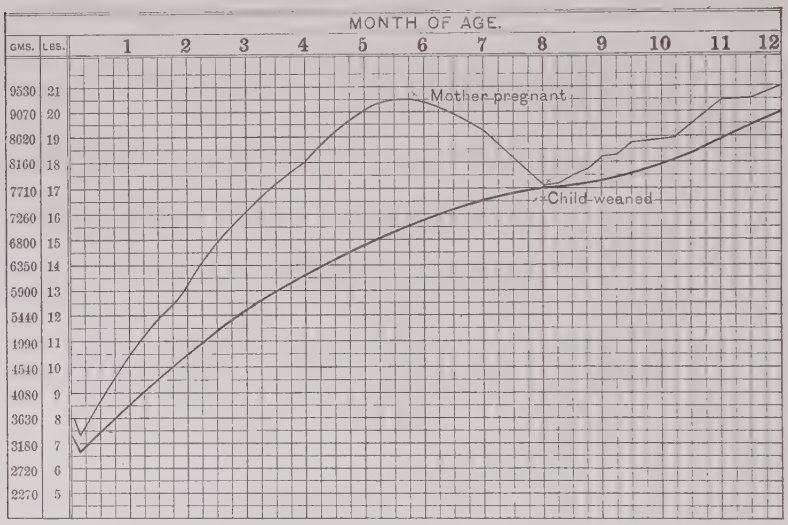


FIG. 17.—CHART SHOWING THE EFFECT OF PREGNANCY UPON THE WEIGHT OF A NURSING INFANT. The upper line is that of the patient; the lower one is the average line for the first year. The infant did unusually well until the sixth month. As it did not seem ill, the parents were not disturbed until the loss had reached 3 lbs. Feeding was at once begun, and child gradually regained its lost weight. It was subsequently discovered that the mother was pregnant.

In cases of sudden weaning, the food should in the beginning be very much weaker than for an artificially fed child of the same age. The change can then be made without causing disturbance. When the infant has become somewhat accustomed to cow's milk the strength of the food may be gradually increased.

The difficulties in weaning a child who up to nine or ten months has had no food but the breast, are sometimes great. Much time and tact are necessary on the part of both physician and nurse in these cases. To try to teach older infants to take the bottle is unwise; feeding from cup or spoon is usually quite as easy. Continued coaxing of food is objectionable; forcing is much worse and prolongs the struggle. In our experience we have found the best way to offer food at regular intervals and to take it away at once if refused. This is repeated every three or four hours. A variety of things may be offered—modified cow's

milk, thick gruels, beef juice, broths, bread and milk, etc. The nature of the food seems to make very little difference. A strong-willed child will often hold out for twenty-four or thirty-six hours, and occasionally a very stubborn one is found who will do so for forty-eight hours. At the end of this time the pangs of hunger are generally so acute that he capitulates. Serious symptoms from withholding food in such circumstances we have never seen.

MIXED FEEDING

By mixed feeding is meant a combination of nursing and artificial feeding. There are no objections to this practice; on the contrary, there are great advantages in giving an infant only a few breast-feedings a day when more are impossible. This may frequently be done in hospital practice, and thus a single wet-nurse may assist in the feeding of several infants. Mixed feeding may be resorted to whenever the milk supply of the mother is insufficient. If at any time the mother's health begins to suffer, she may be relieved of night nursing or of one or more nursings during the day, and the bottle substituted. In this way she may be enabled to continue lactation for some time longer than would otherwise be possible. Mixed feeding is often necessary during the first few weeks, while the mother's milk is insufficient in consequence of something which has retarded her convalescence. For the advantage of the stimulation to secretion afforded by the child's nursing, it is usually better, rather than alternate the breast and the bottle, to put the child at first to the breasts. After he has emptied them, additional food may be given from the bottle if the baby is still hungry. The milk may become abundant and of good quality as soon as the mother is well enough to be up and out of doors, although it was previously scanty and of inferior quality. Two or three feedings a day from the bottle helps to bridge over this period and prevent the child's nutrition from suffering. But before allowing a mother partly to nurse and partly to feed her infant, one should be sure that the quality of her milk is good.

ARTIFICIAL FEEDING

The scientific feeding of infants, whether with woman's milk or some substitute, demands as a basic principle that the food furnish what the body needs for heat and the repair of waste or the "maintenance requirements" and also for its normal development or "growth requirements." In breast feeding there is, under normal conditions, a certain automatic adjustment between the amount of food needed and the amount

supplied. If the milk taken is greatly in excess of requirements, this excess is either disposed of by vomiting or passes through the bowels in large partly digested stools. Sometimes this results in considerable disturbances of digestion; but usually they are slight. If the milk secreted is much below the child's requirements, this fact becomes evident by slower growth and by symptoms of defective nutrition, of which the weight is the best guide.

In artificial feeding, simply because the food given is not a normal one for the individual, it becomes even more important that the requirements of the infant, as nearly as they can be determined, shall be met. With any substitute both an excess and a deficiency are more potent for harm than with the natural food of the infant. The best results with artificial feeding, i. e., most satisfactory growth and freedom from disturbances of digestion, are seen when all that the body needs is supplied but no more than this.

The appetite of the child has been deemed by many a sufficient guide to the amount of food needed; to give a child all he will take at one time and postpone the next feeding until he shows that he is hungry has been advocated as a "natural" method of feeding as opposed to the more commonly followed plan of definite quantities at regular intervals. Though important, the child's appetite alone can hardly be relied upon. There are many infants, like many adults, who will habitually take too much food if it is offered. Disorders of digestion not infrequently are accompanied by an unnatural desire for food.

Formerly, it was customary to indicate the amount of food given to an infant by stating the number of ounces in twenty-four hours. This, however, is really meaningless unless the strength of the food is also mentioned. In deciding the amount of food to be given the nutritive or caloric value of the food must be taken into account. We must know approximately the infant's needs, best stated in calories, and then in what form these may best be furnished, best stated in the percentages of the different food elements.

From numerous observations the nutritive needs of an infant of average size and weight and activity in health have been shown to be 100 to 110 calories per kilo. (45 to 48 per pound) of body weight for the early months of the first year; gradually diminishing to 70 to 80 per kilo. (30 to 35 per pound) by the end of the year. A food much above or one much below normal requirements may be equally unsuitable and therefore unsuccessful. The physician should therefore be able to calculate the caloric value [1] of the food given to see, if possible, when an infant is not thriving, where the mistake lies.

[1] The caloric value of any modification of cow's milk of known percentages may be calculated as follows:

For the average healthy infant the weight is perhaps the most important single factor, but age, size, appetite and general behavior must also be taken into account. The experienced physician or nurse by closely watching a child's symptoms is able to decide whether the food is adequate, excessive or deficient. Food needs based on weight are useful as a general guide until the individual factor can be determined.

General Principles.—There are certain principles in infant feeding upon which all pediatrists are agreed: Woman's milk is not only the best, it is the ideal infant food; in any substitute certain conditions must be fulfilled.

1. All the different food constituents—fat, carbohydrate, protein, salts and water, must be furnished.

2. They must be supplied in sufficient quantity for the physiological requirements of the infant for growth, energy and repair.

In this respect as in many others Nature tolerates considerable varia-

---

For instance, 36 ounces of food having fat, 3.50; sugar, 7.00; protein, 1.75 per cent.

.035 (fat per cent) x 9.3 caloric value of fat = .325 caloric value of fat in 1 gram of food
.07 (sugar " ") x 4.1 " " " sugar = .287 " " " sugar " 1 " " "
.0175 (protein " ") x 4.1 " " " protein = .072 " " " protein " 1 " " "

.684 " " " one gram of food

.684 x 30 = 20.5 (caloric value 1 ounce of food) x 36 = 738, caloric value total food

Such calculations are too laborious for practical use. Fraley (Archives of Pediatrics, 1912, p. 123) has deduced a simple formula which makes this an easy matter and gives results quite accurate. This we have slightly modified:

Twice the fat percentage, plus sugar percentage, plus protein percentage, multiplied by 1.3, gives the calories per ounce of food. Applying this to the formula above mentioned: $7 + 7 + 1.75 = 15.75 \times 1.3 = 20.5$ calories per ounce.

Another simple way is to multiply the caloric value of each of the ingredients in the food by the amount of each that is taken.

Approximate Caloric Value of Different Foods

	Ounce	Even Tabl'sp'l.		Ounce	Even Tabl'sp'l.
Woman's milk	20		Dextrimaltose	120	40
Cow's milk	20		Malt soup extract	80	40
Cream (20 per cent)	60		Barley flour	100	35
Top-milk (7 per cent)	30		Wheat flour	100	28
Skimmed milk (1½% fat)	14		Oat flour	115	40
Whey, buttermilk, fat-free milk	10		Barley gruel (1 oz. to 10 oz.)	10	
Sweetened condensed milk	100		Barley water (1 tablesp'ful to 1 pt)	2	
Evaporated milk	55		Albumin water (white one egg to 1 pt.)	1	
Dried milk (Mammala)	127	40	Beef juice	6	
Milk sugar	120	40	Orange juice	15	
Cane sugar	120	60	Olive oil	245	122

Approximate Measures

Milk sugar	3	even tablespoonfuls	=	1	ounce by weight
Cane sugar	2	" "	=	1	" " "
Dextrimaltose	3	" "	=	1	" " "
Barley or oat flour	3	" "	=	1	" " "
Wheat flour	4	" "	=	1	" " "

tion from what is best without seriously hampering health or growth. Yet there is a maximum which if exceeded causes disturbances of digestion from overfeeding, and a minimum below which the body suffers from imperfect nutrition. Mistakes in the amount of food may be just as serious as those in its composition.

3. The food constituents must be furnished in suitable proportions. The proportions best suited to the infant's needs are shown in the composition of an average specimen of woman's milk. But as the normal variations in woman's milk may be considerable without affecting the infant unfavorably, so a certain amount of latitude in the composition of the artificial food which is substituted for woman's milk is tolerated. To a certain extent the different food elements, notably the fats and carbohydrates, are interchangeable; but this substitution must not be carried too far nor continued too long. In the scientific feeding of animals much stress is laid upon the importance of a properly "balanced ration" or one in which all the food elements are adequately represented. The same necessity exists in infant feeding. Woman's milk is such a balanced ration and we cannot give for a long time a food in which the proportion of the different food elements differs widely from those which woman's milk contains without incurring serious risks.

Cow's milk in some form is now almost universally accepted as the basis of artificial feeding. The milk of the goat or of other animals, though at times advantageous where good cow's milk is not available, has, because of many circumstances, never been general. In adapting cow's milk for infant feeding we must realize at the outset that no matter how it may be altered it is not a perfect substitute for woman's milk. There is no perfect substitute. But while its disadvantages may not be altogether removed, they may be lessened by certain changes, technically known as the "modification" of cow's milk.

Differences between Cow's Milk and Woman's Milk.—There are certain differences between cow's milk and woman's milk upon which these modifications are based. These relate both to the amount of the several constituents and their digestibility. The following table gives the proportions of the various elements which make up the two milks:

	Woman's Milk Average.	Cow's Milk Average.
	Per cent	Per cent
Fat	3.50	4.00
Sugar	7.50	4.75
Protein	1.25	3.50
Salts	0.20	0.75
Water	87.55	87.00
	100.00	100.00

These quantitative differences are important. It will be seen that cow's milk has a great excess of protein and salts and is deficient in sugar, while the proportion of fat in the two milks is nearly the same. When we come to use cow's milk in infant feeding, certain qualitative differences are discovered which from a practical point of view are of even more importance. The proper modification of cow's milk must take account of all these. During the past twenty-five years widely different opinions have been held as to the character of these differences between the two milks and consequently as to the nature of the difficulties which the infant has in digesting cow's milk. At different times the fat, the protein, the sugar and the salts have all been accused of being the chief cause of disturbances of digestion, and it is no doubt true that under certain circumstances any one of them may be a source of trouble.

Protein.—Cow's milk contains nearly three times as much total protein as does woman's milk; the greater part, about five-sixths, being casein, and one-sixth, albumin. In the protein of woman's milk the proportion of casein is about one-third; of lactalbumin, two-thirds. The casein of cow's milk differs in many respects from the casein of woman's milk. The excess of protein, especially the excess of casein, and the differences in the two caseins was long believed to be the chief cause of difficulty in digesting cow's milk. The studies of the past few years have, however, shown that the casein of cow's milk is remarkably well digested and absorbed under nearly all conditions. Like that of woman's milk it is converted into peptones and finally broken up into amino acids. Metabolism experiments, moreover, have shown that nitrogen retention in infants taking cow's milk is quite normal and examination of stools rarely shows evidences of undigested protein.

The chief difficulty in digesting casein of cow's milk seems to be mechanical, owing to its coagulation in the stomach of certain infants in large solid masses which offer some resistance to the action of the digestive fluids. Coagulation in large masses may be prevented in several ways: (1) by greater dilution of the milk; (2) by the use of gruels in the place of water as a diluent; (3) by boiling. Coagulation of milk in the stomach may be almost entirely prevented by the addition to the food of certain substances such as sodium citrate. It seems very doubtful if this is wholly desirable.

The amount of protein of cow's milk required for infant nutrition is greater than that of woman's milk. The reason apparently being that the casein of cow's milk, which is five-sixths of the protein, is deficient in certain amino acids essential for growth. These are supplied abundantly in woman's milk, whose protein is two-thirds lactalbumin. The defects of the casein of cow's milk are in a measure overcome by

increasing the quantity given. There is no evidence that the protein of cow's milk is harmful to the infant even when given in considerable excess of the amount contained in woman's milk. Disturbances of infant digestion are very rarely due to the protein of cow's milk.

Fat.—The high fat content of woman's milk indicates the importance of fat in the nutrition of the infant. The amount of fat in cow's milk is about the same as in a good average sample of woman's milk, i. e., 3 to 4 per cent. But there are certain important differences in the fat in the two milks. Thus the fat of cow's milk contains a much greater proportion (nearly eight times as much) of the volatile fatty acids. The marked difference in digestibility of the fat in the two milks is believed to depend to a considerable degree upon this fact. It is possible also that the freshness of the fat may have an influence. Be this as it may, it is found practically impossible to give to most infants as much of the fat of cow's milk as woman's milk contains. It is not wise to increase the amount of fat until symptoms of intolerance appear, for the intolerance to fat is more persistent than to any other ingredient of the food. Such intolerance once established, it may be weeks or months before a reasonable quantity can again be digested and absorbed. The tolerance to the fat of cow's milk varies greatly in different children. Some can take a large quantity and some only a small quantity. The difficulty is greatest with infants in the first few weeks, with the feeble and with those who have suffered from previous nutritional disturbances. Fat is also badly borne when there is disturbance of gastric or intestinal digestion, also in all febrile conditions, no matter from what cause, and during periods of very hot weather. A failure to regard these contraindications is a constant source of trouble in practice. The ability to digest fat is probably the best index of an infant's digestive capacity. Those who cannot take the usual amount certainly do not thrive as well as those who can. Hence it follows that no part of the milk modification needs to be more carefully watched than the amount of fat given. The percentage of fat that can safely be allowed to a healthy infant varies from 1 to 4 per cent. The latter figure should not be exceeded with any infant and with very many even this cannot be reached until the end of the first year.

Carbohydrates.—That all the carbohydrates of woman's milk are in the soluble form is a strong indication that soluble carbohydrates, or sugars, should be the form supplied in artificial feeding. The high proportion in which sugar exists in woman's milk—being considerably greater than all the other solid constituents combined—shows how important a part sugar serves in infant nutrition. In case sugar is not furnished in the food in sufficient amount, there must be more fat and protein supplied.

The sugar in cow's milk is identical with that in woman's milk, in both cases being lactose in solution. In artificial feeding we have a choice between milk sugar, cane sugar, and maltose.[1] All of these sugars are about equally well borne in health; they all, alike, have the capacity of increasing weight and furnishing heat. Yet there are some differences in their effects which make it advantageous at times to choose one rather than another. Milk sugar, being identical with the sugar in woman's milk, on theoretical grounds would seem preferable. It does not ferment with yeast. It is not so readily broken down in the stomach and hence with infants who have a disposition to vomit it is usually to be preferred to maltose or cane sugar. It is slightly laxative. It is usually well borne in health in proportions up to 6 or 7 per cent of the food. In all intestinal disturbances, particularly where there is a tendency to looseness of the bowels, lactose is badly borne.

Cane sugar has the great advantage of cheapness. In a very large proportion of cases it apparently does quite as well as lactose or maltose. It is distinctly less laxative but rather more likely to ferment in the stomach and cause or aggravate vomiting when given in the quantities mentioned for lactose.

Maltose, in the preparations in which it is used, has some peculiar advantages; first, its laxative effect which is rather greater than that of the other sugars; secondly, in inducing a more rapid gain in weight; and, finally, in a certain corrective action upon some digestive disturbances, especially when given with considerable amounts of starchy food. Maltose preparations have the disadvantage in breaking down more readily both in the stomach and in the intestine, often provoking and, in susceptible infants, always aggravating both vomiting and diarrhea.

For routine use lactose is to be preferred except where cost is a consideration; the other sugars are to be used with the special indications mentioned. There is often an advantage in using the different

[1] Pure maltose is expensive and practically not available for infant feeding. The maltose preparations used for infant feeding are mixtures of maltose and dextrins. In speaking of the use of maltose hereafter these preparations will be meant. Many such preparations are on the market. Loeflund's "malt soup extract" is reliable but expensive. Reliable and more moderate in price are the "neutral maltose" of the Maltzyme Co., the malt soup of the Maltine Co. and the "malt syrup" of the Freihofer Co., Philadelphia. These preparations are somewhat acid. To the first five grains and to the last ten grains of potassium carbonate should be added for each ounce of the malt used in the food. All of these liquid preparations contain from 65 to 85 per cent of carbohydrates, of which about two-thirds is maltose and the balance chiefly dextrins. Besides these liquid preparations, Borcherdt's "malt soup extract" and Mead's "dextri-maltose" in powder should be mentioned as convenient and reliable forms of maltose. None of the above preparations has any appreciable diastatic action.

sugars together since the amount that is well tolerated of the combined sugars is often greater than if the entire amount were one form of sugar.

Starches.—Even very young infants are able to digest starch, though their capacity during the early months is limited. After the fourth month it notably increases and after six or seven months most healthy infants can readily digest as much as one ounce of starch daily, and some can do much more than this. This fact makes it possible to use starch in the form of cereal gruels under a variety of conditions when they may be thought desirable. With very young infants their use is mainly as diluents for milk when the coagulation of the casein in the stomach in large masses is an obstacle to digestion. With older infants starches may supply a considerable part of the carbohydrates when there is marked intolerance of all sugars. For the very slow change of the starch into sugar in the intestines is much less likely to cause symptoms than when sugar itself in considerable amount is thrown at once into the intestine. Again, starches are useful to increase the total carbohydrates when all the sugar is being given that the patient can readily tolerate and especially when, on account of intolerance of fats, it is desirable to raise the total carbohydrates to a point considerably higher than is usually given.

Salts.—It has been customary in the past to add certain inorganic constituents to cow's milk used for infant feeding. Lime water has been most widely employed. As has already been stated in the previous chapter, not only calcium but practically all the salts of woman's milk are present in greater abundance in cow's milk, even when the latter has been diluted to the customary degree. These substances need not be added to milk to supply a deficiency in inorganic constituents, for there is none, except in iron. Their addition to correct the "excessive acidity" of cow's milk is unimportant, for as used they do not do this. In considerable amounts, lime water, sodium bicarbonate and sodium citrate all delay the coagulation of milk in the stomach, and in large amounts may entirely prevent it. Under certain conditions the first-mentioned effect may possibly be desirable. It is questionable whether the latter ever is. At the present time we are not in a position to assert that the addition to milk of lime water or any of the substances mentioned is of value as a routine practice in infant feeding. They may therefore be wisely omitted with all healthy children.

Feeding of Healthy Infants during the First Year.—It is absolutely necessary to consider separately the changes required by healthy infants with normal digestion and those required by infants with feeble or disordered digestion. From a failure to make this distinction much confusion has arisen. The digestion of all healthy infants is very much

alike and they can be fed in much the same way; while the variations afforded by infants with disordered digestion are very great.

There are two general plans according to which the indications outlined in the previous pages may be met. The first plan is to use whole milk as indicated in the table given below, the different formulas being derived by simple dilution and the addition of needed sugar or other carbohydrates. The table gives the quantities of the different ingredients, the approximate percentage composition and caloric value per ounce of the formula obtained. The age indications are not intended to be closely followed. Successful infant feeding cannot be done by rule of thumb. However, these formulas are a useful guide as a starting-point with an average child until his individual needs and capacity can be determined by observation. They indicate what such a child in health may be expected to take and also how rapidly and in what way the food may be increased.[1]

Formulas from Whole (4 per cent) Milk
Giving Approximate Percentage Composition and Caloric Value

	I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.	X.
Milk (ounces)	6	7	8	9	10	11	12	13	14	15
Water (ounces)	14	13	12	11	10	9	7	5	1	0
Gruel[1] (ounces)	....	....	....	....	....	....	1	2	5	5
Sugar[2] (even tabl'sp'ls)	2½	2½	2½	2½	2½	2½	2	1½	1	1
Total	20	20	20	20	20	20	20	20	20	20
Fat, per cent	1.20	1.40	1.60	1.80	2.00	2.20	2.40	2.60	2.80	3.00
Sugar, per cent	5.70	6.00	6.00	6.50	6.50	6.50	6.00	5.50	5.50	5.00
Starch, per cent	....	....	....	....	....	....	.40	.80	2.00	2.00
Protein, per cent	1.00	1.20	1.40	1.60	1.75	1.90	2.10	2.25	2.40	2.60
Calories per ounce	11.5	12.5	13.5	14.5	15.5	16.5	17.0	18.0	20.0	21.0
Approx. age indication	2 da.	1 wk.	3 wk.	2 mo.	3 mo.	4 mo.	5 mo.	6 mo.	8 mo.	9–11 mo.

[1] The gruel here indicated is made in the proportion of 1 oz. by volume to 10 oz. of water.

[2] Milk sugar is here indicated; of cane sugar use two scant tablespoonfuls instead of two and a half, and one instead of one and a half, etc. Maltose may be used in the same amounts as milk sugar.

[1] A simple method of calculating a milk formula for an average healthy infant on the basis of caloric requirements is to start with the daily amount of protein of cow's milk needed. This by experience has been found to be furnished in 1½ ounces of milk for each pound of body weight. An infant weighing 10 pounds will thus require 15 ounces of milk. His caloric needs calculated at 45 per pound will be 450. Of this there will be furnished in the milk (20 calories per ounce) 300 calories, leaving 150 to be made up by more fat or by carbohydrates—sugar or starch. One ounce of sugar will add 120 calories; or 1¼ ounces, 150 calories. This will give the food values for a day. There is still to be determined the amount of diluent, which will depend upon the infant's daily need of fluid. This has been shown to be about 3 ounces for each pound of body weight in the early months, and 2 ounces for each pound in the later

According to the second plan of feeding, after the first few weeks somewhat higher fat is employed than indicated above. This is accomplished by using the upper half of a quart bottle of milk, i. e., a 7 per cent top-milk [1] instead of whole milk. If this is done the amount of the milk used should be one-fourth or one-third less than is given in the table. These formulas may be used up to seven or eight months, when, with the introduction of larger amounts of starchy food, formulas from whole milk may be given. Such formulas are designed for infants who are able to take more fat than is contained in the formulas from whole milk. In this group will be found strong children with good digestion.

Relative Advantages of Formulas from Whole Milk and Those with Higher Fats.—Whole milk formulas are somewhat simpler to prepare and the method is therefore more easily understood by the average mother or nurse. With the ignorant or careless there is less chance of going wrong, for it eliminates one error, by no means an uncommon one, of using too high fats. There is quite a large group of infants who are unable to digest higher proportions of fat than are given in this series of formulas and who are seriously disturbed if they are given: but there is a third group, also a large one, who can easily take higher fats and some thrive much better when they are given. Constipation also is somewhat less frequently seen when top-milk mixtures are used. There are then advantages in having formulas with higher fats for use under proper conditions. If no more fat is used than is obtained by using a 7 per cent top-milk, as here advised, disturbances from fat will very seldom be seen in healthy children. When less fat is given the caloric value of the food must be made up by increasing the carbohydrates and the protein. Only to a limited degree is such a substitution possible. When fats are replaced by carbohydrates chiefly, quite serious disturbances of digestion may be produced. The great argument for the need of more fat than is obtained with dilutions of whole milk is the proportion present in woman's milk. On the whole, while one

months; i. e., for a 10-pound infant it will be 30 ounces a day. There will need to be added, therefore, 15 ounces of water. The formula will then be:

15	ounces	milk,	giving 300	calories
1¼	"	sugar	" 150	"
15	"	water		

The 30 ounces of food could be divided into seven feedings of 4¼ ounces each, or into six feedings of 5 ounces each according to circumstances. The approximate percentage composition of the formula, using 4-per-cent milk, would be: fat 2.00; sugar 6.00; protein 1.75.

[1] Before this top-milk is removed the milk should stand in the bottle at least four hours, and the top-milk should be carefully removed with a milk dipper, not poured off.

may get on very well with such simple formulas as those from whole milk, in experienced hands excellent and sometimes better results are obtained with healthy children with somewhat more fat. In infants with feeble or disturbed digestion top-milk formulas should not be used at all. The most important thing in artificial feeding is to recognize at the earliest possible moment the indications making necessary an alteration in the food.

Quantity at One Feeding and Frequency of Feedings.—The strength of the food and the daily quantity having been decided, the next question is the number of feedings in which it is to be divided and the intervals at which they shall be given. Experience has shown that the average infant can digest his food better if the intervals are made longer than was formerly the practice. With longer intervals the quantity given at one time and the strength of the food may be correspondingly increased. There are few healthy infants who cannot readily be trained to the intervals given in the table below, in which the infant is placed upon three-hour feedings at the outset and upon four-hour feedings when six months old. The reduced number of feedings also materially lessens the labor of the mother or nurse.

Schedule for Healthy Infants during the First Year

Age	Interval Between Feedings	Night Feedings After 6 p.m.	Feedings in 24 Hours.	Quantity for One Feeding	Quantity for 24 Hours
	Hours			Ounces	Ounces
2nd to 7th day.....	3	2	7	1 — 2	7 — 14
2nd, 3rd and 4th weeks..	3	2	7	2½ — 4½	18 — 32
2nd and 3rd months	3	2	7	3½ — 5	24 — 35
4th and 5th months....	3	1	6	5 — 6	30 — 36
6th, 7th and 8th months.	4	1	5	6½ — 7½	32½ — 37½
9th and 10th months...	4	1	5	7 — 8	35 — 40
11th and 12th months..	4	1	5	8 — 9	40 — 45

A large and vigorous infant will require the larger quantities allowed, but these seldom need be exceeded; for a small infant the smaller quantities mentioned, and sometimes less, will be sufficient.

This table really gives only the volume of food for the different ages. This is important as it secures to the infant a proper amount of water daily. The following table shows how the actual food requirements of an average infant may be met, using the formulas given on page 187, and in quantities mentioned.

A schedule like the following indicates the needs of a healthy infant of average size, weight and activity. But no schedule can be closely followed with any given child. One cannot conclude because an infant is

Age.	Average Caloric Requirements.	Furnished in
1 month	400	7 feedings 4¼ oz. of No. III.
2 months	500	7 " 4¾ " " No. IV.
3 "	560	7 " 5 " " No. V.
6 "	640	5 " 7½ " " No. VIII.
9 "	740	5 " 7¾ " " No. IX.

six weeks old he is able to digest a certain amount of food and a certain other amount because he is six months old. To attempt to follow any schedule too closely is to violate the fundamental principle of intelligent feeding, which is to adapt the food to the child's requirements and powers of digestion at the time. Because these figures represent averages they form a useful basis for feeding healthy children.

How and Where to Begin.—With all young infants, even those having presumably normal digestion, it is desirable to begin with a weaker food than would be indicated by their caloric requirements, and gradually increase both the strength and quantity according to the child's digestion. With small or feeble infants still weaker formulas should be used and the increase made more slowly.

For a healthy child with normal digestion who has previously had no cow's milk one should begin with a lower formula than would usually be given to a healthy child of his size and age, but may increase the strength and quantity of the food more rapidly than with a younger infant.

A stationary weight for a week or two, or even a loss of a few ounces, is of no importance, provided the change in diet can be effected without disturbing digestion; for as soon as a child becomes accustomed to cow's milk the percentages can be raised and progress is assured. Nothing is easier than to disturb the digestion in the beginning by the use of too strong food.

Indications for Increasing the Food.—While it is important to begin with weak food, it is a serious mistake to continue long with it. The powers of digestion are strengthened by gradually increasing the work the organs are given to do. Abrupt increases are almost certain to disturb digestion.

How rapidly the increase is made will vary much with the individual infant. With a vigorous child above average weight, and with good digestion, the strength and the quantity may be increased more rapidly than with a smaller or less robust one. We cannot increase the food every week or every month regardless of other conditions. The progress in weight is important, yet one should not be guided by it alone. When it is made the chief concern, there is a constant temptation, if the child is not

gaining as rapidly as the mother thinks he should, to increase the food, regardless of conditions and often beyond his requirements, usually with the result of seriously disturbing the digestion. The best of all guides to increasing the food is the child's demonstrated capacity of digestion. To determine this the child's symptoms should be carefully watched. If he is not satisfied and is digesting well it is usually safe to increase the food; but not more often than every three or four days in the early months, and every week in the later ones.

In increasing the quantity, it is not wise to add more than two or three ounces to the food for the day, or a quarter or half an ounce to each feeding. During the early weeks both the quantity and the strength of the food should be increased every few days. It is well to alternate, first increasing the quantity: then after a few days, if still unsatisfied, increasing the strength; the next time increasing the quantity again, etc. In this way will be avoided the error into which mothers and nurses often fall who adopt a single formula and keep on simply increasing the quantity indefinitely whenever the child is unsatisfied. The increase in strength should not be greater than from one formula to the next of the series given. It is sometimes advisable to make the increase by steps only half as great as specified.

A caution is necessary against changing the formula too frequently. It is not possible to modify the milk in such a way as to relieve every trivial discomfort or disturbance an infant may have. Nurses are usually ready to ascribe every slight symptom to the food, particularly if they have strong opinions of their own upon the subject of feeding and are not in full sympathy with the method employed. Very often the cause is outside the food and even of the organs of digestion.

To Determine the Percentage Composition of any Milk Formula.— In order to appreciate the composition of any milk formula which a patient may be taking it is desirable to reduce this to its approximate percentages. One who forms the habit of making such calculations soon finds it easy, and secures a basis for comparison with the percentages given as proper for the average normal child. A simple method of calculation is as follows: To determine the percentage of any constituent in the food, multiply its percentage in the original milk, cream, or top-milk by the number of ounces of each in the food, and divide by the total number of ounces of food prepared.[1]

---

[1] A child is taking the following food: Whole milk (4 per cent) 20 ounces, milk sugar 3 even tablespoonfuls, and water up to 35 ounces.

The fat in the food will be $\frac{20}{35}$ of 4 or 2.27 per cent.
The protein " " " " " $\frac{20}{35}$ " 3.50 " 2.00 " "
The sugar " " milk " " $\frac{20}{35}$ " 4.75 " 2.71 " "

Three even tablespoonfuls may be reckoned as 1 ounce of milk sugar, which.

Symptoms and Conditions Requiring Special Food Variations.—In a new case the most important guide in the first food prescription is a knowledge of the condition of the digestive organs. One should know besides the age and weight, the nature and quantity of the food which has been taken, the appetite, the number and character of the stools, and also whether digestive symptoms are present, such as vomiting, flatulence, diarrhea, colic or constant discomfort. In any case the first prescription is somewhat in the nature of an experiment. Success will depend on how intelligently the symptoms have been judged.

Even with infants who are properly fed there are few whose digestion remains perfectly normal throughout the entire first year. Changes in the food are therefore necessary from time to time to meet special symptoms which may arise. Many of these are due to disturbances of a minor character, but if they are recognized early and proper changes promptly made, more serious and protracted derangements of digestion can usually be avoided. This is not always an easy matter, but there are some indications which are very clear and definite.

Hot Weather.—The depressing effects of very hot weather upon young infants should be appreciated. At such times less food can be digested and less is required. Owing to an increase in perspiration, the amount of water, consequently the volume of the food, should seldom be reduced. The indications are best met by reducing the milk, the sugar and the starch in the formula and making up the deficiency by adding water, i. e., simply by diluting the food. Especially should the fat of the milk be reduced. An immediate change therefore should be made from any top-milk formula to one from whole milk or at times even to one from skimmed milk. Water should also be given freely between the feedings. But as some infants will not take it, the only alternative is to give an extra amount, half an ounce to two ounces, in each of the feedings. As soon as the period of excessive heat has passed, the infant can gradually be brought back to the usual food.

Minor Illnesses.—In attacks of acute rhinitis, otitis, tonsillitis, bronchitis, etc., even though not especially severe, the food should be reduced. The reduction should depend upon the severity of the attack and the amount of fever. The child's apparent appetite is often only a demand for water. At least as much is needed as in normal conditions and usually more should be offered. The indications may be met in the same way as outlined in the preceding paragraph.

Vomiting.—The common causes of habitual vomiting referable to the

---

in a 35-ounce mixture adds about 3 per cent of sugar. The total sugar in the food therefore is $2.71 + 3 = 5.71$ per cent.

The percentage composition of the food is: fat, 2.27; sugar, 5.71; protein, 2.00.

food are: too frequent feedings and too much food at one time; too much fat or too much sugar, especially if the sugar is either maltose or cane sugar. An infant who vomits often should not usually be fed at shorter intervals than four hours, even if only a few weeks old. If considerable quantities are ejected almost immediately after feeding, it is generally because too much food has been given. A diminution in the amount of food should bring about immediate improvement. When the sugar is in excess, or the fat, or both, there is vomiting or regurgitation of curdled milk or of a sour, watery fluid, which occurs frequently and often long after the feeding. The sugar should be greatly reduced or for a time entirely removed; cream mixtures or top-milk mixtures should not be used. If this is not sufficient, the fat should be still further reduced by using less milk or by partially skimming the milk. A return to the former diet should be gradual and for some time neither maltose nor cane sugar should be given.

Other causes must be considered also. The child may be moved about too much or sometimes the clothing may be too tight. More often this frequent regurgitation of food soon after feeding is in consequence of swallowed air which the child has taken with his bottle. This is more likely to be the case when an infant is fed while lying upon the back and when taking his food very slowly owing to a very small hole in the nipple. He is unable to expel the gas in that position, but if lifted to the erect position or placed over the shoulder once or twice during the feeding or after it, he will often bring up a large amount of gas, after which the vomiting ceases.

Constipation.—The principal causes of constipation referable to the food are, too small an amount of carbohydrates, and too small an amount of total solids, occasionally too low a proportion of fat. Habit and general training are also important factors. Sterilization, and to a slight degree pasteurization, cause milk to be somewhat constipating. During the first few weeks, if the food is rather small in amount, there is often a species of constipation present which is simply the result of the low total solids in the food given. The bowels may move every day, sometimes even twice a day, but the stools are often small and rather dry. Unless there is manifest discomfort on the part of the infant, such a condition may be disregarded, especially if the odor and color of the stools are nearly normal. As the proportions of all the elements of the food are gradually increased this form of constipation passes away. Mothers and physicians often expect that the bottle-fed infant will have during his first one or two months the two or three large stools daily to which they have been accustomed with healthy breast-fed infants; but finding instead only one movement a day, and that small and sometimes dry, they resort to laxatives or enemata, and by their use really

cause much of the trouble they are seeking to remove. If milk mixtures are made up without the addition of carbohydrates, constipation frequently results. This is often due to the alteration in the reaction of the contents of the intestines brought about by putrefaction of the protein.

Milk sugar is somewhat laxative and if a smaller amount is being used the raising of the proportion of this ingredient as high as 7 per cent will often be all that is needed. Maltose is more laxative in its effects and may be substituted wholly or in part for milk sugar. Its use will be more fully discussed later. Maltose should not be given if there is vomiting. Cereal gruels, especially oatmeal, also have a favorable influence upon constipation.

Colic and Flatulence.—The habitual colic of early infancy may occur with any form of intestinal indigestion; its causes therefore are varied. Colic and flatulence are especially common in infants who suffer from constipation. Excessive flatulence may occur also when cereal gruels are added to the milk of young infants, particularly if the amount is large. If symptoms are severe a reduction in all the elements of the food may be necessary.

"Curds" in the Stools.—The undigested masses appearing in the stools of infants taking milk are usually spoken of as "curds." These may be small, soft and white, and may make up a large part of the loose stool. An excess of mucus is usually present. Such masses are composed almost entirely of fat. There are also seen, but much less frequently, larger, smooth, hard masses of a yellowish-brown color, but white on section. They are generally present in small numbers in a stool, the rest of which may be quite normal. These hard or "bean curds," so called from their resemblance to lima beans, are composed chiefly of protein, usually with an envelope of fat. They are undoubtedly formed in the stomach, where the casein coagulates in masses, some of which are so firm and hard that they pass the intestine without being digested. Curds of this description are rarely seen unless the proportion of casein in the food is high.

Curds of the first variety, if numerous, call for a considerable reduction in the amount of fat. The large, smooth, hard curds, if numerous and persistent, may usually be made to disappear by boiling the milk. This causes the precipitation of the casein to occur in smaller masses which are more readily attacked by the gastric and intestinal secretions.

Loose, Green, or Yellowish-green Stools of a Sour Odor.—These are usually due to too much sugar, especially lactose, sometimes also to an excess of fat. The number of stools is usually from two to five daily. In appearance the stools resemble thin scrambled eggs. Stools such as those described are often seen in nursing infants as well as in those artificially fed, and the condition is not incompatible with steady and

regular gain in weight. After it has persisted any length of time, mucus is regularly present.

Large, Dry, Light-colored Stools.—Such stools are seen only if infants are fed preponderately or entirely upon cow's milk. The bowels are constipated and the stools may not be passed oftener than once in forty-eight hours. They are relatively large, however, and are so dry that the diaper may be hardly soiled. In addition, they are putty-colored or grayish-green and are very foul with the odor of putrefaction. On analysis they are found to be alkaline in reaction and to contain a large proportion of calcium and magnesium soaps. For a time, infants with such stools may improve and gain in weight. After a time, however, they cease gaining and eventually lose weight while anemia appears of increasing severity and eventually a condition of marasmus may develop. To this condition the name *milchnährschaden* has been given by Czerny, who believed that an excess of fat in the diet was responsible for it. It is probably due not so much to an excess of fat as to an insufficient amount of carbohydrates. In the absence of this latter, putrefaction of the protein goes on unchecked. This accounts for the character of the stools. It is the insufficient amount of carbohydrates that is chiefly responsible for the symptoms. Many infants may take diluted whole milk without additional carbohydrate and never show such symptoms, but some are rapidly and seriously affected by the absence of carbohydrates.

The condition is readily amenable to treatment. The indications are to diminish the milk if this has been in excess, and to add sugar alone or sugar and some cereal. The mere addition of milk sugar or cane sugar in the quantities usually given may be sufficient. At times, however, even when given in amounts up to the point of tolerance, no improvement is seen. It is then advantageous to give a preparation of maltose in the form of one of the malt soups, with wheat or barley flour in addition. The improvement is seen at once. The stools become acid in reaction, soft and brownish: the general condition shows a distinct amelioration and gain in weight again occurs.

No Gain in Weight without evident Symptoms of Indigestion.—This is sometimes due to too little or too weak food, the child usually manifesting signs of hunger. Occasionally it is due to the fact that the food has been too concentrated or that too much fat has been given. In the latter case it frequently happens that the appetite is much reduced, so that the infant takes perhaps less than half his usual allowance. Too frequent feedings and the practice of constantly coaxing the infant to take more food often produce the same aversion to food. It is much better to offer food only at four-hour intervals and take away the bottle as soon as the child shows that he does not want more.

Modifications in the food to meet the indications afforded by more serious conditions than those here described are considered in the later pages devoted to Difficult Cases of Feeding.

The Apparatus Required for the Preparation of Milk at Home.—This includes a glass graduate, a glass or agate funnel, a cream dipper, a pitcher for mixing food, feeding-bottles, a tall cup for warming the food, and a small ice-box. Other articles needed are milk sugar, rubber nipples, absorbent cotton, bottle-brushes, borax or boric acid, bicarbonate of soda, and an alcohol lamp, an electric stove, or a Bunsen burner. The best style of bottle is that which can be most readily cleaned. The graduated cylindrical bottles with wide mouths are to be preferred. The best nipples are those of plain black rubber, which slip over the neck of the bottle, and are not so thick as to prevent their being turned inside out for cleansing. Those with a long rubber tube going to the bottom of the bottle should not be used. In many places their use is prohibited by law. The hole in the nipple should be large enough for the milk to drop rapidly when the bottle is inverted, but not so large that it will run in a stream. New nipples should be boiled; but the daily boiling of nipples is unnecessary. It soon makes them so soft as to be useless. They should be rinsed in cold water immediately after using and washed daily in soap and water. When not in use, nipples should be kept covered in a solution of borax or boric acid. Bottles should first be rinsed with cold water, then washed with hot soap-suds and a bottle-brush. When not in use they should stand full of water. Before the milk is put into them they should again be placed in boiling water for ten minutes.

Directions for Feeding.—The food should be warmed to about 100° F., best by placing the bottle in a tall pitcher or cup filled with hot water, not by pouring the food from the bottle into a saucepan. The temperature of the food may be tested with a thermometer, or by pouring a few drops upon the front of the wrist; it should feel warm, but not hot. The nurse should never take the nipple of the bottle into her own mouth. A bottle should not be warmed over for a second feeding. A child should not be more than twenty minutes in taking his food, and should not sleep with the nipple of the bottle in his mouth. It is preferable to have a young infant held while taking his bottle. If this is not done, the bottle should at least be held in such a position that the neck of the bottle is kept full. After feeding, the child should be held upright over the nurse's shoulder, and patted on the back, to allow him to bring up the gas, usually air which he has swallowed. He is then placed in his crib and left alone. It is even more necessary than in breast-feeding that rules as to frequency and regularity of meals be observed.

Directions for Preparing the Food.—All the food needed for twenty-four hours should be prepared at one time. The first thing to be decided is the formula to be used; next, the quantity of food for twenty-four hours, lastly the number of feedings into which it is to be divided.

Let us suppose for example that the child to be fed is an average healthy infant three months old, weighing about twelve pounds. Formula No. V of the series given would be an appropriate one to begin with. The food requirements would be furnished in about 35 ounces. This amount should be given in six feedings. When more than 20 ounces is needed for a day's supply the quantity of each ingredient should be increased: for 30 ounces one-half more of each is used; for 35 ounces three-quarters more; for 40 ounces twice as much. Thus, using No. V, the quantities would be as follows:

	For 20 Ounces.	For 30 Ounces.	For 35 Ounces.	For 40 Ounces.
Whole milk	10 oz.	15 oz.	17½ oz.	20 oz.
Sugar	2½ tabl'sp'ls	3¾ tabl'sp'ls	4½ tabl'sp'ls	5 tabl'sp'ls
Water	10 oz.	15 oz.	17½ oz.	20 oz.

When barley water or gruel is used it replaces part or all the water in the formula.

The milk sugar should be dissolved in boiled water, which is then mixed with the milk in a pitcher. The food is now divided into the required number of feedings and the bottles stoppered with cotton. They are placed at once in an ice chest, or first sterilized, then cooled, and afterward placed upon ice.

Milk Laboratories.—Many of our large cities have milk laboratories which put up on the prescription of physicians milk for infant feeding containing any desired percentages of fat, sugar, protein, etc., raw or heated, and with the addition of any cereals when these are wanted. In his prescription the physician indicates simply the percentages he wishes, together with the number of feedings and the quantity for each feeding. The milk is delivered daily in the bottles from which it is to be fed, requiring only to be warmed. The milk laboratory is of much assistance in infant feeding, particularly when there is no one in the home who has the time, the facilities or the intelligence to prepare the food properly there. To one with experience in ordering milk by prescription the milk laboratory is a great practical aid. The laboratories are particularly useful in preparing milk for long journeys or ocean travel.

The Observation of Cases of Infant-Feeding.—Attention to de-

tail is most essential. Much of the want of success in infant feeding is due to a failure of the physician to keep in close touch with the case. For the first few weeks he should see the infant every few days, inspect the stools, hear the nurse's report, and see how directions are being carried out. When the child is well started and has begun to gain regularly in weight, a weekly visit may be sufficient. Still later, monthly visits but with regular weekly reports in writing should be continued until the child is a year old and is taking whole milk and solid food. The weekly report should include answers to certain questions, viz.:

1. Weight: gain or loss since last report.
2. Stools: frequency and general character.
3. Vomiting or regurgitation: when and how much?
4. Flatulence or colic?
5. Appetite: Is the child satisfied? Does he leave any of his food?
6. Is he comfortable and good-natured and sleeping well?
7. The formula of the food now given: quantity and frequency of feedings.

An excellent plan is to furnish the mother with a printed form containing the questions to be filled out and returned. With information regarding the points indicated, it is possible for the physician to know pretty accurately how the child is doing, what changes, if any, are desirable in the food, and whether he ought to see the patient.

It is essential to success with any method of feeding, first, that one should have good raw materials—the freshest and cleanest milk obtainable; second, that at least the fat content of the milk or cream used be definitely known; third, that directions for the mother or nurse be clear, explicit and in writing; fourth, that one have the coöperation of an intelligent mother or nurse; finally, it should be remembered that practical success in infant feeding depends upon how intelligently a method is used, rather than upon the method itself, and that the one indispensable thing is systematic observation.

The Use of other Food than Milk during the First Year.—Reference has already been made to the addition of farinaceous food in the form of barley water and other cereal gruels in the modification of cow's milk. These are useful in the first place for their mechanical effect upon casein coagulation in the stomach. For this purpose only a small amount of the cereal making a weak gruel is necessary, e. g., one or two teaspoonfuls of the flour to the daily food. Farinaceous food may also be given when, because low fats are used from choice or necessity, the carbohydrates should be increased. Instead of doing this entirely by some form of sugar, part of the carbohydrates may in many cases advantageously be furnished in the form of starch. This may be given as a gruel made from wheat, oat, or barley flour, or arrowroot. The amount of the flour used

in the daily food should seldom be over one-fourth ounce under three months of age; from three to six months, from one-half to one and one-half ounces may be given; from six to ten months, from one and one-half to two ounces; all the above being by volume, not weight. The flour should be cooked for ten to twenty minutes in the water used for diluting the milk. If grains instead of flour are used the cooking should be for at least three hours and the gruel should be carefully strained before using. After ten or eleven months cereal may be given with a spoon. This may be almost any form of well-cooked cereal which has been strained. It may be cooked with milk or the milk may be added subsequently. Beginning with an ounce a day the quantity may be gradually increased to two ounces twice a day. While many children easily digest the amounts of starch mentioned, there are others who are much disturbed by them, and some to whom, owing to flatulence and other symptoms of intestinal indigestion, starch can not be given at all.

The only other things to be advised during the first year are beef juice and the juice of some fresh fruit. Beef juice may be begun in the ninth or tenth month, earlier with anemic children; at first not more than two teaspoonfuls daily, later the amount may gradually be increased to one ounce. The best fruit juice is that of the orange, which should be fresh and sweet. It may with advantage be given to all healthy infants eight months old, and to most when six or seven months old. Beginning with half an ounce, the quantity may gradually be increased to two ounces daily, given preferably about one hour before the second milk-feeding.

The Tolerance of Healthy Infants for the Different Food Elements.—In the foregoing pages we have indicated the proportions and amounts which, in our experience, have been shown in the majority of instances to be the best for feeding healthy infants. However, Nature will often tolerate quite wide variations from what is best. The desire for a rapid increase in weight often leads to an increase of the fat in the food much beyond the limits which are usually safe. There are some children of vigorous constitution and strong digestion, living in good surroundings, who tolerate this for a long time; some may even go through infancy to a period of mixed diet without any visible disturbance, and appear to thrive exceedingly well. There are others who bear for a considerable time very high proportions of carbohydrates and show phenomenal gains in weight. In both the conditions mentioned tolerance usually breaks down after a time, often from a trivial cause. This may be some intercurrent illness like a cold or a mild bronchitis, or the advent of very hot weather; or, sometimes even so slight a thing as dentition may bring about an upset of a most alarming character. In other children there gradually develop subacute or chronic disturbances of digestion and

nutrition which may last for months. One should be very cautious, therefore, in inferring that because a few infants thrive on unusual proportions or excessive amounts of some one of the food elements this is to be taken as a guide in feeding the average child.

FEEDING IN DIFFICULT CASES

In the aggregate the number of infants included under the head of "difficult feeding cases" is a large one, and their management constitutes the most "special" branch of Pediatrics. The problem is often one of great complexity, the symptoms presented are of almost endless variety and even one of large experience often finds himself baffled. Let no one, therefore, expect to solve these problems without careful study of the individual cases and the closest attention to detail.

Causes.—In some of these infants difficult feeding is due to feeble digestion or some individual peculiarity because of which they do not thrive, even from the outset, upon the usual milk modifications although used intelligently. In a much larger group the cause is to be found in prolonged disturbances of digestion, the result of previous improper methods of feeding. The difficulties are greatest in early infancy, in cities, in institutions, in hot weather, and they are further increased by the existence of constitutional debility, and when the trouble is of long standing. It is not infrequently found that the failure is due not to any fault with the food prescribed, but to other conditions. The food may be improperly prepared or given—e. g., it may be cold or given too rapidly; the bottles or nipples may be dirty; the proper quantities and intervals not observed, etc. Another factor of importance is the environment as affecting the nervous system of the infant. Among the well-to-do this may be the chief trouble. The constant or frequent excitement by visitors, or playing with a child by parents or nurses, may result not only in lack of sleep, but in disturbances of digestion, often in habitual vomiting, though the food itself is proper. In such circumstances the removal of the child from its surroundings or placing him in charge of a competent nurse will often cause an immediate and marked improvement without any change in the food. Another minor cause of disturbance is the habitual use of the "pacifier," frequently resorted to in these cases, but which should under no conditions be tolerated.

That a prolonged disturbance of digestion in a young infant is a serious thing is often not appreciated. The mother is apt to think the problem one easy of solution; she "only wants to be told what to feed her baby," imagining that a single food prescription should set the child

right at once. The physician too, sometimes, regards the condition lightly because these infants do not seem really ill; he therefore considers the subject hardly important enough for his careful, continuous attention. The fact should be emphasized that these cases are serious, that they are difficult, that in most of them nothing can be accomplished without close and continuous personal observation, that they do not tend to right themselves, and that infants' lives are often sacrificed as a result of bad management.

Clinical Types.—The greater number of these cases may be divided into three groups: (1) those whose chief symptom is habitual vomiting, or regurgitation of food; (2) those with intestinal symptoms, most frequently with loose stools; (3) those without any marked symptoms of indigestion, yet whose weight is much below the average, who do not gain on weak food and are upset if stronger food is used. They have feeble digestion rather than indigestion.

Cases with Vomiting.—The causes producing this are usually rather obvious. When cream and milk mixtures or top-milk mixtures are used, altogether the most frequent mistake is the use of too much fat. The amount used may not be more than many healthy children will take, but it is excessive for the particular patient. It is surprising how great the intolerance to fat is in some of these infants and also when once established how long it persists. Another frequent cause is the use of too much cane sugar, milk sugar, or one of the proprietary foods containing maltose or much starch. Other factors of importance are too frequent feedings, too much food and the use of unsuitable and indigestible foods. The vomiting may also be the result of a neuropathic constitution. (Page 262). The condition may be a sequel of any of the acute infections and is more intractable in the course of a severe constitutional disease such as rickets, syphilis or tuberculosis.

With such severe and prolonged symptoms as are often present, pathological changes in the stomach might be expected. These, however, are strikingly absent. The stomach may be slightly dilated and there is usually a large amount of mucus present but macroscopically and even microscopically there are no important or even constant changes.

The most important symptom is vomiting. It may occur soon or long after feeding. Some of these infants vomit only occasionally and in large quantities; but it is more common for frequent regurgitation of small amounts of food to take place. This may begin soon after one feeding and continue quite to the time for the next. After a time, the vomited matters nearly always contain mucus, and sometimes this is a conspicuous feature. The regurgitation of a sour irritating fluid occurs even when but little food is ejected, and usually accompanies the belching of gas.

The results obtained in the examination of stomach contents have not been uniform, and in practice one should not lay much stress upon the absence of the normal secretions. The presence of mucus in the vomited matters or in the washings from the stomach is nearly a constant feature. This greatly interferes with digestion, even though the secretions are normal. The reaction of the stomach is almost always acid. The hydrochloric acid is almost invariably diminished in quantity. Free hydrochloric acid is very seldom present. There is usually a marked odor of butyric and other volatile fatty acids. One would expect, therefore, to find these in excess, but the studies of Huldschinsky have shown that they are little if at all increased in the stomach contents of vomiting infants. The rennet ferment and pepsin are almost invariably present in normal amount, hence the administration of digestive ferments is not indicated.

In addition to air which is swallowed, there is an increased production of gas. Some of the most striking symptoms are due to distention. The epigastrium may be tense and hard most of the time, and often so much gas is present that infants find difficulty in taking food. Though evidently hungry, they can take so little at a time that an hour or more may be required to take four or five ounces. There is motor insufficiency of the stomach and probably in some cases a certain degree of pyloric spasm which causes gastric stagnation. That the food remains long in the stomach is best demonstrated by aspiration or stomach-washing. Instead of the stomach's being empty in two and a half or three hours, as it should be, food may be found four or five hours, and in some cases six or eight hours, after feeding. There may be dilatation of the stomach, especially in older infants who are rachitic.

The appetite may be abnormally great, or it may be poor. As a rule, children take less food than in health. The tongue is usually coated. The general symptoms are those of malnutrition; there is constant fretfulness, and sleep is irregular or disturbed; the weight is stationary, or there is a steady loss; there is also anemia, and the child's development is arrested. There is nearly always some derangement of the bowels, more often constipation than diarrhea.

Infants who vomit as the result of a neuropathic constitution may show at first no symptoms but the vomiting. If this is severe and continued, later they show evidences of malnutrition, sometimes of an extreme grade.

There is little tendency to spontaneous improvement or recovery, the prognosis depending almost entirely upon the treatment employed. Unless relieved the condition is apt to continue, until some serious acute disease develops which may be fatal. In very young infants

such gastric disturbances should not be confounded with hypertrophic stenosis of the pylorus.

In the treatment, the question of diet is of first importance. It is the chief therapeutic measure. The indications for varying the quality and quantity of the food when there is habitual vomiting have already been discussed (page 193). The feedings should be at least four hours apart and the amounts smaller than normal infants of the same age would receive. The usual practice when an infant suffers from vomiting is to dilute his food and, in some instances, this is perfectly proper; but to continue increasing the dilution because the patient does not do well may be the very worst treatment. Small feedings, not weak food, are what benefit some of these children most, the balance of the daily amount of water needed by the infant being given between the feedings. Unless cream or top-milk mixtures have been employed the sugar is more likely to be the exciting cause of the vomiting than any other ingredient of the food. This should be greatly reduced in amount or temporarily removed altogether. When the vomiting has ceased the sugar may gradually be increased. Milk sugar is less likely to ferment in the stomach than cane sugar or maltose. The latter should never be used with vomiting infants. Buttermilk, on account of its low fat and moderate sugar content, is frequently of value, but it cannot advantageously be continued very long without the addition of carbohydrates in some form. The very factors that make it of value for temporary use make it disadvantageous for permanent use.

Wet nursing does not bring immediate improvement in the vomiting and sometimes none at all. The large amount of sugar and fat in breast milk sometimes aggravates the symptoms. Usually, however, the infant when breast-fed improves; but the vomiting may continue so severe as to make it necessary to return to artificial feeding. When the vomiting has ceased, however, nothing brings about such rapid recuperation of the general health as does breast milk.

At times, nothing succeeds so well as giving semi-solid food with the spoon. Cereals cooked with milk as described on page 263 are readily borne by many infants, especially those with vomiting due to nervous causes.

Stomach washing is frequently useful, especially with persistent cases. It removes the mucus, cleanses the organ and acts as a stimulant to the gastric secretions, especially the hydrochloric acid. Plain boiled water, or a weak alkaline solution—sodium bicarbonate, one dram to the pint —may be employed. In the early part of the treatment the washing should be done daily; later, every second or third day. The time selected is not of great moment, but it is better to make this about three hours after feeding.

The general treatment is apt to be ignored, but is important. The best possible hygiene should be secured,—a large, roomy nursery, and plenty of fresh air by night and by day; equally necessary are quiet surroundings and freedom from disturbing conditions which sometimes obtain in the nursery. General friction of the body is useful in delicate children with poor circulation. Infants must be properly covered, and it is of the utmost importance that the feet be kept warm.

Drugs have a very limited application in the treatment of this condition in infants. They have been too much used, and too little attention has been given to the details of feeding, by which means alone permanent improvement is usually reached. The continued use of pepsin and other digestive ferments is irrational and without benefit. Hydrochloric acid may at times prove of value, but it must be given in rather large doses,—i. e., five to fifteen drops of the dilute acid after each feeding.

Cases with Intestinal Symptoms.—These are found most frequently in infants born prematurely, in those with constitutional debility, who have never been vigorous, in those brought up in poor surroundings with unintelligent care or in those who have suffered from any acute disease, especially inflammation of the gastro-intestinal tract, such as ileocolitis. Usually there has been artificial feeding from the beginning or after a few weeks of nursing. Some of the infants also belong to the neuropathic type. To the extent that it is usually avoided by maternal nursing, the condition is a preventable one. But there are a few infants that develop these symptoms even while nursing; and a considerable number, in spite of intelligent artificial feeding. There seems to be with these infants a particular lack of resistance on the part of the intestinal tract. It never seems capable of accomplishing the work devolving upon it.

In infants fed on top-milk mixtures, the most common cause of disturbance is an excessive amount of fat. When whole-milk modifications are used the fault is usually an excess of sugar, and with older infants too large quantities of farinaceous foods, often insufficiently cooked. The carbohydrates may not be more than the average child takes well, but these infants are particularly sensitive.

There are no constant or characteristic pathological changes. There may be a hyperplasia of the lymphoid tissue of the intestines and sometimes there is a similar process in the mesenteric lymph nodes. Usually, however, these are absent.

The symptoms are general and local. So far as the intestinal condition is concerned, diarrhea is the most frequent and serious symptom. It may happen that the same child will suffer for a long time from diarrhea and then from constipation, but the constipation is usually the result of dietetic measures directed against the diarrhea,—i. e., a

reduction in the fat or the carbohydrates, or both. As a result, the energy value of the food is reduced to a point at or below the maintenance requirement. When, in order to produce gain in weight, these substances are increased in the food, diarrhea again results. There may thus be over long periods, alternating constipation and diarrhea. The stools are of all varieties, depending on the severity of the symptoms and the character of the food. They are usually more frequent than normal and generally contain undigested food and mucus. In some cases the stools contain but little solid matter, the character being that of yellowish-green water. The stools usually have a sour, unpleasant odor, but are rarely very foul. They may be irritating to the skin and cause troublesome excoriations and intertrigo. There may be much gas and flatulence.

If there is constipation, the stools are usually gray or white; they are smooth and pasty like hard balls and passed after much straining, often coated with mucus and sometimes streaked with blood. Such stools are not infrequently seen when the food contains a large amount of fat. With the constipation there may be much flatulence and colic, the attacks of which may be severe.

The general symptoms are those of malnutrition. These are more fully described elsewhere and need only be mentioned here. The most important are: stationary or falling weight, anemia, poor circulation, often subnormal temperature, almost constant fretfulness and crying, with very little quiet sleep. The tongue may be coated but more often is quite clean. The appetite is frequently good, these infants taking food whenever given, and in an almost unlimited quantity. There are few cases in which occasional vomiting does not occur, sometimes it is marked and persistent, but it is rare for it to be so. When so much of the food is regurgitated by vomiting, as in the cases just described, the intestinal tract, even with highly erroneous methods of feeding, is thereby protected.

The duration of these symptoms is indefinite. Even with the greatest care there is little or no tendency to spontaneous improvement. They may drag on for many months with frequent exacerbations and remissions. The symptoms may be relieved, but at the same time to insure growth and a gain in weight may be, for the time being at least, well nigh impossible. The least increase in the food, especially the carbohydrates or fats, may be sufficient to precipitate an attack of diarrhea with further loss in weight. Thus, there may alternate slight gains and losses, the weight for months being nearly stationary.

A danger to these patients is that of intercurrent infections. To a delicate infant an attack of rhinopharyngitis with otitis may be more serious than a frank pneumonia to a vigorous child. Any infection is

to be feared, bronchitis and pneumonia particularly so. Death seldom results from the severity of the condition itself. With appropriate treatment a gain in weight usually results, although this may be delayed many weeks or months. With infants over six months of age the problem is usually an easier one than with those younger. Especially is there difficulty with premature infants and those much under weight at birth, i. e., five pounds or less.

Drugs have no part in the treatment of these cases; in nearly every instance they had best be omitted altogether. The treatment is dietetic. Prophylaxis is important. Maternal nursing will do much to prevent the development of such cases. It is necessary to obtain a careful and minute history in order to direct matters intelligently. The previous feeding should be thoroughly known, the different changes made and their effect upon the intestinal symptoms and the infant's weight. With this information one can often at once determine where mistakes have been made and in many instances it is found that the same mistake has been repeated with each change of food.

Occasionally diarrhea develops with maternal nursing and it is by no means infrequent when, on account of a tendency to attacks of diarrhea, wet nursing is resorted to. The cause of this is the large amount of fat and sugar in breast milk, both of which readily undergo change in the intestines with the production of irritating lower fatty acids. Breast feeding should not be interrupted under such circumstances but supplementary feeding with a food low in fat and sugar should be resorted to. The most available food is buttermilk. This may be given at alternate feedings or may be given in amounts of one or two ounces just before the nursing. When the symptoms have been overcome, the buttermilk may gradually be withdrawn from the dietary. Breast feeding is altogether the safest method of treating such conditions in those infants under three months of age. Many of those older may be successfully treated by artificial feeding but progress, in order to be sure, must be slow. In protracted cases minor variations in the composition of the food or in the plan of feeding rarely accomplish much. The most brilliant results are often obtained from as complete a change in the diet as possible. Notwithstanding the fact that these patients are usually much below the normal weight and often losing steadily, the treatment should be directed first of all to allaying the most marked intestinal symptoms. Until these are relieved, no permanent improvement can be expected. For the time being, the weight must be disregarded.

So far as the elements of cow's milk are concerned, the greatest difficulty is seen when both fat and sugar are given in considerable amount. A moderate amount of fat with a minimum of sugar usually

causes no diarrhea. Sugar, however, even in the absence of fat, will produce it. For this reason, the use of skimmed milk and even fat-free milk usually causes no improvement in the diarrhea, there being too much sugar in fresh milk, even without the addition of any extra amount. Top-milk or milk and cream mixtures are not admissible. If fresh milk mixtures are to be used, dilutions of whole milk or of partially skimmed milk should be given, with no carbohydrates added. If, upon this diet the stools become normal, sugar may gradually be added, but this must not be lactose or a mixture in which maltose is present in a large amount, such as malt soup. The dry preparations of maltose or cane sugar should be at first tried and in small quantity, not over one teaspoonful daily.

If fresh milk mixtures are not well borne, buttermilk and other fermented milks may be tried. These succeed in a certain number of cases that do not respond to skimmed milk. It is seldom necessary to dilute them more than with an equal amount of water. Additional carbohydrates needed may, after a time, be supplied—best by adding starchy food with small quantities of cane sugar.

Protein milk is one of the most valuable of the recent additions to our resources in feeding cases of this type. The chief advantages here are apparently due to its low sugar content, for it contains a considerable amount of fat, indicating that fat in the absence of carbohydrates is very frequently well borne. The large amount of protein which readily undergoes putrefaction inhibits the formation of the lower fatty acids from the carbohydrates and fats. Only for very young infants need it be diluted and it is seldom necessary to reduce the fat by making it from skimmed milk. Not much gain in weight is seen when protein milk is used alone. Carbohydrates should be added as soon as possible, but always with great caution, beginning with very small quantities. Cane sugar should first be tried, then one of the dry preparations of maltose beginning with not more than a half tablespoonful daily and slowly increasing.

Either of these sugars may be used in conjunction with starchy food which may be wheat or barley flour from one-quarter to one ounce daily, the latter amount to children five or six months of age. Employed in this way protein milk may often be continued for two or three months, but without the addition of carbohydrates it is seldom advantageous for more than two or three weeks.

Peptonized milk has been altogether too frequently employed and offers no aid in the treatment of intestinal conditions. A change to a diet other than milk should be made very slowly and with great care; one relatively rich in carbohydrates is usually badly borne. Carbohydrates in the form of cooked cereals must be added gradually. Eggs

are sometimes of assistance and junket is frequently of value in preventing excessive fermentation. Solicitous care should not cease with these children at the end of the first year, they must be closely watched until they are three or four years old.

The same careful hygiene is as important as in patients with gastric symptoms. The general methods employed should be the same.

Cases with Feeble Digestion.—Infants whose digestion is very feeble, although they have neither pronounced gastric or intestinal symptoms, are very difficult patients to feed. Gains in weight are very slow and one must be content if any regular gain takes place. In case of failure by the usual milk modifications, wet-nursing is altogether the most successful form of feeding. Sometimes it is sufficient if only partial breast feeding can be given, i. e., three or four feedings a day. This is a plan of much value in institutions and saves many babies. If no breast feeding is possible, artificial feeding must be conducted in the most painstaking manner lest serious digestive upsets occur. If these can be avoided it usually happens that as the child grows older and a more varied diet can be given, the problem grows steadily easier.

With some infants, in the event of failure by the usual methods, the first start, which is really the most difficult one, may be made upon sweetened condensed milk which is diluted with plain water or barley water. "Evaporated" or unsweetened condensed milk has at times succeeded when fresh whole milk has failed. The explanation for this cannot be given. Unsweetened condensed milk requires the same addition of sugar and starch as does whole milk.

When there is no vomiting and no tendency to diarrhea, feeding with considerably higher proportions of carbohydrates than are usually employed is also sometimes useful for a short time. It may be carried out with fresh milk as in the various malt-soup mixtures, or with sweetened condensed milk or evaporated milk as a basis. When an excess of carbohydrate is given the percentages of fat and protein, but especially the former, should be lower than in the usual formulas for the age and condition. There is apparently some advantage in using a variety of sugars; a combination of lactose, maltose and cane sugar being given rather than any one of them alone. The total sugar may sometimes be carried above 7 per cent but always with caution. Starchy food is added in the form of barley, wheat or oat flour, cooked for ten to twenty minutes. The daily quantity used may be from half an ounce to two ounces according to age and condition. The larger quantity mentioned may sometimes be given to an infant of five or six months. With infants over six months of age thick gruel like that advised for normal infants of ten or twelve months may be of great assistance in causing gain in weight.

A diet containing an excessive amount of carbohydrate is not adapted to prolonged use and incautiously used may be followed by serious upsets. For a time all may go well; then from some apparently trivial cause a breakdown occurs. As soon as possible the child should be placed upon a more rational food, i. e., a properly "balanced ration" by introducing at first one and then other feedings from whole milk modifications in which fat and protein are raised and carbohydrates reduced.

A foodstuff occasionally useful is olive oil. It is a form of fat which can sometimes be tolerated when the fat of cow's milk habitually disagrees. The amount used at first should be small, not more than one-half teaspoonful twice a day. The maximum amount to be used for infants of the first year should not be over two teaspoonfuls daily.

The chief means by which weight can be increased in children suffering from malnutrition is therefore through the addition of carbohydrates, especially maltose, as soon as these can be tolerated; next by the addition of fat, but neither of these is to be employed in any considerable quantity until the marked symptoms of indigestion have been controlled.

CHAPTER IV

FEEDING AFTER THE FIRST YEAR

HEALTHY INFANTS DURING THE SECOND YEAR

The physician should not relax his vigilance in the feeding of a child after the first year has passed. The ideas of the laity in regard to what is proper for a child after he has outgrown an exclusive milk diet are very erroneous. Most of the disorders of digestion of early childhood are directly traceable to dietetic errors. Among the poor the majority of infants are given solid food too early, in too large quantities and improperly prepared. Among many of the intelligent and well-to-do the disposition is to go to the opposite extreme and to keep the infant too long upon a diet composed exclusively or almost exclusively of milk.

During the second year the diet of a healthy child should consist chiefly of milk, bread, farinaceous foods, fruit juices or cooked fruit, with a small amount of animal food in the form of beef juice, broths, meat and eggs. By the middle of the year with most children, with some even earlier, potato may be added, also green vegetables, at first in small quantities, thoroughly cooked and puréed.

Milk should be the largest item of the diet, but when solid food in any considerable quantity is begun it should be reduced; few children

require more than a pint and a half of milk a day. The popular notion that there are many children who cannot take milk is an erroneous one; the real trouble usually is that too rich milk is given or that the quantity allowed is too large. It is often drunk like water with a hearty meal of other food and the child is simply overfed. On the other hand, to permit a child to give up milk altogether because solid food pleases the palate better is a mistake. It is important, however, that the transition from an entirely fluid diet to one of solid food should be made gradually, and that the habit of taking milk should not cease at the end of the first, or even the second year.

During the second year with average milk and average infants no modification of the milk is required. If the milk is very rich, such as that from a Jersey herd, it should be partially skimmed or diluted with at least one-fourth water. In hot weather especially should these measures be insisted on.

Weaning from the Bottle.—This should always be begun before a child is a year old; by the thirteenth month an infant should take all his milk from a cup, except possibly the 10 P. M. feeding, when for the sake of convenience the bottle may be allowed. Early weaning from the bottle is a matter of no small importance. When the bottle is allowed to older children the temptation to overfeeding, especially during the summer, may be very great. Again there are many children with the "bottle-habit" so firmly developed that throughout childhood, although at any time they will take milk from the bottle, they can never be induced to take it any other way, and sometimes refuse all other food so long as the bottle is allowed.

From Twelve to Fifteen Months.—The daily schedule at this period should be about as follows:

6 to 7 A.M. Milk, six ounces, diluted with two to three ounces of barley or oat gruel.
9 A.M. Orange juice, one to three ounces.
10 A.M. Cereal (thoroughly cooked and strained), one large tablespoonful.
Milk, six ounces, part of it on cereal.
Crisp, dry toast, one piece.
2 P.M. Beef juice, one to two ounces;
or, mutton or chicken broth, three to four ounces;
or, one-half, later one entire soft egg.
Crisp, dry toast or unsweetened zwieback, one piece.
Milk and gruel in the proportions given above, four to six ounces.
6 P.M. Same as at 10 A.M.
10 P.M. Same as at 6 A.M., but given from a bottle.

In preparing the food, the milk and the gruel are simply mixed together while the latter is warm; salt and at first a very small quantity

of cane sugar are added to make it palatable. It is then divided into as many feedings as are required for the day, each one being placed in a separate bottle. As to handling the bottles and pasteurizing or sterilizing, the same rules apply as during the first year.

From Fifteen to Twenty Months.—The diet may be increased by the addition of more solid food. The average child will require:

6.30 A.M. Milk, warmed, eight ounces.
9 A.M. Orange juice, two to three ounces.
10 A.M. Cereal, two good tablespoonfuls, oatmeal or hominy, cooked three hours, not strained, with one ounce of thin cream or top-milk.
Milk, six ounces.
Crisp toast or zwieback, one or two pieces.
2 P.M. Beef juice and two teaspoonfuls of scraped meat;
or, broth, four ounces, and one egg.
One-half of a baked potato;
or, one tablespoonful of a green vegetable (spinach, carrots, fresh peas, string beans, asparagus tips) thoroughly cooked and put through a fine sieve.
Stewed prunes, three or four;
or, one-half a baked apple, strained.
Crisp toast or dried bread.
6 P.M. Cereal, two tablespoonfuls, farina or cream of wheat, cooked one hour, served as at 10 A.M.
Milk, eight ounces.
10 P.M. Milk, six ounces (omitted at eighteen months and sometimes earlier).

From Twenty Months to Two Years.—By the end of the second year the amount of the solid food, especially the quantity of meat and vegetables, may be somewhat increased. The meat allowed may be finely minced or scraped beefsteak, lamb chop or chicken. Only four meals should be given, the 10 P. M. feeding being omitted, and nothing but water between the feedings; this, however, should be allowed freely. Raw fruit except orange juice should not be given. It is usually better to give the fruit and milk at different meals. It is often more convenient to transpose the morning feedings, giving the milk at 10.30 and the principal meal at 7.00 or 7.30 A. M.

FEEDING FROM THE THIRD TO THE SIXTH YEAR

Articles Allowed.—From the following list the diet of a healthy child may be arranged.

Milk.—This should form a prominent part of the diet. No child requires more than a pint and a half (three glasses) daily. Rich Jersey milk should not be chosen. The milk should usually be given warm.

Cream.—Not more than three or four ounces of thin (16 or 20 per cent) cream should be given daily. It should not be used upon fruits, especially sour fruits. It may be used upon cereals, upon potato and in broths. Cream should not be given at all to children who suffer from so-called bilious attacks, with coated tongue, bad breath, etc.

Eggs.—They should be fresh, soft-boiled, poached, coddled, or scrambled, but not fried. Children vary greatly as regards their ability to digest eggs; many children will take two eggs a day, some only one, and a few can not take them at all.

Meats.—Some form of meat should be given once a day. The best are beefsteak, lamb chop, and roast beef or lamb and the white meat of chicken; next to these certain of the more delicate kinds of fresh fish, which should be boiled or broiled. Beef and lamb should be given rare. All meat should be very finely divided. Cold meat should be avoided.

Vegetables.—Potato may be given once a day, baked or mashed, with the addition of cream or beef juice rather than butter. Of the vegetables the best are asparagus tips, spinach, stewed celery, string beans, carrots, and fresh peas. One of these vegetables should be given daily—always well cooked and mashed.

Cereals.—None of the "dry" or ready-to-serve cereals should be given

CALORIC VALUES OF COMMON FOODS

Cereals (cooked).	Ounce.	Even Tablesp'l
Oatmeal	18	10
Hominy	22	10
Farina	16	7
Rice	32	17
Macaroni	26	15
Vegetables.		
Lima beans	22	13
Peas	33	16
String beans	6	3
Carrots	17	10
Spinach	16	10
Squash	14	9
Mashed potato	33	17
Sweet potato	58	30
Butter	225	114
Dry cocoa	146	38
Sugar (cane)	116	32
Lean roast beef, lamb or chicken	50	22
Fish	32	16

	Ounce.
Beef juice	6
Broth	5
Cream Soup	30
Custard	40
Orange juice	15
Milk	20
Cream (20%)	60
Condensed milk	100
Protein milk	15
One slice of bread, 4 x 4 x ½ in	80
One large roll	100
One soda cracker	25
One egg (yolk 60)	75
One medium potato	100
One medium apple	75
One fig	50
Four prunes	100
One large banana	100

In ordering a diet for children a knowledge of the nutritive or caloric value of the different common articles of diet is highly desirable.

As estimated in the table, vegetables are finely mashed, meats are finely divided and even tablespoonfuls are tightly packed.

Cereals are cooked in water in proportions given on the package, i. e., oatmeal one cup to water one pint.

to young children. They are the cause of more disturbances of digestion than almost any other common article of diet. Almost any cereal which has been thoroughly cooked may be allowed—oatmeal, wheaten grits, hominy, rice, cornmeal, farina, and arrowroot. If the grains are used, cereals should be cooked from three to six hours, after having been previously soaked. The partially cooked cereals of the shops should always be cooked two or three times as long as the directions upon the package. The "fireless cooker" is an excellent device for the proper cooking of cereals for children. Cereals should always be well salted, and given with milk or cream, but with little or no sugar.

Broths and Soups.—Both meat and vegetable soups may be given and nearly all varieties of the latter except tomato soup. Plain broths are not very nutritious, but when thickened with arrowroot or cornstarch, and when milk is added, they are very palatable, and at the same time a valuable addition to the diet. Most vegetable purées are useful, and when properly made very digestible. Beef juice may be used as directed for the second year.

Bread and Biscuits (Crackers).—In some form these may be given with nearly every meal, better without butter until the third year. All varieties of bread may be allowed when stale—i. e., two or three days old; also dried bread, zwieback, and oatmeal or gluten crackers.

Desserts.—The only ones besides cooked fruits that should be allowed up to the sixth year are junket, plain custard, rice pudding without raisins, and, not oftener than once a week, ice cream. Of the last three, the quantity given should be very moderate.

Fruits.—Some fruit should be given to most healthy children every day. Oranges, baked apples, and stewed prunes are the most to be depended upon. Raw apples should not be given in most cases. Peaches, pears, and grapes (with seeds removed) may be given when thoroughly ripe and fresh, but only in moderate quantity. The pulpy fruits should be given to young children only when cooked. Much indigestion is produced by too much fruit or improper fruits. Special care should be exercised in the use of fruits in very hot weather, and in cities where they may not always be fresh. The juice of fresh berries may be given in the second year; but the whole fruit should be very sparingly given to all young children, and always without cream.

Articles Forbidden.—The following articles should not be allowed children under four years of age, and with few exceptions they may be withheld with advantage up to the seventh year:

Meats.—Ham, sausage, pork in all forms, salt fish, corned beef, dried beef, goose, duck, game, kidney, liver, meat stews, meat dressings and cold meats.

Vegetables.—Fried vegetables of all varieties, cabbage, raw or fried

onions, raw celery, radishes, lettuce, cucumbers, tomatoes (raw or cooked), beets (unless they are very small and quite fresh), egg-plant, and green corn.

Bread and Cake.—All hot bread and rolls; buckwheat and all other griddle cakes; all fresh sweet cakes, particularly those containing dried fruits and those heavily iced.

Desserts.—All nuts, candies, pies, tarts, and pastry of every description; also all salads, jellies, syrups, and preserves.

Drinks.—Tea, coffee, wine, beer, cider, and soda water.

Fruits.—All dried fruits; bananas, unless baked; all fruits out of season and stale fruits, particularly in summer.

From the third to the sixth year four meals should usually be given daily and at regular intervals—e. g., 7 and 10.30 A.M.; 1.30 and 6 P.M. The second meal should be a small one.

There are a few simple rules in feeding which should always be followed: A child should be taught to eat slowly and thoroughly masticate his food. The food must always be very finely divided, for mastication is very imperfect even up to the sixth or seventh year. It is unwise continually to urge children to eat when they are disinclined to do so at the regular hours of meals, or when the appetite is habitually poor, and in no circumstances should children be forced to eat. Indigestible articles of food should not be given to tempt the appetite when ordinary simple food is refused. Food should not be allowed between meals when it is habitually declined at meal-time. If a child refuses to eat, and examination reveals no fault with the food prepared, it should seldom be offered again until the next feeding time. In all cases of temporary indisposition, no matter of what nature, and during periods of excessive heat in summer, the amount of solid food should be reduced and more water given. If milk is the food, it should be diluted.

FEEDING DURING ACUTE ILLNESS

Infants.—Feeding is an important part of the treatment of every acute disease in childhood, but especially so in infancy. Unless the illness is due to disease of the digestive tract, all cases must be fed in about the same way. It is much easier by proper feeding to prevent disturbances of digestion than to allay them. In infancy this complication often turns the scale against the patient. In every severe acute illness, especially if it is of a febrile character, the power of digestion is much diminished. One evidence of this is the onset with vomiting; another is the anorexia which accompanies the early stage of nearly all acute diseases. We should respect this disinclination and make it our guide in the treat-

ment. But water is needed; withholding this will often cause the temperature to rise even higher than before.

In all acute febrile diseases the general rule should be, less food and more water than in health. For bottle-fed infants this is easily accomplished by simply increasing the dilution of the food; for nursing infants by making the nursing time shorter and giving water freely between feedings either from a spoon or bottle. During febrile conditions, fat, especially, is badly borne, and this should therefore be reduced more than the other elements of the food. The diet should consist largely of carbohydrates.

Regularity in feeding is too often entirely ignored. While it is true that with some capricious children all rules must be disregarded, it is with the great majority a decided advantage to adhere to proper food and regular intervals. Food should never be given at less than three-hour intervals, although there is no limit to the frequency with which water may be given, and unless the stomach is irritable, almost no limit as to quantity. Stimulants, when required, are often best given in a very dilute form with the water.

Forced Feeding—Gavage.—Not a few cases, however, are seen in which, after a child has been several days sick, in consequence of delirium, stupor, sepsis, or some other serious condition, he may refuse all food or take so little that he is in danger of death from inanition. At this juncture forced feeding or gavage serves an excellent purpose. Both food and stimulants can thus be introduced at regular intervals with slight disturbance, and lives saved which would otherwise be lost. If gavage is employed, the stomach should be washed at least twice a day. The intervals of feeding should be made at least one hour longer than is customary in health. Forced feeding is not applicable to chronic conditions.

Older Children.—The same conditions with reference to digestion exist as in the case of infants. Older patients, however, are not so easily disturbed, and the disturbance of digestion is not so likely to be serious as in the case of infants. Even here the physician should direct the food to be given at regular intervals, not oftener than every three hours, and should never—as is so often done—order that milk be given the child every time he asks for a drink. In most cases, for children under five years old, milk should be somewhat diluted. Children who do not take milk readily may be given beef tea, broth, gruel, thin custard, or kumyss, and occasionally plain ice cream, but this, if given in any considerable quantity or very often, is likely to disturb the stomach and take away what little desire for food the child may have. Raw eggs are palatable when beaten up with a little sherry, sugar, and cracked ice. Fruits, especially orange and grape juice, may be allowed in almost every febrile disease, but not given within two hours of a milk feeding.

The water given may be plain boiled water, but often better, are some of the carbonated waters, Vichy, Seltzer, or Apollinaris, these being less likely to disturb the stomach.

It is certainly a mistake to force food upon older children in any disease in which their condition is not dangerous. But when there is sepsis, delirium, or coma associated with other dangerous symptoms, gavage may be resorted to with but little more difficulty, and with no less satisfactory results, than in infants.

IDIOSYNCRASIES TO FOODSTUFFS

It is only in recent years that there has been demonstrated an idiosyncrasy on the part of some children to certain foodstuffs, in all probability to the protein of the foodstuffs and to this alone.

The most conspicuous example is the proteins of egg. Some children are so sensitive to egg proteins that the most minute quantity taken internally or even applied locally to an abraded skin will produce the most marked symptoms. The local symptoms, if taken by mouth, are a burning sensation of the mucous membranes followed by marked congestion and swelling, which is sometimes so severe as to suggest that an irritant poison has been swallowed. The general symptoms which follow almost immediately include persistent vomiting, profuse diarrhea and marked prostration. These are often threatening and may be serious, although they usually last but a few hours. With these severe cases a marked eosinophilia is often present. Not only may there be symptoms referable to the gastro-intestinal tract but sometimes dyspnea which resembles an attack of spasmodic asthma. The above symptoms represent the more severe form of this susceptibility. There is a much larger number of children who show this sensitiveness in a milder form, often only by repeated attacks of vomiting after the ingestion of egg. Such a susceptibility is frequently lost during childhood but may persist to adult life to such a degree that the most minute quantity of egg taken in any form whatever is immediately followed by a disturbance.

Very much less frequently similar symptoms may follow the ingestion of cow's milk. We have seen two infants in whom less than ten drops of fresh cow's milk produced symptoms of a severe form. Such a condition, however, is extremely rare, and to attribute to milk idiosyncrasy the common disturbances incident to artificial feeding is quite improper.

In older children a similar sensitiveness is seen to certain cereals, particularly to oatmeal and buckwheat, and also to certain nuts, especially almonds and walnuts. In still others an extraordinary sensitiveness to fruits, usually raw fruits, is seen. The most frequent example is the

familiar susceptibility to strawberries, less often it may be to raw apples. In most cases the disturbance amounts only to an attack of urticaria. Occasionally in the more susceptible the digestive disturbances above noted are also present. Similarly, but less frequently, other raw fruits, grape fruit, oranges and bananas, may cause symptoms. Certain shell fish, such as crabs, oysters, etc., may produce similar symptoms.

From clinical observation alone many erroneous conclusions are apt to be drawn. The absolute proof of such sensitiveness, as has been indicated, is afforded by the cutaneous reaction which follows the application of the protein of any of the substances mentioned. The application of such tests is a matter of somewhat difficult technic and its use is only possible in the hands of a trained observer.

ACIDOSIS

For the preservation of health it is necessary that the body should always contain an excess of bases, in order to maintain that degree of alkalinity in the fluids of the body with which the various functions are carried on to the best advantage. This degree of alkalinity is maintained under normal conditions with wonderful constancy even though there is a continuous elaboration of acids such as sulphuric, phosphoric and carbonic in the organism. The acids are neutralized and removed from the body by a three-fold mechanism:

1. Carbon dioxid is given off from the lungs.

2. The kidneys are able to excrete an acid urine from a slightly alkaline blood. The alkali spared is available to neutralize more acid or to assist in the renewal of the alkali reserve of the body.

3. Ammonia is formed which is capable of neutralizing acid. The ammonia is formed at the expense of urea, a neutral substance, and thus represents a clear gain of alkali for the body.

There is a normal preponderance of alkali over acid in the fluids of the organism. This depends upon the maintenance of an alkaline reserve, very largely bicarbonates, which is found in the blood, tissue juices and cells of the body. So long as the eliminating mechanism for the excretion of acids is preserved the alkaline reserve is not affected, even though the production of acids may be greatly increased. When acids are produced in excess or their elimination is interfered with, the normal preponderance of bases over acids is disturbed and acidosis results.

It is apparent that acids such as those of the acetone series may be formed in the body in considerable amount and yet be excreted without affecting the alkaline reserve. The acids are neutralized by alkalies that can be replaced by those of the food or by ammonia. Under such conditions there is no acidosis. When the production of acids is so great that

they cannot be neutralized without diminishing the reserve of alkali, acidosis may be said to be present. The dividing line between the two conditions is a very narrow one.

Acidosis may result from the production in excess of acids that are present in small amount in normal metabolism, such as aceto-acetic and β oxybutyric acids. These acids are not directly poisonous but produce their injurious effect by depriving the body of alkali. They are present chiefly in diabetes and cyclic vomiting.

Acidosis presumably may result from the failure to excrete acids formed only in normal amount. It is conceivable, but not yet proven, that acidosis may result from the abnormal loss of bases.

The means for the detection of acidosis are chiefly those that determine a diminution in the bicarbonate or in the total alkaline reserve of the blood, among which may be included the determination of the carbon dioxid in the alveolar air, a change in the reaction of the blood, the presence of acids (such as those of the acetone series) in excess in the blood or urine, an increased tolerance for alkalies, or an increased ammonia in the urine which is evidence of the attempt to neutralize an excess of acids.

The methods of treatment are discussed under those diseases in which acidosis is found.

CHAPTER V

THE DERANGEMENTS OF NUTRITION

Inanition—Marasmus—Malnutrition

The derangements of nutrition, especially those accompanied by a loss of weight, form a distinct and a very large class in the ailments of infancy, particularly during the first year. The symptoms are often definite and characteristic, and for this reason have frequently been considered and discussed as separate diseases. They are rather the result of several different factors and usually represent terminal stages of functional or organic disease. In adults such symptoms are usually seen in connection with organic disease. These cases are often very puzzling, and in a large number of them a diagnosis of some constitutional disease, such as hereditary syphilis, or tuberculosis, or organic disease of the stomach or intestines, is erroneously made. The essential condition in all these cases is the inability of the infant to get from his food what his system needs. He can not digest or assimilate enough to support life. He is unable to replace from his food the daily waste of

his tissues. The constructive metabolism is imperfect; the process is, therefore, essentially one of starvation, which may be rapid or slow according to circumstances.

The fault in these cases may be with the constitution of the child, with the organs of digestion, or, what is more generally the case, with the food. The problem is to adapt the food to the individual child under consideration. The solution is often very easy at first, but the difficulties multiply rapidly the longer the condition has lasted. It is therefore essential that the true explanation of the symptoms should be recognized at the earliest possible moment. Changes occur so rapidly in very young infants that a mistake in diagnosis and a consequent delay of a few days may be sufficient to determine a fatal result. The outcome in cases of imperfect nutrition depends almost entirely upon their management. The condition is not one which tends to right itself. Spontaneous improvement or recovery rarely takes place. Not only is careful observation of the child and his symptoms important but also close attention to the body weight. A child whose nutrition is a matter of difficulty should be weighed regularly, in the early months at least twice a week, and once a week throughout the first year. If this is done, the first signs of failing nutrition are unerringly detected. If an infant does not gain in weight something is wrong; a steady loss in weight is a warning which should never pass unheeded; for, unless the conditions are changed, it is practically certain to continue, and generally with increasing rapidity until the vitality has been reduced to such a point that no means of treatment can restore it. The younger the child the more rapid the loss, and the longer it has continued the greater is the danger.

Acute Inanition.—Rapid loss of weight, frequently spoken of as acute inanition, is common in early infancy, when it often simulates serious organic disease. In older children it is not frequent, and usually is dependent upon some obvious cause. In all the acute diseases of the digestive tract many of the symptoms are due to inanition. The obscure cases are those in which the digestive symptoms, strictly speaking, are not prominent.

The rapid loss of weight usually takes place under one of the following conditions: (1) When a child refuses all food, whether from the breast or the bottle, or can be made to take only an insignificant amount. The cause of this it is often impossible to discover. Symptoms of inanition are sometimes seen at weaning, when a child persistently refuses to take food from a bottle or spoon. (2) When the food given is entirely inadequate, as when an infant is nursing upon a dry breast, or one in which the milk supply is so scanty that the child gets practically nothing. It is occasionally seen later, when the breast-milk, for some unexplained reason, suddenly fails. (3) When the character of the food is improper.

On account of extreme poverty, the infant may be getting only tea or toast soaked in water or albumin water. It may occur in young infants who are fed entirely on starchy foods. (4) When the infant at birth has such feeble powers of digestion, because premature or delicate, that he is unable to take or to digest sufficient food to maintain life. (5) When a sudden change of food is made to one so difficult of digestion that the child is unable to assimilate it. This may happen after sudden weaning. In such cases the symptoms of inanition are mingled with those of acute indigestion, but the former usually predominate.

The mode of development depends upon the antecedent condition. In young infants acute inanition often follows malnutrition, when perhaps there has been nothing noticeable except a gradual loss in weight: or, if the weight has not been watched, it may be observed only that the infant has not been doing well. Severe symptoms may come on quite suddenly, and if the nature and the gravity of the condition are not appreciated the case may terminate fatally in two or three days. The loss in weight is rapid, amounting often to three or four ounces a day. The temperature in the newly-born may be high, but it is more often subnormal. The pulse is weak and may be rapid, but is at times very slow. The heart sounds are feeble. The urine is scanty. The extremities are cold, and the peripheral circulation poor. There is usually complete muscular relaxation. This is especially marked in the abdomen where the muscles almost entirely lose their tone. The skin may be dry or covered with a clammy perspiration. There is extreme pallor, and often a peculiar bluish-gray color to the face. This is always a grave symptom. Cyanosis may be present in children who have previously cried well and in whom there is no suspicion of atelectasis. The respirations are rapid and may be irregular. There may be constant worrying and fretfulness, or a condition of semi-stupor, in which the child makes no sign of wanting food. The fontanel is sunken and the pupils are contracted. The bowels usually move frequently, although there may be constipation, due to the small amount of food taken. When no food is taken for two or three days the stools may resemble meconium.

The progress depends much upon the age of the infants. Those under one month usually succumb quickly. In them the symptoms sometimes last but a few days, seldom more than a week or two. The development of such symptoms in a young infant is a very serious sign. In older infants the progress downward is usually less rapid.

The outcome of such cases is, however, always uncertain, but with proper treatment many may be saved. It is hard for one who is not familiar with the condition to appreciate the great and even the immediate danger in which a young infant may be from inanition, notwithstanding the absence of both vomiting and diarrhea. The treatment must be

immediate and energetic. Breast milk is essential. There is no opportunity to experiment with artificial feeding. No food can be given if there is vomiting or severe diarrhea, but in the absence of these breast milk may be given by gavage if necessary. The intervals should be long—at least four hours. In the event of no vomiting but diarrhea, buttermilk may be given, alternating with the breast milk. If it is impossible to obtain breast milk, buttermilk is probably the best form of diet unless the child is over three months of age, when feeding with protein milk may be attempted. Rectal feeding is of no avail. Often the symptoms are largely due to a lack of water. Injections of a normal salt solution should be given per rectum or under the skin. Hypodermoclysis is often of great value. Absorption is usually prompt. The rapidity with which shrivelled tissues will drink up water is astonishing. Normal saline solution should be employed in amounts from 150 to 240 c.c. once or twice a day. This may be repeated for several days. While the improvement following hypodermoclysis is frequently marked, it must be remembered that the effect is only temporary. Unless proper food is retained and absorbed or the digestion improves, the conditions are soon as bad as ever and subsequent injections produce less and less effect. Saline solution may be given by the drop method into the rectum. This method is seldom satisfactory. Transfusion, by the direct or indirect method, may be life-saving. Energetic stimulation by caffein or camphor, hypodermically, is indicated. Except for the stimulants, drugs are of no use whatever.

Marasmus.—Gradual and progressive loss of weight, wasting, is a symptom of many conditions in infancy. It occurs in tuberculosis, in infantile syphilis, and also as a result of obvious disturbance of the gastro-intestinal tract. At times, however, it appears to be a vice of nutrition only and develops, so far as can be made out, without general or local organic disease. To this type the names of *Marasmus, Infantile Atrophy* and *Simple Wasting* have been applied.

This condition is not very often seen in the country or in private practice; but it is frequent in dispensary practice in all large cities, and is especially common in institutions for young infants. In such institutions, fully half the deaths under one year are directly or indirectly from this cause. It is a very large factor in the immense infant mortality of large cities in summer. Although the cause of death is usually reported under some other name, the determining factor in the fatal result is the previous marantic condition of the patient. The primary cause may be a congenital weakness of constitution which may depend upon heredity. It is often seen in premature children. In the vast majority of cases, however, it depends upon two factors—the food and the surroundings. Among the poor who live in tenements, many artificially-fed infants do

very badly. This is due to neglect, to ignorance in regard to proper infant feeding and inability to procure what the child requires, especially pure cow's milk. A country infant may be neglected in many respects, and is often badly fed; but he has plenty of pure air, and usually thrives. In the city, as long as an infant has a plentiful supply of good breast-milk he continues to do well in most instances, in spite of the fact that his surroundings are bad. When there are not only bad feeding and unhealthful surroundings, but also an inherited constitutional vice, we have all the factors required to produce marasmus in its most marked form. The odds are so against the infant that the feeble spark of vitality flickers for a few months only and gradually goes out.

Another prominent factor in the production of marasmus is the overcrowding and lack of individual care of infants in institutions. Even though artificially fed in an intelligent manner, many infants who are plump and healthy on admission, lose little by little, until at the end of three or four months they become wasted to skeletons, dying of some mild acute illness, such as an attack of bronchitis, the essential cause, however, being marasmus. The common mistake is that of placing too many children in one ward with no chance of obtaining a proper amount of fresh air and with too little individual attention. No house-plant is more delicate or sensitive to its surroundings than is an infant during the first few months of life.

The post-mortem findings in such cases are exceedingly unsatisfactory, and throw little if any light upon the cause of death. Every now and then general tuberculosis is discovered in patients dying apparently of marasmus, the existence of which was not previously suspected. An occasional lesion is fatty liver. This may lead to such enlargement of the organ that its weight is increased by one-half. Both to the naked eye and under the microscope the usual changes of fatty infiltration are present, often to an extreme degree. In the past too much has doubtless been made of this condition of the liver in marasmus. From figures given elsewhere (see article on Fatty Liver), it will be observed that the lesion is not more frequent in this condition than in infants dying from other diseases. Its exact relation to the condition of wasting has not yet been determined.

With these exceptions the autopsies show nothing striking. In the stomach and intestines there is nothing of pathological importance. The theory advanced, that atrophy of the intestinal tubules is the explanation of marasmus, has found little support.

The condition seems rather to be a failure of assimilation, owing to imperfect digestion, improper food, unhygienic surroundings, or feeble constitution. As a result, there is a progressive loss in weight, feeble circulation, imperfect lung expansion, lowered body temperature, and,

finally, a condition incompatible with life, for resistance becomes so feeble that the slightest functional disturbance proves fatal.

The general history of these cases is strikingly uniform. The following is the story most frequently told at the hospital: "At birth the baby was plump and well nourished, and continued to thrive for a month or six weeks while the mother was nursing him; at the end of that period

FIG. 18.—MARASMUS; A PATIENT IN THE BABIES' HOSPITAL, TEN MONTHS OLD, WEIGHT SIX POUNDS. Weight at birth reported to have been nine pounds.

circumstances made weaning necessary. From that time the child ceased to thrive. He began to lose weight and strength, at first slowly, then rapidly, in spite of the fact that every known form of infant-food was tried." As a last resort the child, wasted to a skeleton, is brought to the hospital.

The most constant symptom is a steady loss in weight until a condition of extreme wasting is reached, at which point these patients may

remain for many weeks. Their general appearance is characteristic. They have an old look; the skin is wrinkled, has lost its tone, and hangs in folds upon the extremities (Fig. 18). The legs are like drumsticks; the abdomen is prominent; the temples are hollow; the fontanel is sunken; the eyes large; the features sharp; and the hands resemble bird-claws. Often the children are reduced literally to skin and bones. Anemia is a very marked and almost constant symptom. Accidental heart-murmurs are frequently heard. The body temperature is usually subnormal unless artificial heat is employed. A rectal temperature of 95° or 96° F. is very common, and one of 93° or 94° F. is occasionally seen. In addition to the pallor of the face, there may be a leaden hue.

A not infrequent symptom is *general edema*. The first thing which calls attention to this is often an unexpected gain in weight which may amount to several ounces a day. The edema may increase until the cellular tissue of the entire body is water-logged. There are not, however, effusions into the large serous cavities. The exact pathology of this nutritional edema is not clear. It is of quite frequent occurrence in cases of marasmus, especially in infants under six months of age. It seems impossible to connect it with any definite form of feeding. Thus, we have seen it in infants kept for a long time upon barley water, in others who were receiving nothing but condensed milk, in still others who were taking a milk formula apparently of suitable proportions. The urine in the marked cases shows neither albumin, nor casts, but usually an almost complete absence of chlorids. Coincidentally with the disappearance of the edema the chlorids appear in the urine, showing a close association between the retention of chlorids and the retention of water in the tissues. Thus, in one case at the height of the edema the child was eliminating but .008 gram of sodium chlorid daily; three days afterwards, while the dropsy was rapidly disappearing, the amount exceeded .5 gram. In the treatment of this condition the most satisfactory food in our experience has been protein milk. Whether this is due to its low sodium chlorid content or not it is impossible to say. The administration by mouth of digitalis has seemed also advantageous. An infant of three or four months can take half a dram of the infusion three or four times daily.

The stools are sometimes normal, but usually contain undigested food with mucus. No matter how carefully fed, these patients are easily upset. Vomiting is readily excited. The appetite in many is almost entirely lost; others take their food quite well and have fairly good stools but steadily lose weight.

Frequent complications are thrush and bedsores which are sometimes seen over the sacrum or heels, but most frequently upon the occiput. Occasionally there is seen a reflex spasm of the muscles of the neck, producing a marked opisthotonus, which may last for several days or weeks.

In hospital wards these infants are very susceptible to all infections, particularly to those of the respiratory tract. Otitis, rhinopharyngitis, bronchitis and pneumonia are especially common.

The progress in most cases is steadily downward; but it may be cut short at any time by acute disease. Frequently these infants die suddenly when apparently they are as well as they have been for several weeks. In summer they wilt with the first days of very hot weather, and die often in a few hours from a slight functional derangement of the stomach and bowels.

The symptoms shown by some infants that have been fed for a long time upon a diet almost exclusively of carbohydrates merit special consideration. They suffer from what the Germans call *Mehlnährschaden*. The infants may have received proprietary foods or cereal decoctions in order to overcome diarrhea or because milk is impossible to obtain, and it is a restriction to carbohydrates for a long time that causes the characteristic symptoms to develop. For a while they may hold their weight or may even gain; before long, however, they begin to lose weight and the loss may be extreme. There is in some instances a marked tendency to edema which may mask the loss. Pallor is striking.

Of especial importance, however, are a peculiar rigidity of the musculature and a great lessening of immunity to infection. The rigidity is especially marked in the legs. The muscles are contracted and hard. It is difficult to extend the extremities. In severe cases opisthotonus may develop. The diminution in the resistance to infection allows of the development of furuncles, otitis, bronchitis and infections of the eyes. Especially characteristic is keratomalacia with perforation of the cornea and destruction of one or both eyes.

The condition is a severe one and is frequently fatal. The longer it has existed the worse the prognosis. Infants with keratomalacia seldom recover. The severity of the condition is in large part due not only to the insufficiency of the food as a whole, but to the almost complete absence of fat, protein and salts. It is not unlikely that vitamines are also lacking.

With loss of weight from any cause the older the child the better the chances of recovery. Very young infants are always difficult subjects to deal with. They go down more rapidly and build up more slowly than those who are older. Much depends upon whether everything possible can be done for the child: whether a wet-nurse can be secured and whether the patient can have the benefit of the best surroundings, in the country in summer and in winter a warm climate where he can be kept out of doors the greater part of the time. In institutions cases in infants under four months old are usually hopeless. Of those over eight months quite a proportion can be saved by proper treatment, even though the body-weight

is reduced to eight or nine pounds. When recovery occurs it may be complete, and the child at two or three years may be as vigorous as any child of his age.

The most important treatment is that which relates to prophylaxis. Maternal nursing should be encouraged by every possible means especially among the poor. For those who must be artificially fed the important things are a pure milk supply together with proper instruction as to how it is to be used in infant feeding. At the same time opportunities for fresh air should be secured. This is a large part of the difficulty in institutions.

As far as possible, wet-nurses should be obtained if the infants are under four months old. For these very young patients success by artificial feeding is generally impossible. With those of six months or over, intelligent artificial feeding is very frequently successful. In institutions we seldom succeed without at least partial breast feeding.

For very young infants, with a temperature which is habitually subnormal, some means of maintaining the body heat must be employed. The simplest and usually an effective means is to oil the body and envelop it completely in a cotton jacket and then surround it with hot-water bags or bottles. The room should be kept warm. In institutions it is convenient to have a warm room for such infants, the temperature of which is kept about 80° F. These infants require no drugs but a great deal of careful nursing.

Malnutrition.—Failure to gain properly is exceedingly common among young children, and such cases occupy a large part of the time and attention of one engaged in pediatric practice. The term malnutrition perhaps characterizes them better than any other. Although these children can not be said to be actually ill, they are very far from well, and their condition is often the cause of the greatest solicitude on the part of parents, not only from the existing state of health, but from the apprehension of the development of some serious organic or constitutional disease, especially tuberculosis.

Certain children are delicate from birth, possessing only feeble vitality, though without giving evidence of any actual disease. They are often the offspring of parents of delicate constitution and poor physical development, or of those with tuberculosis, gout, or syphilis. Very many city children are included in this group. Among the poor the condition is the result of bad hygiene, insufficient or improper food, overcrowding, etc. Among the well-to-do it is seen in those who inherit a too highly developed nervous organization with a corresponding amount of physical deterioration. Another group includes those children who were premature or very small at birth, weighing perhaps only three or four pounds. Many cases are traceable to improper feeding or equally poor nursing

during the first few months. These children get a poor start in life, and on that account are handicapped throughout infancy. A frequent cause of malnutrition in infants is the pernicious custom of keeping them in close apartments where the thermometer ranges from 72° to 75° F., and where the greatest anxiety is constantly felt lest they take cold. Such infants may lose in weight, become anemic, and exhibit all the signs of malnutrition when nothing else is wrong except the conditions mentioned. Malnutrition often depends upon some previous acute disease, especially of the stomach and intestines.

In children who are over two years old the condition of malnutrition may be due to any of the factors above mentioned—inherited feebleness of constitution, bad feeding and its resulting indigestion, too little fresh air, and close confinement indoors. It is, however, at this period, much more frequently than in infancy, dependent upon some previous acute disease. As a result, an impression is left upon the child's constitution which lasts for months, often for years, and which manifests itself not by any special local symptoms, but by a general condition of debility. Faulty methods in education, especially overpressure in schools may have a deleterious effect upon the health of older children.

Not only the weight but the general physical development is much below the normal. At one year the body length may be three or four inches less than the average. Dentition may not be materially delayed. Muscular development is backward; many of these children do not sit alone until a year old, and barely walk at two and a half or three years. The muscles are soft and flabby, and the ligaments so weak that paralysis is often suspected. The body is so small that the head seems unnaturally large, and a diagnosis of incipient hydrocephalus is frequently made. Mentally these infants are somewhat backward but the mental development is often strikingly in advance of the physical. Some symptoms of rickets are usually present.

The examination of the blood reveals the usual changes of a moderate secondary anemia. The circulation is poor, the hands and feet are frequently cold. In many children the skin is unnaturally dry; in others there is a disposition to excessive perspiration, particularly about the head. Nervous symptoms are frequently present. These children are restless, fretful, and irritable; they sleep badly. Enlargement of the lymph glands, especially those of the neck, is common.

One of the most characteristic things about these patients is their feeble power of digestion and assimilation. Unremitting care and constant watchfulness are required to keep them up to even a moderate standard of health. The most trivial changes in food may upset them. Attacks of acute indigestion are usually brought on by overfeeding—the mistake which is almost invariably made by mothers who are discouraged

with the slow progress made, and are anxious to make their children grow fat and strong. The balance is so delicately adjusted that the slightest deviation from proper rules of feeding, either as to the quality of the food or the quantity given, is immediately followed by an attack of acute indigestion, often by severe diarrhea. As a result, the child may lose as much in two or three days as he has gained in a month or more. These acute attacks, if in summer, not infrequently prove fatal. Not only do these patients have but little resistance to acute disturbances of the stomach and intestines, but any acute disease is serious—measles, whooping-cough, and pneumonia being especially fatal.

If under six months of age, among the poor or in institutions, such infants are almost certain to go on from bad to worse. In private practice, where it is possible to have the best care and surroundings, with the coöperation of an intelligent mother or nurse, a very large number of these infants can be reared. After the second year has passed the problem becomes a much simpler one, and if infectious diseases and other forms of acute illness can be avoided, the probabilities are in favor of the child's becoming stronger each year and growing to maturity.

Older children are thin, pale, and undersized, particularly if the condition is constitutional or hereditary. Sometimes they are taller than the average for their age, and their symptoms are often attributed to too rapid growth. One of the most striking things about children suffering from malnutrition is their vulnerability. They "take" everything. Catarrhal processes in the nose, pharynx, and bronchi are readily excited, and, once begun, tend to run a protracted course. There is but little resistance to any acute infectious disease which the child may contract. Often one illness quickly follows another, so that these children are not infrequently sick for almost an entire season. Their muscular development is poor; they tire readily; are able to take but little exercise, and their circulation is sluggish. Mentally they are usually bright, often precocious. They are cross, fretful, and any unusual excitement produces an effect which lasts for some time. Their sleep is usually disturbed and restless; they waken frequently, and occasionally suffer from night-terrors.

Digestive symptoms, if not constant, are very easily excited. Children of five or six years have to be fed as carefully as infants. The appetite is usually poor, and mothers are distressed because their children eat so little, yet, when food is urged upon them, attacks of indigestion follow with singular regularity. The tongue is slightly coated the greater part of the time. The bowels are apt to be constipated, apparently more from lack of muscular tone than from anything else. From time to time there may be large quantities of mucus in the stools for two or three days. A

moderate amount of anemia is always present, and this may be the most striking feature.

The duration of the condition depends very much upon the cause. If the cause is constitutional or inherited, it is likely to last throughout childhood, but it often greatly improves about the time of puberty. When it follows some acute illness it commonly lasts for a few months only. The longer the condition has lasted and the greater the general disturbance the slower will be the improvement. The great danger is the supervention of some acute disease.

It is oftentimes difficult to find out to what the failure properly to develop is due. Much regarding inherited constitutional tendencies can be learned from the family history and from the condition of other children in the family. Tuberculosis must be carefully excluded.

Other things to be considered are syphilis, rickets, diseases of the blood, intestinal parasites and, of course, organic diseases of the lungs, heart, stomach, intestines, liver and kidneys. Even malignant disease, though rare, should not be overlooked. It may take careful observation for several days, and sometimes for weeks, with repeated physical examinations, before all these conditions can be positively excluded.

In private practice a large proportion of cases are due to improper feeding or nursing; next in importance are improper surroundings, and last come inherited constitutional conditions. In other words, most of these children are born healthy, but become ill or delicate in consequence of improper management.

In older children, after excluding constitutional and local diseases, the whole life of the child must be investigated to discover the fundamental condition which is at fault. A carefully obtained history from infancy is of the greatest assistance. It is often difficult, and sometimes impossible, to get at the primary factor, for in cases of long standing there may be symptoms connected with almost every function of the body. One should scrutinize closely the quality and quantity of food given, the amount of sleep, the hours of study and recreation, the amount of exercise in the open air, and the physical conditions surrounding the child. Usually the most important factor in the case can be discovered.

The problem of nutrition is to be solved by diet and general management; drugs occupy a very small place in treatment. With infants whenever possible breast feeding should be employed. Next in importance to diet is fresh air. The natural tendency of a mother who has a delicate infant is to house him closely and never allow him a breath of fresh air. It is of the greatest assistance if these children can be sent to a warm climate for the winter. If this is not possible, fresh air may be obtained by changing apartments, or by an airing in the room with the windows open.

Cold sponging is a valuable tonic that can only be employed, however, with fairly vigorous infants that react promptly. If the child remains blue and cold for some time afterward, the cold sponging should not be repeated.

Friction and massage are useful in many cases. The child should be laid upon the lap of the nurse, if possible, before an open fire, and should always be covered with a blanket. The entire body should then be rubbed for ten or twenty minutes with the bare hand, or, better, with cocoa butter. Professional operators are inclined to be too energetic for little children.

The only tonics of much value are iron, preparations of malt, nux vomica, and cod-liver oil. Cod-liver oil is too much used in these cases, and in too large doses. Many of these infants can not take it at all. It should not be given when the tongue is coated and the appetite poor. The dose should always be small, e. g., ten to twenty drops of the pure oil three times a day, or twice as much of an emulsion.

Experiments in treatment are nearly always unfortunate. The physician should indicate in writing, for the guidance of the mother, specific rules with regard to the amount of food, the time at which it is to be given, the hours for bathing, sleep and airing. He should see the patient at regular intervals and often enough to be sure that his orders are being enforced. Good results are obtained only by constant watchfulness.

The same general principles are to be applied to older children as to infants. The diet is of the first importance. Only the simplest and most easily digested articles of food should be given. Milk, beef, eggs, the lighter and more easily digested cereals and vegetables, bread, and fruit should form the diet. All sweets, pastry, highly seasoned food, candy, nuts, tea, and coffee should be absolutely prohibited, and, in fact, all the articles mentioned as "forbidden" in the Chapter on the Feeding of Older Children. When the appetite is poor and simple food not well taken, the child should not be allowed to take indigestible articles for the sake of eating something. Nothing should be given between meals, and regular hours of feeding must be followed. Three meals a day, for children over three years old, are better than the practice of giving more frequent feedings. Under no circumstances should children be coaxed, urged, or hired to eat; much less should they be forced to do so. There is a popular misapprehension in regard to the variety in diet which children need. Most children do better when a very simple and fairly uniform diet is continued.

The nervous factor is a very large and a very important one. Many of these children are essentially cases of neurasthenia at as early an age as four or five years. Excitement and activity are what they crave and what must be most carefully avoided.

The general habits of children should be directed; there should be regular and early hours for retiring, freedom from undue excitement, and interest should be awakened in outdoor amusements. Children should be kept as much as possible in the open air, but the amount of active exercise should be strictly limited. Usually they do much better if they can be in the country during the entire year. Only a limited amount of reading and study should be allowed; and if children are at school, care should be taken that overpressure is not the cause of the symptoms, particularly in an ambitious child. Cold sponging given in the morning, as described in the introductory Chapter on General Therapeutics, is extremely beneficial to children who take cold readily. In general, these children require early hours, a simple diet, a quiet, regular life, and very little medicine.

In recent years there has been a disposition to attribute many of the symptoms included in the foregoing pages to insufficiency in the secretion of the ductless glands. Extracts from these glands have been widely employed in treatment. There is no satisfactory evidence that such an etiology is correct or such a treatment beneficial.

CHAPTER VI

DISEASES DUE TO FAULTY NUTRITION

The diseases due to faulty nutrition are numerous. There are two, however, which have been so clearly shown to originate in this way that they may be put in a class by themselves. These are scorbutus and rickets. The purpose of considering them in connection with the disturbances of nutrition is to emphasize this relationship.

SCORBUTUS (*Scurvy*)

Scorbutus is a constitutional disease due to some prolonged error in diet. It is characterized by spongy, bleeding gums, swellings and ecchymoses about the joints, especially the knee and ankle, hemorrhages from the nose, and occasionally from other mucous membranes, extreme hyperesthesia, and often pseudo-paralysis of the lower extremities. Added to these local symptoms there is in advanced cases a general cachexia with marked anemia. While scorbutus and rickets are very frequently associated, they can not be considered as different forms of the same disease.

Cases of scorbutus were, however, described in older writings under the title of Acute Rickets.

Scurvy was well recognized and graphically described by Glisson as long ago as the middle of the seventeenth century. For our earliest modern knowledge of the pathology of this disease we are indebted to the observations of Barlow and Cheadle. On the continent of Europe scurvy is most frequently known as Barlow's disease.

Scurvy is not a rare disease. In active hospital and private practice many cases are seen each year.

Etiology.—Age is an important factor; more than four-fifths of the cases occur between the sixth and the fifteenth months, and half of them between the seventh and the tenth months. Scurvy has been seen in infants under a month old. The majority of the cases reported have been observed in private practice, often in the best surroundings. Previous disease is not a factor of much importance. Most of the children attacked have been in good health up to the development of scurvy. In about one-fourth of the number some previous derangement of the digestive tract has existed.

The only etiological factor yet known to bear any constant relation to the production of scurvy is diet. The important facts regarding the previous diet have been well brought out by an investigation of the American Pediatric Society. They were as follows:

Previous food	Breast-milk	in	12	cases;	alone in	10.
	Raw cow's milk	"	5	"	" "	4.
	Pasteurized milk	"	20	"	" "	16.
	Condensed milk	"	60	"	" "	32.
	Sterilized milk	"	107	"	" "	68.
	Proprietary infant-food	"	214	"		

This table shows that while scurvy may occasionally develop with almost any variety of food, three stand out prominently—viz., proprietary infant foods, condensed milk, and sterilized milk. In all of these it would appear that something needed for normal healthy nutrition is wanting. Scurvy is not likely to follow unless an improper diet is continued for a long period, usually several months. In some instances when it developed in nursing infants, the nurse's milk has been examined and found totally inadequate to the needs of nutrition, many of the children having exhibited serious disturbances of nutrition before any signs of scurvy appeared.

Several cases have come under our observation where scurvy has developed in children who have been kept for four or five months upon raw milk, very greatly diluted. Scurvy may result from taking milk which has been pasteurized, usually when the temperature has been high

(167° F.), and the time prolonged (30 minutes). With the lower temperature now more generally employed (155° F.), it is less likely to develop. We do not believe scurvy to be a frequent result of the pasteurization of milk, and not to be weighed against the advantages of pasteurization; but still a danger to be reckoned with. Since the general use of pasteurized milk the number of cases of scurvy is certainly on the increase. The number of cases which develop while on a diet of boiled or sterilized milk is so large that we are driven to the conclusion that the heating alone is the cause, especially since prompt recovery has often followed when no other change is made than to discontinue the heating. These facts show that the sterilization of milk is attended with some disadvantages, and should not be continued as the sole diet for long periods.

The addition of carbohydrates to the food affords no protection, but rather increases the danger of scurvy.

Scurvy frequently develops after the prolonged use of condensed milk or proprietary foods as the sole diet.

There is certainly a predisposition to this disease on the part of some infants, for with the same diet one child may develop the disease while another remains free. We have seen twins fed in exactly the same way, one of whom developed scurvy while the other did not.

While it may be regarded as established that the cause of scurvy is dietetic, no single dietetic error can be held responsible for the disease. It has been recently shown that there are substances in foods vitally necessary for health, the vitamins. It is quite clear that scurvy depends upon the absence of some of these. Either they are lacking in the food or have been destroyed by prolonged heating. Typical scurvy can be produced in some animals by giving a diet chiefly of grain with no fresh vegetables or fruit. The addition of these latter articles immediately cures the disease. So it is with children who are at once relieved by orange or lemon juice, or as Freise has shown, by the dried alcoholic extracts of vegetables.

Lesions.—The most marked effects of scurvy are seen in the bones, blood-vessels, and the blood. The number of recorded autopsies in this disease is not large. We have had the opportunity of making examinations in seven cases. The findings are remarkably uniform, but represent, of course, the extreme results of the disease. The most striking lesion is subperiosteal hemorrhage, which is practically constant and may occur almost anywhere in the body, but affects chiefly the bones of the lower extremities; it is often very extensive, and may reach from the knee to the great trochanter, or from the ankle nearly to the knee. Extravasations may also be found between the muscles, and blood may infiltrate the cellular tissue in the neighborhood of the joints. Besides

these lesions resulting from hemorrhagic periostitis the bone itself may be affected. Separation of the epiphysis from the shaft of some of the long bones, generally at the shoulder, lower end of the femur or lower end of the tibia, is found in most of the fatal cases. Notwithstanding the serious lesions near the large joints, the joints themselves are usually normal.

The microscopical changes in the bones due to scurvy are quite characteristic. They consist in hemorrhages within the marrow as well as beneath the periosteum. There is a diminution of osteoblastic activity; the osteoblasts are relatively few in number and the formation of new bone is decreased or has altogether ceased. What bone has been formed, however, is well calcified. The absorption of bone is not increased. For this reason the shaft of the bone is firm but there is a place of least resistance in the subepiphyseal zone owing to the lack of bone formation. It is through this weakened zone that separation occurs as the result of very slight traumatism.

The marrow undergoes extensive changes. The marrow cells in areas, especially in the neighborhood of the epiphyses, have largely disappeared, leaving only the supporting cells. In addition there are almost always found some of the changes characteristic of rickets.

The visceral lesions are inconstant. Those most frequently found are small hemorrhages beneath the pleura, pericardium, and peritoneum, sometimes into the various organs, also bronchopneumonia and nephritis, which occasionally occur as complications.

There may be small extravasations found upon the surface of any of the mucous membranes. Alterations in the blood-vessels are undoubtedly an important factor in bringing about the disposition to hemorrhage. The changes in the blood, in the gums, and the lesions of the skin will be considered with the symptoms.

Symptoms.—In many cases a period of indisposition, fretfulness, pallor, and failing nutrition precedes the local symptoms, but usually tenderness of the legs is the first symptom noticed. In the beginning this is occasional and so slight as to cause the infant to cry only upon being handled. Later it becomes almost constant and is very acute. At first this soreness is not very definitely localized, but is generally more marked about the knees and ankles. Some swelling may be noticed, often just above the ankle joints. Coincident with these may be seen the changes in the mouth. The gums are of a deep purplish color, swollen, particularly about the upper central incisors, and may quite cover the teeth. They bleed from the slightest irritation, and sometimes spontaneously. The child now becomes fretful and cross, sleeps badly, loses color, weight, and appetite. He may become quite cachectic in appearance. All these symptoms come on very gradually, often with periods of

a few days in which apparent improvement is seen. Sometimes they may continue for several weeks without making any perceptible impression upon the child's previously good condition.

If the disease is recognized, and proper treatment instituted, rapid improvement follows, with complete and permanent recovery. If not recognized, and the faulty diet is continued, the disease advances to the more severe form. The tenderness of the legs becomes exquisite, so that any movement or even the slightest touch causes the child to scream with pain or apprehension. The posture is very characteristic. There is semiflexion of thighs and legs and outward rotation at the hip. (See Fig.

FIG. 19.—SCURVY SHOWING CHARACTERISTIC SWELLINGS AND POSTURE. Patient 8½ months old, fed exclusively upon malted milk after the age of 3 months. Epiphyseal separation at the upper extremity of both humeri, lower extremity of both femora and lower extremity of left tibia. Prompt and complete recovery.

19.) In this position the child often lies motionless and voluntary movements of the extremities can not be excited. Paralysis is often suspected. The disability is chiefly owing to the extreme pain which motion provokes, but may depend upon epiphyseal separation. Small ecchymoses are frequently seen about any of the large joints, resembling the ordinary "black-and-blue" spots, and these often confirm the opinion previously formed that the child has met with some accident. The swelling near the joints, particularly the knee, may be so great that the limb is nearly twice the size of its fellow. The buccal symptoms are usually striking. In addition to spongy, swollen, bleeding gums, dark purplish bags may be seen over teeth not yet through. There may be bleeding from the roof of the mouth or from the pharynx. The pain is sometimes so severe as seriously to interfere with taking food; there is moderate though rarely extreme salivation. Blood may be vomited or passed with the feces or the urine. In the severe cases the stools are rarely normal, more or less catarrhal colitis usually being present. The general condition is one of grave anemia, accompanied by a marked cachexia and progressive wasting. The child cries almost constantly, sleeps little, and is truly a pit-

iable object. Slight fever is usually present; and in some of the more rapidly progressing cases with extensive lesions a temperature of 102° or 103° F. is common. Unless recognized and the cause removed, the condition grows steadily worse, the symptoms continuing until death occurs either by slow asthenia, or suddenly from heart failure, or from some intercurrent disease, such as bronchopneumonia or acute gastro-enteritis. The duration of the illness in the fatal cases is from two to four months.

The onset is gradual in the great majority of the cases, the earliest symptoms noticed in the order of frequency being pain and tenderness of the legs, soreness and sponginess of the gums, disability, anemia, cutaneous hemorrhages, and very rarely hematuria.

Pain and tenderness are very prominent, being noted in about 95 per cent of the cases; in the majority they are present only on motion or handling. The location of the pain and tenderness in 184 cases was as follows: Lower extremities alone, 133; upper extremities alone, 2; lower and upper, 42; lower and trunk, 7. In all but two cases, therefore, the lower extremities were affected, the lower part of the thigh and the leg just above the ankle being the usual seat.

Disability, or pseudo-paralysis, is a very common symptom, and in all severe cases a constant one. It exists in varying degrees from a slight disinclination to use the limb to complete helplessness. In many cases it is more marked than the pain, and has led to a diagnosis of poliomyelitis.

Swellings are associated with pain and tenderness in most of the severe cases. They are most marked near the joints, but may extend for some distance along the shafts of the bones. In nearly all cases the location is the lower part of the thigh or the lower part of the leg, and usually of both sides. Swellings are occasionally seen at the shoulders or wrists; in rare cases there may be swelling about the elbows or hips or over the ribs, scapula, or ilium. Redness is not generally present, but the parts may have a dark purplish color. It is to the hemorrhages that both the swellings and the discoloration are chiefly due. There is often marked edema of the affected limbs.

Protrusion of the eyeball is present in a small proportion of the cases; an extreme exophthalmus is sometimes seen, and is due to orbital hemorrhage.

The gums are affected in nearly all cases, the exceptions being those recognized and treated early. Hemorrhage occurs in about one-half the cases, and frequently there is ulceration not unlike that of a mercurial stomatitis. It is rather curious that, though the lower teeth are cut first, the upper gum is almost always most affected, and in the milder cases usually alone involved. Of 45 cases in which no teeth had been cut, the gums were affected in 24 and normal in 21. This is sufficient to disprove

the old opinion that the gums are affected only when teeth have appeared. The severe inflammation and ulceration sometimes seen seem to be the result of secondary infection.

Hemorrhages beneath the skin are present in about half the cases. They are rarely extensive, usually multiple, and their location is no doubt often determined by a slight traumatism. Hemorrhages from the mucous membranes are not quite so frequent. There may be bleeding from the gums, nose, bowels, and rarely from the stomach. Hemorrhages in most cases are frequently repeated, but seldom profuse.

Epiphyseal separation is seen in most of the very severe cases. It is most frequently either of the lower epiphysis of the femur or the tibia, or the upper epiphysis of the humerus, and is often bilateral. The actual separation may be caused by some slight injury, the condition of the bone predisposing to this occurrence. In several cases of our own with separation which recovered, rapid union occurred under anti-scorbutic treatment.

Early in the disease, even though marked swelling of the limbs may be present, an X-ray examination may show little or nothing. The subperiosteal hemorrhages can not usually be made out until there is a deposition in the effusion of the salts of calcium. Then they appear with great clearness as spindle-shaped thickenings of the bones, sometimes running the whole length of the diaphyses. These are absorbed very slowly and require weeks or months to disappear. Changes at the epiphyses are also found. They consist in distortions and irregularities of the normal line. Separation of the epiphysis can be occasionally made out. Some rachitic changes also can usually be recognized.

Anemia is slight in the early stage, but increases as the disease progresses. Blood examinations may show marked reduction of the hemoglobin, sometimes to thirty-five or forty per cent; also in nearly all cases a proportionate reduction of the red cells. The changes are those of an ordinary secondary anemia.

The urine contains albumin in about one-fourth of the cases; in nearly half of those containing albumin casts also are found. In rare cases hematuria is an early symptom. Blood cells usually in moderate numbers are found in practically all but the mildest cases, and are of some diagnostic importance.

Evidences of general malnutrition are present in all advanced cases, varying, of course, greatly in degree. In a few infants under our own observation the weight, color, and general appearance of health have continued in spite of very decided local symptoms. In most of them the impaired nutrition is shown by loss of appetite, occasional attacks of vomiting, and still more frequently by derangements of the bowels, which vary from slight indigestion to a serious catarrhal condition of

both small and large intestine. It is with the latter that the discharge of blood is usually seen.

Association with Rickets.—In the American Pediatric Society's investigation great pains were taken to obtain definite and accurate data regarding this. Of the cases, 340 in number, in which this point was noted, symptoms of rickets were present in 152, or 45 per cent. Mild grades of rickets are, of course, impossible for us to recognize. Post mortem, rickets is almost invariably found associated with scurvy, for the reason that during the age at which scurvy may develop rickets is, in hospital patients, a well nigh universal disease. There is no reason for believing rickets and scurvy to be different forms of the same disease. The two most striking characteristics of scurvy, viz., tendency to hemorrhages and prompt curability by fresh food and fruit juices, have no counterpart in rickets.

Diagnosis.—The disease with which infantile scurvy is most frequently confounded is rheumatism. In fully four-fifths of the cases which have come to our notice this has been the previous diagnosis. The extreme rarity of rheumatism under one year should always make one cautious; pain and tenderness of the legs only, should, in an infant, invariably suggest scurvy rather than rheumatism. The extreme disability has often led to a diagnosis of poliomyelitis, but here again the acute tenderness should set one right. Many cases of scurvy come into the hands of the orthopedic surgeon with a diagnosis of joint or spinal disease. Where the swelling was mainly of one limb we have twice known a diagnosis of malignant disease to be made, from the cachexia, the shape of the swelling, the discoloration, and the pain. We have known two cases to be operated upon by eminent surgeons, once with a diagnosis of sarcoma and once of ostitis of both tibiae. Not until the subperiosteal hemorrhages and epiphyseal separation were discovered was the nature of the trouble suspected.

The diagnosis of scurvy seldom presents any difficulties to one who has once seen a case. No one need err if the essential features of the disease are kept in mind: the extreme soreness of the legs, spongy, swollen gums, swelling near the large joints, a tendency to hemorrhages, and usually a history of the prolonged use of some proprietary infant food, or sterilized or condensed milk. The epiphysitis of hereditary syphilis has many symptoms in common with scurvy, but it usually occurs at an earlier age (before the fifth month) and other evidences of syphilis are usually present. If any doubt exists, this will be removed by the prompt improvement and generally rapid cure following an antiscorbutic diet.

Prognosis.—This is invariably good if the disease is recognized early. No patients with symptoms so serious improve with such marvelous

rapidity as do the great majority of those with scurvy, under proper management. The figures of the American Pediatric Society's report on this point are interesting. The average duration of the disease before treatment was begun in over three hundred cases was somewhat over three weeks. In eighty per cent striking improvement was noticed during the first week of treatment, and in forty per cent within three days. Over two-thirds of these cases were well within three weeks, and nearly one-third within one week, after the beginning of treatment.

It is only when the disease is of long standing, when the malnutrition is severe, or when serious complications, usually involving the digestive tract, are present that the symptoms persist and the issue becomes doubtful. It is difficult to tell what the exact mortality of scurvy is. Any case allowed to go on may result fatally. The younger the infant the more likely is this to occur. We have seen five fatal cases. In one of our patients death resulted from hemorrhage which followed an incision into an epiphyseal swelling at the lower end of the femur, made before the patient was seen and which persisted despite all treatment. Barlow's early article included thirty-one cases with seven deaths. It is rare that scurvy leaves any permanent effects. Recovery is not only rapid but complete. Relapses are extremely rare and have been observed only in one or two cases, where chronic indigestion existed of so extreme a character that proper feeding was impossible. The after-effects are usually the result of prolonged malnutrition, of which the attack of scurvy was only one manifestation.

Treatment.—Prevention requires that all infants reared on sterilized or pasteurized milk should be given other food much earlier than was formerly thought necessary. It is not enough to add gruel or farinaceous foods. Fruit juices should be begun as early as the fifth or sixth month. Beginning with two teaspoonfuls the amount may gradually be increased to two or three tablespoonfuls daily and continued as a regular part of the diet. The early use of broth in which green vegetables have been cooked is also of value, or the grated vegetable may be given to the infant as a purée. The treatment of scurvy is usually very simple—viz., to discontinue all proprietary foods, condensed milk or sterilized milk, and to substitute a diet of fresh cow's milk, modified to suit the child's digestion. With this change alone improvement will soon begin and gradual recovery take place. However, when fresh fruit juice is added improvement is much more rapid. It should always be combined with the change in diet. Orange juice is to be preferred, but the juice of any fresh ripe fruit will answer the purpose. Oranges should be sweet and fresh. From two to four ounces of the juice a day are required, best given in divided doses, about one hour before the milk-feeding. It may be given plain or diluted with water. In some cases when not well tolerated by the

stomach, it is better given at night when no food is taken. Potato also has marked anti-scorbutic properties, and may be given in the form of a purée to infants as young as eight or ten months. The only really difficult cases to manage are those in which the general condition approaches one of marasmus, or when scurvy is accompanied by marked gastric or intestinal disturbance. When an intestinal catarrh is present, with the bowels moving five or six times a day, one may hesitate to give the fruit juice for fear of increasing these symptoms. In a number of instances we have seen intestinal symptoms, which had resisted ordinary measures, immediately improved by the fruit juice, thus establishing their intimate connection with the scorbutic condition.

Other things of value are fresh beef juice, and for older children all fresh vegetables, especially potato. The anemia and malnutrition call for iron, cod-liver oil, and other tonics, which should be given after active symptoms of the disease have disappeared. Infants with scurvy should be handled as little as possible, and should be particularly protected against exposure in their extremely susceptible condition. To relieve pain and prevent deformity the affected limbs should be immobilized by splints during the period of marked symptoms if epiphyseal separation has taken place, and in many other severe cases.

RICKETS (*Rachitis*)

Rickets is a chronic disease of nutrition. While the only important anatomical changes are found in the bones, it is not to be regarded as a disease of bone, but as a very complex pathological process, the result of disturbed metabolism, which affects chiefly the bones, but also the muscles, ligaments, mucous membranes, and nearly all the organs of the body, including the nervous system. It occurs especially between the ages of six and eighteen months. While not a fatal disease *per se*, rickets adds very greatly to the danger from all acute diseases in infancy, and even to some degree also to those of later life.

The great frequency of rickets has only recently been recognized. It is probably, at least in cities, the disease from which infants most frequently suffer. It has been possible to determine this only since the pathology has been firmly established, for many cases give no clinical evidence and the disease can be recognized only post mortem. The symptoms by which we recognize rickets are chiefly due to bone changes, and these must be quite well marked before they are discovered clinically. For this reason rickets may run its course without any suspicion having been aroused as to its presence. Schmorl found in 386 consecutive autopsies upon children dying between the second month and the fourth

year, evidence of rickets in 90 per cent, while 96.6 per cent of infants between the fourth and eighth month were rachitic. There can be no doubt that among the poor in cities, rickets is an almost universal disease.

Etiology.—Certain facts in the causation of rickets are well known. It is closely related to improper feeding and bad hygienic surroundings. Artificially-fed children are much more prone to the disease, especially those who are badly fed. Breast feeding does not entirely protect against the disease, though it greatly modifies its character. Severe forms of rickets are not common in nursing children unless lactation is unduly prolonged, as, for example, when nursing is continued for fifteen to eighteen months without other food. There is a predisposition on the part of certain children to acquire rickets quite independent of the food. Of two children that are nursing the same woman, one may develop rickets perhaps in a severe form and the other may escape it; and allowing a rachitic infant who has been breast fed to nurse a woman whose own child is not rachitic, brings no assurance that the rickets will be cured.

The diet of children who develop rickets upon artificial feeding is most frequently deficient in fat and often at the same time in protein, while it is apt to contain an excess of carbohydrates. It has been believed that the most important factor is the deficiency of fat. Rickets is exceedingly common in children reared upon the proprietary foods, nearly all of which are very low in fat and contain an excess of carbohydrates. It is also common in children who are reared upon sweetened condensed milk.

According to Feer, infants in the mountainous parts of Switzerland seldom develop rickets although they may have been breast-fed for only a short time and thereafter are given a diet almost exclusively of carbohydrates. It is doubtful if diet has the importance that has been ascribed to it in the past.

Though animals under domestication suffer from rickets, it is impossible to produce the disease by even the most abnormal diet. Certain experiments have been made which show that a condition of the bones resembling rickets may be produced in animals by a diet deficient in calcium salts, and furthermore that this may be cured simply by the addition of these salts to the food. The conclusion can not, however, be drawn that rickets in children is produced in this manner. In the first place the bony condition in the artificial disease is not histologically the same as that seen in rickets; again, rickets in the child is not cured simply by the administration of calcium salts; and, finally, rickets develops when these elements have not been deficient in the food.

Rickets is essentially a disease of cities, being most often seen in children living in crowded tenements where, in addition to improper

food, the hygienic surroundings are the poorest. For this reason poor ventilation, filth and lack of sunlight have been regarded as potent factors in producing the disease. Their exact influence is difficult to determine.

Distribution of Rickets.—It was formerly held that rickets was almost unknown in many parts of the world. It is now apparent that practically no region escapes. The greatest frequency of the disease, however, is in the temperate zone. Tropical and semi-tropical countries are relatively free from rickets. But the inhabitants of these countries, particularly the negro and the Italian, when removed to cities of the temperate zone, suffer most frequently and severely. In the cities of America no race is exempt from the disease. In New York the greatest susceptibility is among the negroes and Italians. The extreme cases of rickets seen are almost invariably in one of these nationalities. It is exceptional to see in a dispensary or hospital a child of either of these races who does not show, to a greater or less degree, the signs of rickets. These two southern races seem to bear very badly the climate and the confined life of the northern cities. So far as our observations are concerned, there is no peculiarity in the food of these people which explains the prevalence of rickets among them, and it must be attributed to a race peculiarity. In the country, the immunity from rickets may be partly due to the more prevalent custom of maternal nursing, and partly to the better surroundings, the increased resistance of the children rendering them much less susceptible to unwholesome influences than children in the cities. Among dispensary and hospital patients of our large cities rickets is exceedingly common, and is seen chiefly in the foreign elements of the population.

Season.—This apparently has an important influence upon the development of the disease. The figures from four large outpatient clinics show that from January to June there were treated more than twice as many rachitic patients as from July to December. Schmorl has reported that he found early cases at autopsy rather more commonly in the cold months than in the warm, that the most active cases were considerably more frequent in the cold months, and that the vast majority of cases with evidences of healing were seen in the summer and early fall. The active symptoms of rickets are more frequently seen and are more severe in the winter and spring. What it is that determines this we are as yet quite unable to say.

Heredity.—The influence of heredity is difficult to determine. It is believed by some excellent authorities to be a factor in the production of the disease. Siegert has reported numerous instances where children with rachitic parents developed rickets while other children of non-rachitic parents living in the same environment and receiving the same

food did not develop rickets. Elgood has given the history of a woman who was married three times. By her first and third husbands, who had not had rickets, she bore children who remained free from the disease, while by her second husband, who had suffered from rickets, she bore five children, all of whom developed rickets. There seems to be no greater reason for denying the influence of heredity in rickets than there is in arteriosclerosis or tuberculosis.

Previous Disease.—Rickets not infrequently develops in syphilitic children; the connection, however, seems to be no closer than with any other cachexia. Chronic disorders of the digestive tract sometimes precede and often follow the development of rickets. It appears to develop quite independently of previous disease.

Rickets affects both sexes with equal frequency. The symptoms usually manifest themselves between the sixth and eighteenth months. Congenital and late rickets will be considered separately.

Experimental Rickets.—Rickets is never found in wild animals; in those under domestication, especially with in-breeding, it is by no means unusual. In zoölogical gardens it is quite prevalent. It would appear easy, therefore, to produce rickets, but the attempts have been almost always unsuccessful. By depriving animals of calcium and phosphorus severe lesions of the bones have been produced, enlarged epiphyses, bending of the bones and even fractures, but the condition is an osteoporosis and not rickets.

By bacterial inoculation Morpurgo produced true rickets in white rats. Findlay restricted the activity of puppies and saw rickets develop. Klose and Matti claim that true rickets results in dogs from early thymus extirpation. It is undoubtedly true that rickets did follow some of their operations, but that it was due to the removal of the thymus seems open to question.

Pathology.—Rickets is a disorder of nutrition, the result of some disturbance of metabolism in which calcium plays a very important rôle. The exact nature of this disturbance is not yet understood. Three theories have been advanced in explanation of the deficiency of calcium in the bones, which is the most striking characteristic of the disease. The first one, that rickets is due to a lack of calcium in the food, is not supported either by clinical or experimental evidence. The second theory is that the disease is due to an increased excretion of calcium as a result of disturbances of digestion. It is very likely that the increased excretion of calcium occurs only in rachitic children. Diet alone or disturbed chemical processes are not sufficient to account for it. The third theory advanced is that although sufficient calcium is furnished in the food, it is excreted in excess because the bones are incapable of absorbing it. This is the theory that has the most clinical and experimental evi-

dence in its favor; though what produces the incapacity of the bone to retain calcium is quite unknown.

Lesions.—The only constant and characteristic lesions of rickets are found in the bones; these changes are sufficiently definite to give it a place as an essential disease. One of the most striking features of rachitic bones is their unnatural flexibility. This is due to the lack of mineral salts in the bones and especially to the lack of calcium. Normally bone contains about one-third organic and two-thirds inorganic matter. In marked rickets the proportions are reversed, the bones often containing twice as much organic as inorganic matter. While all the inorganic elements are actually diminished the phosphorus and magnesium may be relatively increased. The chief loss is in the calcium.

The changes in the shafts and flat bones are universal. Those at the epiphyses show a marked parallelism with the activity of growth. Where growth is most rapid the lesions are most advanced. The middle ribs are earliest and chiefly affected, then the other ribs and the lower femoral epiphyses, the lower extremities of the radius and tibia, and eventually in some cases all the long bones, including the metacarpal and the phalanges. There are characteristic changes in form. The most constant is enlargement at the epiphyses, which is most strikingly seen at the lower extremities of the radius and tibia and at the costochondral junction of the middle ribs. All the sharp angles, borders and prominences of the bones are effaced. The curvatures of rachitic bones are allowed by the increased flexibility due to the loss of mineral salts. They may be due to a variety of causes. Some are simply an exaggeration of the normal curves much increased by the swelling of the epiphyses; others are due to muscular action, to atmospheric pressure, to some unnatural posture, such as the cross-legged position, to the weight of the limbs or the weight of the body. Marked deformity is usually due to displacement of the epiphysis or to fracture. Displacement of the epiphyses is rare except in the ribs, where it occurs to a certain extent in every advanced case. Fractures of the long bones are very common. The bones most frequently broken are the radius and ulna; next in order the ribs, humerus, femur, fibula and clavicle. The fractures are usually of the green-stick variety with more or less impaction and are generally followed by the production of considerable callus, though subperiosteal solution of continuity is occasionally found with no deformity and little if any callus. When bending occurs there is a production of new tissue beneath the periosteum to compensate for the mechanical disadvantage of position in which the new bone is placed. The shafts are frequently greatly thickened. The principal change in the form of the flat bones consists in the production of large bosses or prominences upon the parietal and frontal bones, due to an increase of vascular, immature

A

COSTOCHONDRAL JUNCTION IN EARLY RICKETS. (a) cartilage, (b) rib, (c) irregular epiphyseal line, (d) invasion of cartilage by loops of vessels growing up from marrow cavity of rib at points unprotected by calcified cartilage, (e) cartilage protected by calcification.

B

NORMAL COSTOCHONDRAL JUNCTION. (a) cartilage, (b) rib, (c) epiphyseal line, (d) columns of calcified intercellular cartilage which at (e) have been surrounded by bone and converted into trabeculae. The epiphyseal line is quite regular and sharply separates bone from cartilage.

bone beneath the periosteum. Bosses are found where the normal bending produces the greatest stress upon the bone. The deficiency in calcium over areas in the occipital bone that are thin even under normal conditions, allows them to indent under the finger. This is craniotabes.

In a longitudinal section of one of the long bones the principal change seen at the extremity is that the cartilaginous layer which unites the epiphysis and the shaft is very much enlarged both in width and thickness, the latter being sometimes four or five times the normal. The transitional zone is a whitish or bluish-white color, rather softer than normal cartilage. On one side it blends with the cartilage of the epiphysis, on the other it presents an irregular dentated border. The normal red marrow may cease a quarter or half an inch from the epiphysis, its place being taken by a light gray or whitish layer that microscopically is seen to be fibrous tissue. The replacement of so much marrow is perhaps the reason for some of the anemia that is prominent in severe rickets. The epiphyseal centers of ossification are but slightly affected.

In the process of healing the epiphyseal swellings slowly diminish in size and may quite disappear; the slight curvatures may be entirely overcome and the greater ones much lessened. Some of the long bones remain more or less permanently thickened and with a denser and thicker cortical layer. The beading of the ribs becomes almost imperceptible; the bosses upon the skull shrink very markedly and may leave scarcely a trace of their existence. In most cases except in Italians and negroes the active process in rickets comes to an end by the time the child is two and a half years old, often at two years.

Microscopical Appearances.—When normal conditions obtain at the epiphyses, the cartilaginous intercellular substance between the lowest of the four layers of cartilage cells becomes infiltrated with calcium, forming rigid columns. These direct the vessels budding up from the marrow against the cartilage cells which are then destroyed by erosion. The columns themselves are partly consumed by osteoblasts but the remains of them act as the centers around which bone is formed by osteoblastic activity. The new bone is first formed as osteoid tissue, which differs from mature bone only in its containing no calcium. When it absorbs calcium it becomes true bone. It absorbs calcium so soon after its formation that only a narrow layer of osteoid tissue is ever found in health. Marrow cells accompany the capillary loops. The cartilage itself is nourished by vessels that spring from the perichondrium and run transversely in the so-called cartilage canals. Throughout the whole skeleton all the bone is well calcified with the exception of the narrow zone of osteoid tissue.

In rickets the most striking feature is the presence of large amounts of limeless bone, or osteoid, throughout the whole skeleton. It is more marked in some situations than others but it is a universal process. At

the epiphyses the calcium is also absent from the intercellular ground substance. The marrow vessels are not directed against the cells but they grow in all directions, breaking up the normal contour of the epiphyseal line. Some of the cartilage grows down undisturbed, or islands of cartilage cells are formed and not destroyed. The cartilage is not formed in excess. It is found in excess because it is allowed to remain. The transitional zone, or "metaphysis," is weakened and nature attempts to remedy this by the production of fibrous tissue and osteoid tissue. In this way the metaphysis is increased greatly in diameter and also in thickness; for, on account of its inelasticity, it expands laterally as the result of muscular action or weight and does not return to its former position. Vascularization of the metaphysis is accomplished by a persistence of the cartilage canals.

When healing takes place the osteoid tissue in the flat bones and the shafts of the long bones absorbs calcium, and the transformation into normal bone is rapidly completed. At the epiphysis the first step is the deposition of calcium in the cartilage on the epiphyseal side of the last cartilage canals persisting in the metaphysis. Vessels from these bud back and destroy the cartilage. The metaphysis is thus protected from a further production of cartilage. That which remains is gradually disintegrated and normal bone takes the place of the osteoid tissue and connective tissue. There is no anatomical explanation of the deficient growth which is occasionally encountered. It must result from permanent damage to the function of the proliferating zone of cartilage cells.

Healing is not always a continuous process. Relapses of the disease occur. As proof of this lines of calcification may be found buried in the rachitic zone. Two and occasionally three of these are encountered. They represent abortive attempts at healing.

Visceral Lesions.—These are not infrequent, but are not essential to rickets. In the lungs they are due to deformities of the chest wall and to complications. Beneath the deep lateral furrows which are so common, there is found a part of the lung in a state of more or less complete collapse. This is accompanied by emphysema of the portion just anterior to it. Acute and chronic bronchitis and bronchopneumonia are exceedingly frequent. A low grade of chronic catarrhal inflammation of the stomach and intestines is common, and is often associated with dilatation of these organs. The spleen is enlarged in most cases during the period of active symptoms. This is usually moderate in degree. The swelling of the spleen is chiefly due to simple hyperplasia. Enlargement of the liver is less frequent, and may occur with or without that of the spleen. There are no constant changes in the structure of these organs. The lymph nodes are frequently enlarged. This is due to simple hyperplasia, and has no close connection with rickets. Cerebral

changes are rare, and those described are rather of accidental occurrence than dependent upon the rachitic process. As stated under Symptoms, enlargement of the head is usually due to thickening of the cranial bones. Although hydrocephalus is occasionally seen, it is extremely doubtful whether it is more frequent than in patients not rachitic. Hypertrophy of the brain has been described in connection with rickets, but as yet this does not seem to be established by sufficient pathological evidence. The muscles are flabby from imperfect nutrition, and sometimes atrophied

FIG. 20.—COSTOCHONDRAL JUNCTION IN MARKED RICKETS. (A) cartilage, (B) rib, (C) masses of cartilage cells, (D) metaphysis or transitional zone, composed of masses of cartilage cells, osteoid tissue, blood vessels and fibrous tissue. Normal marrow in this zone is absent.—Note that the epiphyseal line no longer exists.

from disuse, but no essential anatomical changes have been demonstrated in them.

Symptoms.—The symptoms upon which a diagnosis of rickets can be based are chiefly bony symptoms. Lesions of the bones must exist some weeks before they reach a degree that can be recognized clinically. Schmorl has found microscopical evidences of rickets as early as the end of the second month. In the clinic we seldom see unmistakable rickets before the fourth or fifth month. A well-marked case of rickets makes a striking picture (Plate III), and one not easily mistaken.

There are seen the large head, beaded ribs, narrow chest, prominent abdomen, symmetrical swellings of the epiphyses of the wrists and ankles, and curvatures of the extremities. The beginning of symptoms is nearly always insidious, and the patient does not usually come under observation until they have existed for several weeks, often several months.

Early Symptoms.—The most constant early symptoms are sweating of the head, extreme restlessness at night, constipation, beading of the ribs, and craniotabes. The head-sweating is rarely absent, and may continue for several months. It is especially profuse during sleep, the perspiration standing out in large drops upon the forehead, often being sufficient to wet the pillow. This is one of the causes of the nasal and bronchial catarrhs so common in rachitic infants. There is marked restlessness during sleep: the children tossing about their cribs, kicking off the clothes, and never having the quiet, natural slumber of healthy infants. This may be due to many causes, but when persistent and associated with marked perspiration of the head, rickets should be suspected. In many rachitic infants serious nervous symptoms may be seen due to associated tetany, such as laryngismus stridulus, and general convulsions. Constipation is frequently seen as an early symptom, although it is more marked in the later stages of the disease.

The beading of the ribs is almost invariably the first appreciable change in the bones, and it is well-nigh constant. This forms the so-called "rachitic rosary," consisting of nodules at the line of junction of the costal cartilages and the ribs. It may be slight, or there may be a row of knobs as large as small marbles. In many cases with marked thoracic deformity, little or no beading of the ribs is seen externally, although at autopsy it is found to be very marked upon the internal surface of the chest. The costochondral junctions of newly-born infants, especially the more vigorous ones, are readily palpable. Care should be taken not to confound these with the rachitic rosary which appears only after several months. In infants under six months there may be found soft spots in the cranium, usually over the occipital or posterior portions of the parietal bones. These are from one-fourth to one inch in diameter, and there are usually several of them present. By pressure with the finger they give a sort of parchment-crackling sensation. This condition is known as craniotabes. Craniotabes is a rachitic manifestation and depends in no wise upon syphilis. A rachitic cachexia is not usually present until the symptoms have existed for several months, and in many cases it is not seen at all.

Deformities.—The deformities of rickets are almost invariably symmetrical in character, and usually numerous. In extreme cases almost every bone in the body is affected.

Head.—This usually appears to be too large, and although it may not

TYPICAL RICKETS

Showing the large head, narrow chest, prominent abdomen, marked enlargement of the epiphyses at the wrists and ankles. There are also curvatures of the forearms and legs which are not so well shown.

The patient a child two and a half years old.

be greater in circumference than that of a healthy child of the same age, it is out of proportion to the rest of the body. In marked cases the increase in circumference may be one or two inches. The enlargement is chiefly due to thickening of the cranial bones. In one case with marked deformity, we found the skull over the parietal bones half an inch in thickness (Fig. 21). This thickening diminishes with recovery, but in most cases the head remains throughout life larger than it should be.

Fig. 21.—Rachitic Skull. From colored child two years old, horizontal section, inner surface; showing thickening of the bones, especially the frontal, and open fontanel.

The shape of the rachitic head is somewhat square (Fig. 22), owing to the formation of large bosses over the parietal and frontal eminences. It is flattened at the occiput from pressure, and flattened also at the vertex. In extreme cases, the prominences upon the frontal and parietal bones may be so great as to produce quite a marked furrow along the line of the sagittal and frontal sutures, and one at right angles to this along the coronal suture (Fig. 23). This condition gives unusual prominence to the forehead. Marked deformity of the head has been observed in about one-third of our cases. The sutures may remain open for an

unnatural time, occasionally until the end of the first year. The fontanel is late in closing, being frequently found open at two and a half, and sometimes even at three years. Often at eighteen or twenty months the fontanel is two inches in diameter. The veins of the scalp are often prominent, and the hair is frequently worn from the occiput, owing to restlessness during sleep. Occasionally rickets and hydrocephalus are associated, but the association is accidental.

FIG. 22.—RACHITIC HEAD. Italian child two years old; square, prominent forehead and flat vertex.

Chest.—Beading of the ribs has already been mentioned. This is the most characteristic feature, but in the majority of cases there are, in addition, lateral depressions over the lower third of the chest, at the line of junction of the cartilages with the ribs, with eversion of the lower border of the ribs. In severe cases these depressions or furrows are so great as

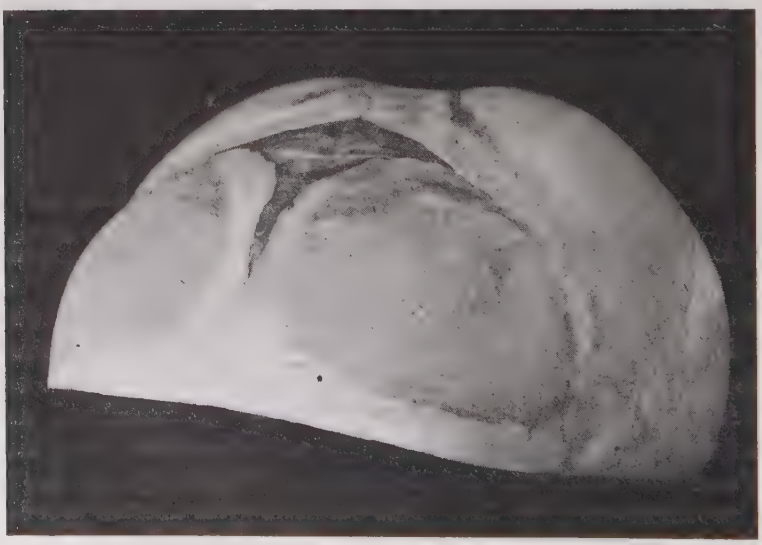


FIG. 23.—RACHITIC SKULL FROM A CHILD ONE YEAR OLD. Showing frontal and parietal bosses and wide fontanel.

to cause serious deformity (Plate IV). Usually there is a great diminution in the transverse, and an increase in the anteroposterior, diameter of the chest. Fig. 24 shows the outline of the chest of a rachitic child of two years, compared with that of a healthy child of the same age. Another frequent deformity is the "rachitic girdle," which consists in a transverse depression about two inches broad, extending from one side of the chest to the other, a short distance above its lower border. The chest wall yields at the attachment of the diaphragm which becomes more nearly horizontal. As a result of this the liver becomes somewhat displaced downward. Marked thoracic deformity was seen in about twenty per cent of our cases, but in only a small proportion was the chest normal.

The factors in the production of the thoracic deformity are the con-

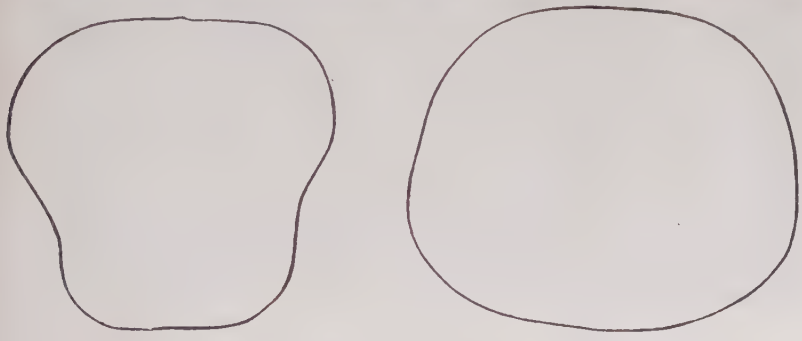


FIG. 24.—A, HORIZONTAL SECTION OF A RACHITIC CHEST, child two years old, showing lateral furrows; B, SECTION OF CHEST OF HEALTHY CHILD OF THE SAME AGE.

traction of the diaphragm, atmospheric pressure, and soft chest walls, these yielding at the point where they have least resistance, viz., at the junction of the costal cartilages and the ribs. The swelling of the costochondral junction, which is much accentuated by the displacement of the cartilages on the ribs, limits to a marked degree the capacity of the thorax. When there exists any obstruction to the entrance of air, as with bronchitis, hypertrophied tonsils, or adenoid growths of the pharynx, the thoracic deformities are exaggerated. Irregular chest deformities depend upon the co-existence of pathological conditions in the lungs. Pigeon-breast is occasionally seen, but it is doubtful if this depends upon rickets alone.

Spine.—In very many of the milder cases this is normal. The most characteristic deformity consists in a posterior curve (kyphosis), which is a general one, usually extending from the mid-dorsal to the sacral region (Fig. 25). This existed in nearly half of our cases. In the early part of the disease it disappears entirely on suspending the

child, or making extension upon the extremities: but in cases of long standing it may not disappear entirely by these tests. Very much less frequently there is seen a rotary curvature. This, in our experience, has been more frequently with the convexity to the left side than to the right—the opposite of the common form of lateral curvature seen in young girls. Marked lateral curvature in children under three years is usually rachitic.

The clavicle is affected only in severe cases. The usual deformity consists in an exaggeration of the anterior curve at the inner third of the bone, which is somewhat shortened and its extremities enlarged. It is not infrequently the seat of green-stick fracture.

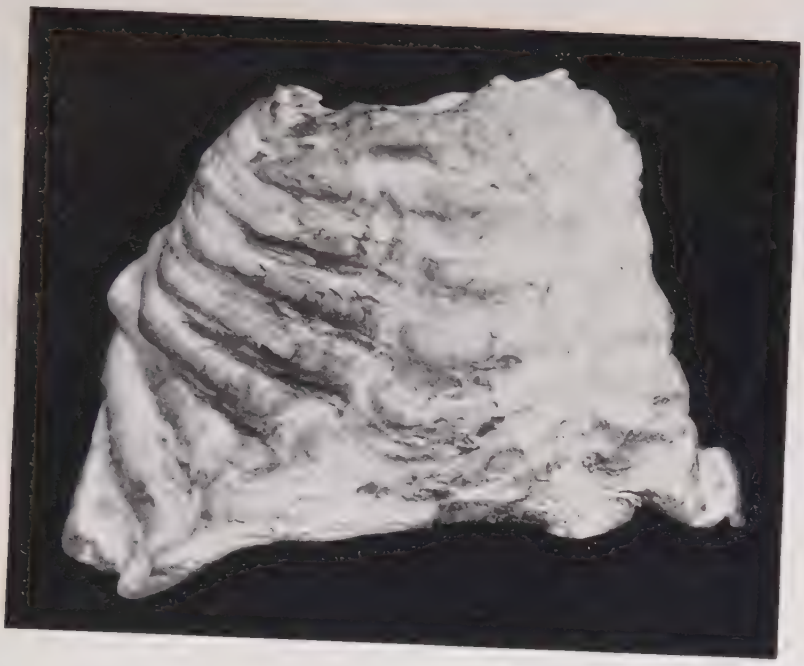
FIG. 25.—RACHITIC CURVATURE OF THE SPINE.

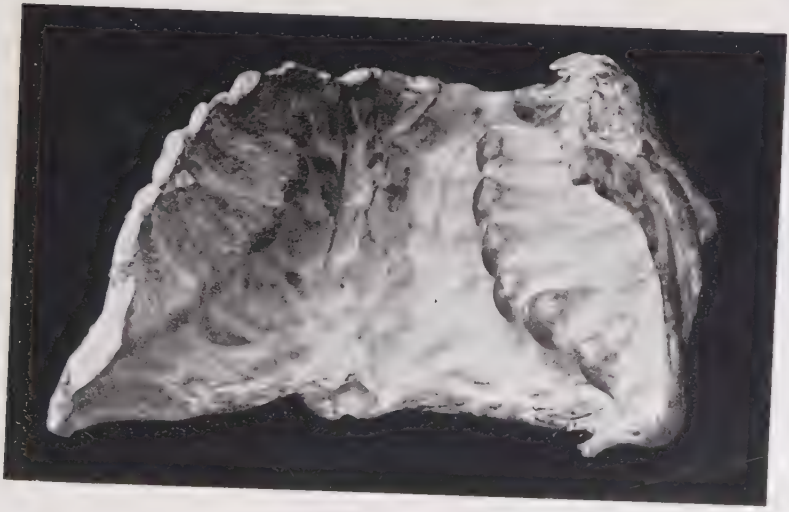
Deformities of the pelvis belong to obstetrics rather than to pediatrics. The most common rachitic change is a diminution of the anteroposterior diameter and a narrowing of the subpubic arch.

Extremities.—Deformities of the upper extremities are usually symmetrical. The humerus is affected only in severe cases. It has a forward and outward curve, although rarely a very marked one. Both the epiphyses are enlarged, although the upper one can not well be made out unless the child is very thin. The radius and ulna are frequently affected. They present a convexity upon their extensor surfaces, which in some cases is very marked, particularly in children who have been creeping. Green-stick fractures are quite frequent here as they are also in the femora. They are frequently multiple and occur from very slight causes, sometimes apparently from muscular contraction. Multiple fractures may be found with no separation, the periosteum apparently still remaining intact. They are frequently found in the fibula. Rachitic changes at the epiphyses are more common than in the shaft, enlargement of the epiphyses at the wrist being one of the most constant bony deformities of rickets (Plate III). Less frequently similar swellings are seen at the elbow. Enlargement of the ends of the metacarpal bones or the phalanges we have seen but seldom and only in extreme cases.

The lower extremities are rather more frequently affected than the upper, but in a similar way. The femur is involved only in severe cases; it commonly presents a general forward and outward curve, which is mainly due to the weight of the legs as the child sits. Occasionally there is also an outward rotation of the femur, when children

PLATE IV





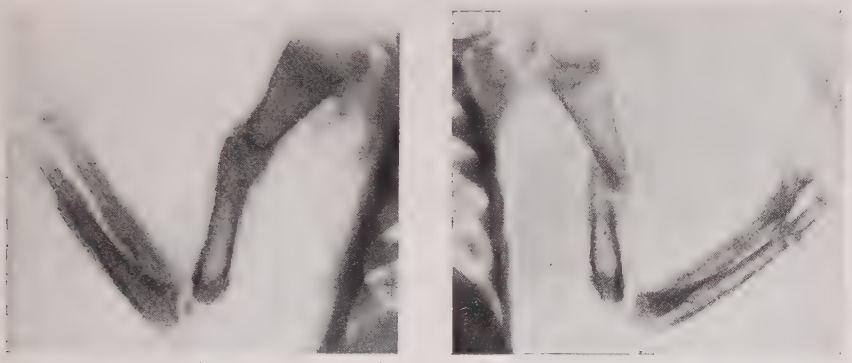
Deformity of the Chest in Severe Rickets

In the upper picture, giving the external view, is shown a deep oblique furrow at the junction of the ribs and costal cartilages, these meeting at an acute angle.

In the lower picture the ribs have been separated from the spine and spread open, showing the same deformity as it appears from within, looking forwards.

From a colored child ten months old.

have been allowed to sit much in a cross-legged posture. When such children begin to walk, the toes are turned very far outward. The principal deformities of the lower extremity are bow-legs and knock-knees. Knock-knees are more common in females, and are believed to be due to an overgrowth of the inner condyle of the femur. Enlargement of both condyles can be demonstrated in most of the marked cases of rickets. The cases of slight bow-legs may be due simply to swelling of the epiphyses, the shaft of the bone being quite normal. This point we have verified by post-mortem observations. Such are probably most of the deformities which disappear spontaneously. The most severe cases of bow-legs are often associated with some degree of antero-posterior curvature, and the latter may be the principal deformity.



A B

FIG. 26.—MULTIPLE FRACTURES IN RICKETS. Showing both arms of the same patient; fractures also of both femora.

Enlargement of the epiphyses at the ankles is usually present when it is seen at the wrists, and nearly to the same degree. Enlargement of the upper epiphyses of the tibia and the fibula is seen only in severe cases. The cause of the deformities of the leg is not, primarily, at least, walking too early, since they are common in children who have never walked; slight deformities, however, may be aggravated by early walking. A change which has not been sufficiently emphasized is the arrested growth of the long bones; this is one of the most characteristic features of rickets. A rachitic child of three years often measures in height four or five inches less than a healthy child of the same age, the difference being almost entirely in the lower extremities.

All the ligaments, but particularly those about the large joints, are lax and frequently elongated. This may lead to the deformity known as weak ankles, or to an over-extension at the knee (*genu recurvatum*); also to unnatural mobility at the hips, shoulders, elbows, or wrists. The

condition of the ligaments plays an important part in the production of spinal deformities.

Muscles.—The muscular symptoms of rickets are almost as constant and as characteristic as those of the bones. The muscles are small, very flabby, and poorly developed; hence rachitic children are unable to sit erect, or to stand or walk at the usual age. Of one hundred and fifty-one cases in which the date of walking alone was investigated, only twenty-seven, or eighteen per cent, walked before the fifteenth month; forty-seven per cent were not walking at the eighteenth month; twenty per cent, not at two years; and ten per cent, not at two and a half years. Late walking is one of the most common symptoms for which advice is sought by parents with rachitic children. The muscular power in the extremities is sometimes so feeble as to suggest paralysis. We have seen a number of cases in which the symptoms so resembled paralysis, that even expert diagnosticians were unable to differentiate rickets from poliomyelitis except by the electrical reactions, those in rickets being usually normal or exaggerated. In other cases the symptoms may suggest cerebral palsy of the flaccid type. The muscular symptoms may be marked when the bony changes are slight, and conversely. As no lesions of the muscles have been demonstrated, the symptoms are probably due to imperfect nutrition. Two other symptoms depend chiefly upon the condition of the muscles, viz., pot-belly and constipation.

Pot-belly is quite an early symptom, and in most cases a very marked one (Plate III). It was noted in sixty per cent of our cases. The enlargement of the abdomen is uniform. It is everywhere tympanitic, and it may be as tense as a drumhead. It is due to a loss of tone in the abdominal muscles, and in the muscular walls of the stomach and intestine. It is aggravated by chronic indigestion and excessive intestinal fermentation. The enlargement is thus mainly from tympanites. There may be a marked degree of dilatation both of the stomach and the colon. To a very small degree only, does the large abdomen depend upon swelling of the liver or spleen.

The constipation of rickets, as already suggested, depends upon the loss of tone in the muscular walls of the intestines. It may alternate with diarrhea. It rarely happens that a rachitic child has habitually normal evacuations from the bowels. Hard, dry, constipated stools frequently set up a condition of chronic catarrh of the colon in which large masses of mucus are discharged.

Fever.—According to some observers there is a febrile movement which belongs to the active stage of rickets, but we have never been able to satisfy ourselves of the truth of this observation.

Dentition.—As a rule, dentition is late and apt to be difficult, i. e., it is associated with attacks of indigestion or other disturbances which

may be serious. Individual cases, however, present great variation in regard to this symptom. A study of the progress of dentition in one hundred and fifty rachitic children gave the following results: in fifty per cent the first teeth were cut on or before the eighth month; twenty per cent of the cases had no teeth at twelve months, and in eight per cent none had appeared at fifteen months. Even though the first teeth come at the usual time, the progress of dentition is usually retarded by the development of rickets. The character of the teeth in rickets is usually good. This is in striking contrast to hereditary syphilis, where the tendency to early decay is constantly seen.

General Appearance.—Children suffering from marked rickets are almost always anemic. The majority are fat and flabby. The tissues are soft and have but little resistance. Rarely, they may be thin, like patients suffering from marasmus.

Rachitic patients are very prone to suffer from hypertrophied tonsils, adenoid growths of the pharynx, and enlargements of the lymph nodes of the neck. In all forms of acute illness the feeble resistance of these patients is very evident. This is especially true in acute disease of the lungs.

The mucous membranes are very vulnerable in all rachitic patients. From the slightest indiscretion in diet an attack of acute indigestion or diarrhea may be brought on, and from a very insignificant exposure, catarrhal inflammation of the upper or lower air passages is excited. In rachitic patients all such attacks are prone to run a protracted course. Inflammation of the trachea and larger bronchi is likely to extend to the smaller bronchi and the lungs.

The downward displacement of the liver and spleen from contraction of the chest should not be mistaken for enlargement of these organs. Moderate enlargement of the spleen is very common during the stage of most active symptoms, i. e., from the sixth to the twelfth month. Great enlargement of either liver or spleen is infrequent.

Blood.—Anemia is present in most of the marked cases, its intensity varying with the severity of the rachitic process. The blood picture is usually that of an ordinary secondary anemia. Leucocytosis is often present; it is more marked in cases accompanied by an enlarged spleen.

Nervous Symptoms.—These are among the most frequent manifestations of rickets. Restlessness at night has already been mentioned as a prominent early symptom. Pain and tenderness are rare. A disposition to muscular spasm is seen in many cases. There may be laryngismus stridulus, general convulsions or other manifestations of tetany. It was formerly believed that rickets was the cause of the convulsions. It seems now apparent that it is the associated tetany which is intimately dependent upon rickets. The clinical evidences

of rickets may be very slight yet the nervous symptoms be very marked.

Calcium Metabolism.—Owing to the remissions and relapses that occur in rickets and the impossibility of determining whether the disease is active or not, it has been a difficult matter to study the calcium metabolism of rickets. The experiments of Schabad show plainly that in early cases either the retention of calcium is very low or there is an actual loss. In older cases there may still be a diminished retention or it may be nearly normal, depending upon the stage of the disease. In convalescence the retention is two or three times the normal.

Course and Termination.—Rickets is essentially a chronic disease, and its course is measured by months. The active symptoms in most cases continue from three to fifteen months, being interrupted from time to time by remissions, but these are seldom appreciated clinically.

The earliest symptoms of improvement are a diminution in the nervous symptoms, especially in the restlessness at night; increased muscular power, as shown by a disposition to stand or walk; diminution in the head-sweats; disappearance of the craniotabes; and improvement in the anemia. The changes in the deformities are very slow, and from month to month almost imperceptible. When improvement once begins, however, it usually goes steadily forward.

Congenital Rickets.—In the middle of the last century, all bone abnormalities apparent at birth were believed to be due to fetal rickets. Further investigation has shown that most of them were examples of chondrodystrophy or osteogenesis imperfecta. Kassowitz and more recently others have maintained that rickets is usually, if not always, congenital in origin. More careful clinical observation and especially pathological studies have shown, however, that evidences of rickets are not to be found at birth. There is probably no such condition as fetal rickets.

Late Rickets.—Rare instances have been reported of bony deformities in all respects like those of rickets, developing in children from six to twelve years old. The course is slow and the deformity frequently extreme. A number of cases studied microscopically by such authorities as Schmorl and Schmidt leave no room for doubt as to the existence of the condition. It is very unusual in this country. We have never seen a case.

Acute Rickets.—Although from time to time cases have been reported with this title, from a study of the histories it is clear that the great majority, if not all of them, were cases of infantile scurvy. It is doubtful whether, strictly speaking, there is such a thing as acute rickets.

Diagnosis.—The diagnosis of rickets is not usually difficult. The most important early symptoms for diagnosis are sweating of the head, craniotabes, great restlessness at night, delayed dentition, and enlarged

fontanel. Each of these, taken separately, may mean something else, but collectively they can mean nothing but rickets. In the later stages some of the characteristic deformities are usually present; the most constant are beading of the ribs, enlargement of the epiphyses of the wrists and ankles, and bow-legs.

Special symptoms, when unusually prominent, may give rise to difficulty in diagnosis. The enlargement of the head may be mistaken for hydrocephalus. The delayed dentition and large fontanel of the cretin may be mistaken for rickets. Muscular weakness may be so great, especially when affecting the legs, as to make it easy to mistake a rachitic pseudo-paralysis for actual paralysis due to a cerebral or spinal lesion. When walking is much delayed, rickets may be passed over as simple backwardness. In nearly all of the last-mentioned group of cases the diagnosis may be established by a careful search for the bony changes, and by the fact that in rickets there is only a general weakness of all the muscles, and not actual paralysis of any limb or group of muscles. The greatest difficulty is usually found when the muscular symptoms are marked and the bony changes slight, as is not infrequently the case. Here the question is, whether rickets is sufficient to explain all the symptoms, or whether in addition some other condition is present. The electrical reactions will usually decide the question of poliomyelitis, while the presence of cerebral symptoms, exaggerated knee-jerks, and rigidity of the legs, will usually mark infantile cerebral paralysis. The bony enlargements of syphilis may be confounded with those of rickets. The bony changes of early syphilis, although affecting the epiphyses are seen at an earlier age and are generally accompanied by pain and tenderness, sometimes by epiphyseal separation, none of which are seen in rickets. The bony changes of late syphilis affect the shaft rather than the extremities of the long bones; when the bone is enlarged near the joint it is usually upon one side only. In syphilis there may be necrosis, while in rickets breaking down of bone is never seen. From scurvy, rickets is differentiated by the absence of marked hyperesthesia, ecchymoses, and other hemorrhages, the changes in the gums, and most of all by the fact that anti-scorbutic diet produces no immediate change in the symptoms. The diagnosis of rachitic curvature of the spine from vertebral caries will be considered in connection with the latter disease.

Prognosis.—Rickets *per se* is seldom, if ever, a cause of death. It is, however, a large factor in the mortality of the first two years, as it predisposes strongly to many forms of acute disease. It is an important etiological factor in certain serious nervous conditions, especially tetany. Rickets adds very greatly to the danger from all acute diseases of infancy, particularly those of the respiratory tract. The encroachment upon the capacity of the lungs by a marked thoracic deformity,

may in itself be enough to keep a child in a delicate condition and retard its growth. At the same time such a condition is a constant invitation to acute attacks of bronchitis or pneumonia. The effect of rickets upon the future health of the child depends chiefly upon the presence and extent of the thoracic deformity. When this is severe, the child usually succumbs to some acute respiratory disease during the first few years of life. When this is absent, although children may remain somewhat dwarfed on account of their short legs, in other respects they may be as well as if they had never been the subjects of rickets.

Treatment.—In considering the treatment of rickets, the natural course of the disease is to be kept in mind, viz., that active symptoms frequently continue only until the end of the first year, rarely longer than the eighteenth or twentieth month. The most important period for treatment, therefore, and the one in which it is most effective, is from the sixth to the eighteenth month. The earlier the treatment is begun the better will be its results. General treatment after the eighteenth month, has very little effect upon the disease, for by this time most of the harm has been done. The course of the disease when untreated is toward spontaneous recovery. Most of the cases seen in private practice are of a mild type and recover without special treatment, often no diagnosis being made until later in life, when the bony deformities or stunted growth indicate the previous existence of rickets.

Diet.—The most frequent dietetic error in rachitic patients being an excess of carbohydrates and an insufficient supply of fat, it follows that condensed milk, proprietary infant foods, and large amounts of farinaceous foods of every description should be stopped. A suitably modified cow's milk should be substituted or for young infants a wet-nurse should be secured. But supplementary feeding of cow's milk should be given so as to insure a sufficient supply of calcium. As soon as possible other food, such as thick gruels, scraped meat, fruit juices or stewed fruit, should be offered and vegetable soups from which the vegetables have been strained out or in which they are very finely divided. Most infants are eight to ten months old before rachitic symptoms are observed; to them the above mentioned articles of diet may be given almost immediately unless digestive symptoms are marked. Breast feeding should be interrupted. Cream is often badly borne and some other form of fat must be substituted. The fat of crisp bacon upon stale bread or zwieback serves well. The change to solid food should be made earlier than with normal children, and not more than a pint of milk should be allowed a day.

Hygiene.—In large cities it is almost impossible to secure for rachitic patients the surroundings they require. Whenever possible, such chil-

dren should be sent to the country; but when this is out of the question, much may be accomplished by frequent excursions upon the water or into the country, by keeping children as much as possible in the parks and open squares of the city, and securing plenty of fresh air in sleeping rooms. Cold sponge-baths given every morning, do much to lessen their susceptibility to rhinopharyngitis and bronchitis. Sunshine, though difficult to obtain in large cities, is a most efficient therapeutic agent. The establishment of suburban hospitals and homes for these cases would do much to lessen the mortality from rickets.

Medicinal treatment.—In a disease which tends so uniformly to recovery when causal conditions are removed, it is difficult to estimate, by clinical observation, the real value of medicinal treatment. Arsenic and iron are valuable in the treatment of rickets, the special indication for their use being the presence of marked anemia. Profuse sweating may be relieved by small doses of atropin, i. e., gr. 1/800, three or four times a day, to a child of six months. The special remedies most used are cod-liver oil, phosphorus, and preparations of calcium.

Various preparations of calcium have long been employed with the belief that they could supply lime to the tissues. It is now practically certain that calcium is present in sufficient quantity in the blood. It cannot be utilized. Calcium, therefore, in active rickets has no value. In convalescence, during the stage of extreme calcium retention, it may be of assistance. It may be offered in the form of acetate or lactate. The two important remedies for rickets are cod-liver oil and phosphorus. No remedy for rickets has held its place so long as has cod-liver oil. Phosphorus, popularized in the treatment of this disease by Kassowitz, has also some value; its most striking results are seen in the early cases and when nervous symptoms are marked. The best results are obtained by a combination of these two remedies. The officinal oil of phosphorus is used in combination with cod-liver oil, gr. 1/300 to 1/200 is given three times a day with one-half dram to one dram of the oil. Striking confirmation of the clinical observations regarding the value of this combination is furnished by the metabolism experiments of Schabad who found the percentage of calcium retention greatly increased by the use of cod-liver oil and phosphorus.

Treatment of the Rachitic Deformities.—The deformities of the chest are less amenable to treatment than are most of the others. After the third year something can be done by gymnastics to develop the chest muscles and to increase the pulmonary expansion.

The deformity of the spine (kyphosis) may usually be overcome by postural treatment. The patient should lie upon a hard bed; no pillow should be allowed under the head, but in severe cases one should be placed beneath the back, so that the head and buttocks are slightly lower

than the lumbar spine. While sitting, the shoulders should be kept back and the trunk supported. For a few minutes each day the child should be placed upon the face, and the deformity overcome by raising the buttocks while pressure is made upon the spine. In severe cases, an apparatus for giving spinal support, either by a steel brace or a plaster-of-Paris jacket, may be worn a few hours each day when the child is sitting up. Other means should be employed, especially friction and massage, to develop the spinal muscles.

In very many cases slight deformities of the extremities are outgrown when the general treatment can be properly carried out. If the deformity is not great and not increasing, it is safe to continue with general treatment only. If the deformity is marked or if it increases in spite of the constitutional treatment, orthopedic apparatus should be applied. Something may be done toward straightening the bones by intelligent manipulation. Walking should be discouraged until the bones are quite firm. Friction of the extremities and massage will do very much to increase muscular development. The habit of sitting cross-legged—a very common one in rachitic children—should be prevented, and in fact any other habitual posture, on account of the danger of increasing certain deformities. But little is to be expected from the use of apparatus for the correction of rachitic deformities after the child is two and a half years old; since at this time, and often even at two years, the bones are so firm that no amount of pressure from a steel brace will have any effect.

Without going fully into the question of the surgical treatment of rachitic deformities, for which the reader is referred to text-books of general and orthopedic surgery, we will only state that osteotomy seems to us to offer decided advantages over the other means of treating severe deformities. The best results from osteotomy are obtained when the operation is delayed until the fourth or fifth year, by which time the bones are sufficiently firm and solid. Operations in the second year are generally unsatisfactory, and those in the third year often so, because of the bending of the bones which takes place subsequently. The deformities which require operation are bow-legs and knock-knees, less frequently the curvatures of the femur or the bones of the forearm.

CHAPTER VII

DIATHESES

The conception of constitutional differences is not a new one. It has been recognized for more than a hundred years that, under the same conditions, one person reacts physically in a different way from another,

and that this is especially true of infants. To explain this, a peculiarity of constitution has been assumed. Before the development of bacteriology this idea was generally accepted to explain such a condition as scrofula. When it became apparent that many of the symptoms of scrofula were, in reality, symptoms of tuberculosis, the conception was gradually given up. But in the last few years emphasis has again been laid upon variation in constitution and this has come into more and more prominence. It should be recognized, however, that the basis of a division into groups rests upon clinical symptoms only, and for this reason there have been great differences of opinion in regard to the limits of the various diatheses and what infants should be included in one or the other group. While many diatheses have been described, there are but two that stand out with sufficient clearness to justify their consideration as entities. These are the "exudative diathesis" of Czerny and the "neuropathic" or "psychoneuropathic diathesis."

THE EXUDATIVE DIATHESIS

This diathesis has been described under different names by many observers. It is the one which was first recognized. Many of the symptoms were formerly classed under the old name of "scrofulous" diathesis. But the symptoms which are now considered by Czerny to belong to the exudative diathesis depend in no way upon tuberculosis. They are manifested early in life and are largely confined to lesions of the skin and mucous membranes. Infants with this diathesis often show early seborrhea of the scalp, and they are particularly liable to eczema, which may develop upon the face alone or all over the body. They are usually well nourished, oftentimes very fat infants, but their musculature is usually flabby and there is almost always anemia of greater or less intensity. Depending upon the extent of the eczema, eosinophilia is present. Less commonly, in this country at least, the papules and lesions of lichen strophulus are formed. There is a marked tendency to rhinopharyngitis and as a result there is frequently otitis media. The superficial glands, especially those in the neighborhood of the lesions, are somewhat enlarged.

The general nutrition, as has been said, is usually fairly maintained, but when the eczema is severe the irritation from this and the consequent loss of sleep may seriously affect the infant's general condition.

Though chemical changes have been described in these children, there are none sufficiently striking to justify a diagnosis without clinical symptoms. There is a tendency to retention of chlorids, and an increased

sugar content of the blood has been claimed, but both of these are inconstant.

After the first year the manifestations of the exudative diathesis usually diminish in intensity; they are frequently absent after the second or third year, though they may remain in evidence for a longer period. There can be no doubt that giving a large amount of food increases the severity of the symptoms and that such children do better upon a restricted diet. Fat in excess in the diet increases the severity of the cutaneous symptoms and a diet of milk alone, after the first few months, aggravates the condition. It is wise to employ carbohydrates as early as possible. For this reason thick gruels should be used as diluents, even in the first few weeks, and the milk replaced by them as far as this can be done. By the eighth or ninth month, or even earlier, cereal may be given with a spoon once or twice a day. Thereafter, milk should form only a small part of the diet and throughout infancy and childhood the quantity of food should be regulated and restricted more particularly than with other children.

THE NEUROPATHIC DIATHESIS

The neuropathic child may give evidences of his peculiar constitution during infancy, or sometimes not until he is several years of age. No matter at what age the symptoms are manifest, it is usually inheritance that is responsible for the highly irritable nervous system of these children. In almost all instances one or both parents are neurotic. Environment plays distinctly a secondary part, but is important even in infancy.

The Neuropathic Infant.—The condition may reveal itself even in the first weeks of life in an unusually early reaction to sights and sounds. Infants may fix their attention upon people and objects as early as the third or fourth week, and thus are readily startled and terrified by things to which the normal infant pays no attention. Sleep is oftentimes disturbed. Such children are precocious and on this account often receive much attention from parents and nurses, which practice has a tendency greatly to increase their symptoms. Ordinarily, however, not much notice would be given to the abnormal constitution except for the development of two symptoms which are particularly striking. The first of these is vomiting and the second, diarrhea.

The vomiting is usually characterized by the fact that it takes place very readily without any apparent discomfort and that the simplest forms of food and even water may be vomited. Vomiting may develop without sufficient cause and the usual symptoms ordinarily associated with it are entirely absent. Frequently the food is simply regurgi-

tated into the mouth where it may be held and swallowed again or it may run out at the corners of the mouth.

The vomiting may be only occasional with no interference with weight and growth, or it may be so severe as to cause a marked loss of weight and even threaten life. It sometimes ceases spontaneously; at other times it may be most obstinate. The diarrhea also varies in severity. It may occur with breast-fed as well as artificially-fed infants. The stools may be only slightly more frequent than normal, three to five a day, and well digested; or they may be much more numerous and passed through the intestinal tract so rapidly that they are undigested and frequently contain mucus.

The diarrhea is apparently caused by an excessive irritability of the intestines, an increased reaction of the nerves to the stimuli which ordinarily produce moderate peristalsis. As a result, the food is hurried along more or less unchanged, together with increased intestinal secretions. The diarrhea may be most obstinate. Marked and even serious malnutrition may result.

A recognition of the essential condition is necessary for proper treatment. Such infants should be kept as quiet as possible with no excitement or unnecessary handling. If vomiting is present, the food should be given at four-hour intervals. When, in spite of reduction of the fat and elimination of sugar, vomiting continues, solid food given with a spoon is usually retained. This food is preferably some form of cereal such as farina or barley thoroughly cooked, but so thick that it must be given with a spoon. Infants as young as four or five months take this admirably,—two or more ounces every four hours. The proportion of one part of cereal to ten parts of milk is usually thick enough; at times, however, it must be as thick as one part of the cereal to five of milk, in order to prevent regurgitation. If a flour is used, this should be cooked for at least an hour,—for coarse cereals three to four hours are necessary. This diet may be continued until other food is added at the end of the first year. Water should be given between the feedings.

The treatment of the diarrhea is conducted along the same lines as with diarrhea from other causes. The essential condition, an increased peristalsis, is the same in either case. The irritation of the intestinal contents should be diminished. The irritating products, the lower fatty acids, are found in smaller amount when there is an excess of protein in the diet and when the fats and sugars are much reduced. For this reason with nursing infants striking benefit is often seen after substituting buttermilk for one or more feedings of breast milk. With artificially fed children a reduction of the sugar is usually necessary. Carbohydrates in the form of gruels are much better borne than the sugars. When diarrhea is excessive, protein milk may be necessary, at

first without and later with the addition of a preparation of maltose. Success is only obtained with continuous and intelligent care.

The Neuropathic Child.—He is the product of both hereditary conditions and the environment in which he lives. The child who is nervous by inheritance is rendered much more so by continual association with nervous parents, especially if, being an only child, he is the subject of their undivided solicitude. Acquired nervousness is by no means infrequent as the result of disease or bad environment, but is lost as soon as the influence that is responsible for it is removed. Nervousness is more common in girls than in boys and is especially seen in the Hebrew and Latin races. It is much increased by a faulty method of living, by late hours and especially by tea and coffee and, in boys, by cigarette smoking.

The symptoms relate not only to the nervous system but to the physical condition of the child as well. Neurotic children are almost always poorly nourished. They have labile vasomotor systems and for that reason blush readily and very often have cold hands and feet. The pulse is apt to be rapid and undergoes a marked increase in rapidity after slight exertion, or as the result of the slightest nervous impression. These children are usually anemic; their appetite is poor and they often suffer habitually from constipation. It is not infrequent for diarrhea to occur, particularly as the result of excitement. Cardiac palpitation is frequently complained of. Nervous vomiting is seen with children, girls especially, of the school age. It occurs in the morning immediately after breakfast, is accomplished without effort and there is usually no nausea. The appetite may remain fair and there is no vomiting at any other time. Nocturnal enuresis is found with many neurotic patients, and masturbation is not infrequent even in those of two or three years.

Mentally, neuropathic children are apt to be bright, often precocious, but they usually show a great lack of concentration. They are frequently animated, talk rapidly, oftentimes stammering. They are never quiet, are full of restless energy, changing rapidly from one occupation to another but soon tire and constantly complain of fatigue. Headache is frequent and often persistent. Vague pains in almost every situation are complained of. Some of these children are confirmed hypochondriacs. Many are affectionate and attractive, but they are usually self-willed and often tyrannize over the household. They are greatly affected by nervous impressions, often timid and readily cry or laugh. Tremor of the hands or eyelids is not uncommon and the facial phenomenon (Chvostek's symptom) is present in many. All sorts of habit spasm are of frequent occurrence and in rheumatic children chorea is a common manifestation.

Sleep is usually poor. Such children have great difficulty in going to sleep and occasionally have night terrors. In general, nervous children demonstrate a combination of irritability to all impressions with a ready exhaustion. Untreated, they are apt to grow up into nervous, often hypochondriacal adults. Even with the greatest care and wisest treatment it is a long and tedious process to bring about an approach to the normal.

Treatment consists largely in the wise management of the daily life. It is frequently necessary to remove the child entirely from the environment in which he has been living. The person in charge should be one who will not spoil or indulge the child and will bring about a proper régime with a gentle but firm control. It is necessary to observe with the greatest care all of the measures which promote the physical welfare of the child and especially to prevent any unnecessary stimuli to the nervous system.

Nervous children are much benefited by association with others of their own age. No greater mistake can be made than to keep such a child by himself for a prolonged period; but it must be remembered that he is usually unable to bear either the physical or the mental strain to which normal children are constantly subjected. For that reason the periods both of study and play should be short. Education at home is usually undesirable; but school hours must be carefully adjusted to the child's endurance. He should not be allowed to become either physically or mentally exhausted. Frequent short periods of rest are necessary; it is often desirable to keep a child in bed for two or three days once or twice a month. Particularly to be avoided are such things as motoring, children's parties, theaters, moving picture shows, etc. Altogether the most satisfactory way of bringing up such a child is in the country away from the excitement and distractions of city life.

Drugs play a very insignificant part in treatment and should be given only for particular symptoms. Tonics, when indicated, may be given, but sedatives to the nervous system should be avoided. It is quite useless to expect relief from such operations as the removal of the tonsils, adenoids, circumcision, etc. Unless the necessity for them is plain, they often do more harm than good.

SECTION III

DISEASES OF THE DIGESTIVE SYSTEM

CHAPTER I

DISEASES OF THE LIPS, TONGUE, AND MOUTH

MALFORMATIONS

Harelip.—This is one of the most frequent congenital deformities. It is caused by an incomplete fusion of the central process with one or both of the lateral processes from which the upper half of the face is developed. This deformity may be single or double; the fissure is never in the median line, but usually just beneath the center of the nostril. There may be simply a slight indentation in the lip, or the fissure may extend to the nostril. Both single and double harelip—more frequently the latter—may be complicated by fissure of the palate. Double harelip is usually accompanied by a fissure between the intermaxillary and the superior maxillary bone of each side.

Cleft Palate.—This is second in frequency to harelip. It may involve the soft palate only, or the fissure may extend into the hard palate, producing a wide gap in the roof of the mouth. The most frequent form is that in which only the soft palate is affected.

For the surgical treatment of both these deformities the reader is referred to text-books upon surgery. As to the time of operation with either harelip or cleft palate,—in general, operation should be performed as soon as the condition of the child will admit. With a vigorous child, it should be done in the first two weeks of life.

If the child is premature or feeble, it is not wise to operate at once, but it is always to be remembered that it does not necessarily follow that the child's condition will be better at another time. The nutrition is always a matter of much difficulty and without operation a very large number of these cases die of inanition and marasmus, even with the best care. The medical treatment consists in the care of the mouth and in the nutrition of the patient. The mouth, in all cases, must be kept scrupulously clean, but the greatest care is necessary not to injure the epi-

thelium. A camel's-hair brush and plain, luke-warm water, or a weak alkaline solution, are to be recommended. Both of these deformities are exceedingly likely to be complicated by thrush. This is a serious menace to the success of any operation, and even to the life of the patient. In cases of harelip, if the fissure is so great as to interfere with nursing, the mother's milk should be pumped and the child fed with a spoon or a medicine dropper until the operation can be performed. In cleft palate there may be attached to the rubber nipple of the nursing bottle a flap of thin sheet-rubber in such a way that it closes the fissure in the mouth when once the nipple is in place. This flap should be shaped like a leaf, one extremity being sewed to the neck of the rubber nipple and the other end left free. In many cases, both before and immediately after operation, feeding by gavage may be resorted to with the greatest benefit and with very little inconvenience.

Congenital Hypertrophy of the Tongue.—This is usually due to disease of the lymphatics, and is to be regarded as a lymphangioma. In a few cases hypertrophy of the muscular fibers has been present. The tongue may reach an enormous size, so that it is impossible for it to be contained within the cavity of the mouth, and it may thus interfere with nursing, deglutition, and even with respiration. The treatment is surgical; but some of these patients have been strikingly benefited by radium.

Cases like the above are to be distinguished from those of enlargement of the tongue seen in sporadic cretinism. In this disease the tongue is considerably enlarged and may protrude slightly from the mouth, but it is rarely, if ever, large enough to cause other symptoms. It diminishes notably under treatment with thyroid extract.

Bifid Tongue.—These cases are extremely rare. Brothers has reported to the New York Pathological Society a case of cleft tongue in a child of one month. There was, in addition, a fissure of the soft palate.

Tongue-tie.—This deformity is due to such a shortening of the frenum that it is impossible to protrude the tongue to a normal extent. It differs considerably in degree in different cases. In some, the tongue can not be protruded beyond the gums. Tongue-tie may interfere with articulation, and even with sucking. The treatment consists in liberating the tongue by dividing the frenum with scissors and completing the operation with the finger nail. This should be done in every case unless the child is a bleeder. In many cases the mother may think the tongue tied when the frenum is of normal length.

Bifid Uvula.—This is not very uncommon. It usually occurs in connection with cleft palate, but is occasionally seen when there is no other deformity present. It may be complete or partial, and it does not of itself require treatment.

DISEASES OF THE LIPS

Herpes.—Herpes labialis is an exceedingly common affection in children, occurring in acute febrile diseases, particularly pneumonia, and sometimes alone. It is the familiar "fever sore" or "cold sore" of domestic medicine. The appearance is similar to herpes in other parts of the body. There is first a group of vesicles, then rupture and the formation of crusts. It is often quite difficult to cure on account of the disposition of children to pick the lip with the fingers. Although it heals without treatment, recovery is facilitated by the use of some antiseptic lotion, such as dilute boric acid, followed by a dusting powder of zinc oxid and boric acid. This treatment is generally more successful than the use of ointments. Young children should wear mittens or elbow splints at night, to prevent picking at the crusts.

Eczema of the Lip.—This is an exceedingly common condition, and a very troublesome one. The vermilion border is dry and rough, and prone to deep cracks or fissures. These are usually seen at the angles of the mouth or in the median line. When severe they are exceedingly painful, bleed freely, and are the cause of very great discomfort, especially in the cold season. The lips should be covered at night by boric acid ointment, and this should be used as much as possible during the day. When deep fissures form, they should be touched with burnt alum, or with the solid stick of nitrate of silver. Syphilitic fissures are considered with the symptoms of that disease.

Perléche (French, *perlécher* = *to lick*).—This name was first given by Lemaistre to a form of ulceration occurring usually at the angle of the mouth. It begins in most cases as a small fissure, which, by constant licking and irritation, to which there is usually added infection, may develop into an intractable ulcer of considerable size. It often resembles the mucous patch of hereditary syphilis. The ulcer is of a grayish color, is quite painful, and is associated with considerable swelling of the lip. It lasts from two to four weeks. The treatment is the same as in simple fissure—viz., the use of burnt alum or nitrate of silver, and covering the part with bismuth or oxid of zinc.

DISEASES OF THE TONGUE

Epithelial Desquamation.—This is a disease of the lingual epithelium, which is characterized by the appearance upon the dorsum or margin of the tongue, of circular, elliptical, or crescentic red patches, with gray margins which are slightly elevated. The gray margins are apparently due to thickening of the epithelial layer and the red areas

to desquamation of the epithelium. It is sometimes improperly called psoriasis of the tongue. It is quite a common condition, and is probably congenital.

As usually seen, there exist upon the tongue from two to half a dozen of these red patches surrounded by a gray border, which is about one-twelfth of an inch wide, and slightly elevated. The outline of the patch is nearly always crescentic (Fig. 27). From day to day the configuration of the patches changes; the gray lines advance across the tongue from side to side, or from base to tip, disappearing as they reach the border or the extremity. They are followed by the red patches, and as the old ones fade away new ones form and run the same course. The red patches are of a bright color nearest the border, gradually shading off into the normal color of the tongue. Only the epithelium is involved, the deeper structures being unaffected. The duration of the disease is indefinite; it usually lasts for years. Guinon reports several cases which recovered during an intercurrent attack of measles or scarlet fever.

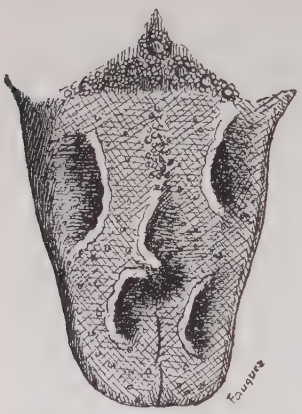


FIG. 27.—EPITHELIAL DESQUAMATION OF THE TONGUE. (Guinon.)

The cause is unknown. The condition occurs rather more frequently in females than in males, and Gubler has reported an instance of several members of the same family being affected. The condition has been thought to depend upon nearly every disease of childhood. It is not accompanied by pain, salivation, or by other symptoms of stomatitis, and is of little practical importance. Its symptoms are so characteristic that it can hardly be mistaken for any other condition. Treatment is unnecessary.

Two other forms of epithelial desquamation have been observed, both much more rare than that described. In one of these the red denuded portion occupies the margin of the tongue, while the center is gray or white; the irregular wavy outline which separates the two suggests strongly an outline map, and the condition is sometimes called the "geographical tongue." This term is frequently employed to designate the common form. In another variety nearly the whole organ may be uniformly red, from loss of the epithelium, there being no borders or patches. Both these varieties are of much shorter duration than the more common form, usually lasting only a few weeks.

Glossitis.—Inflammation of the tongue is not very common in children. It is usually of traumatic origin. The injury may be due to bit-

ing the tongue in a fall or in an epileptic seizure. Glossitis is sometimes excited by the irritation of a sharp tooth, causing a wound which may be the avenue of infection; or it may result from taking into the mouth irritant or caustic poisons. In a small number of cases no cause can be found. The symptoms are marked swelling of the tongue, so that it may protrude from the mouth; and it may even be so great as to cause severe dyspnea. There are also seen profuse salivation, difficulty in swallowing and in articulation, and often considerable local pain. There may be a rise of temperature to 102° or 103° F. The treatment consists in the use of fluid food, which in severe cases may be introduced through the nose by means of a catheter. Ice may be used externally, or, better still, pieces of ice may be kept in the mouth continually. If there is obstruction to respiration, and in all severe cases, scarification should be done on the dorsum of the tongue along the side of the raphé.

The acute swelling of the tongue and lips occurring in some cases of urticaria may be mentioned in this connection. This is a rare condition in children, but it may develop rapidly and to such a degree as to cause alarming symptoms. The treatment consists in the use of ice locally, free purgation by salines, and, in extreme cases, needle punctures to relieve the edema.

Tongue-swallowing.—This term is used to describe a rare condition seen in infants, in which the tongue is turned backward into the pharynx, so as to obstruct respiration. It may be drawn quite into the esophagus. Several marked cases have been collected by Hennig. While most frequently occurring with paroxysms of pertussis, tongue-swallowing has been seen in other diseases. This should not be forgotten as one of the explanations of sudden asphyxia in a young infant. The conditions necessary for its production are a somewhat relaxed organ or a long frenum. In none of the fatal cases reported, however, had the frenum been divided. In some weak infants, falling back of the tongue, so that its base partly covers the epiglottis, produces asphyxia, precisely as it occurs in adult life under full anesthesia. The recognition of the condition is a very easy one, and its treatment is to relieve the obstruction by drawing the tongue forward by the finger or forceps.

Ulcer of the Frenum.—The friction against the sharp edges of the lower central incisors frequently causes an ulcer of the frenum in infants. We have never seen it in older children. It usually occurs in pertussis, but is seen in other conditions. In some it appears to be produced by friction of the teeth during nursing from the breast or bottle. It is more often seen in children who are delicate or cachectic than in those who are healthy and well nourished. The ulcer may be confined to the frenum, or it may extend quite deeply into the tongue. It is

usually about one-fourth of an inch in diameter, and of a yellowish-gray color. When not readily cured by touching with alum or nitrate of silver, the child may be fed by gavage for several days, or the teeth may be covered by a bit of absorbent cotton.

DENTAL CARIES

Although the teeth do not strictly belong to the province of the physician, they have an important influence upon the general health. The pernicious effects of dental caries have only recently been appreciated. Routine examinations of public-school children, made in various cities, have shown that fully 80 per cent have extensive dental caries. Among the inmates of institutions the proportion is fully as great as this, possibly greater, unless, as in a few modern institutions, special attention is given to this subject.

Among the causes of dental caries the most important without doubt is want of cleanliness—the almost entire neglect of the toothbrush among the children of the poor. This leads to decomposition of food and secretions, acid fermentation, erosions of the enamel, etc. But not all caries of the teeth can be ascribed to this cause. Diet has certainly much to do with it. It is our belief that the opinion commonly held, that excessive indulgence in sweets is responsible for dental caries, is well founded. Malnutrition and improper food, especially in early childhood, certainly affect the teeth. In some children a congenitally defective enamel is present. Hereditary syphilis is also a cause, and in children with congenital mental defects the teeth are prone to early decay.

The symptoms are both local and general. Locally, as a result of decomposition and infection, there are present foul breath, gingivitis, alveolar abscess, ulcerative stomatitis, toothache, etc. The lymph nodes in the neighborhood frequently become enlarged and sometimes tuberculous. Tuberculosis of the submaxillary and submental lymph nodes is nearly always the result of infection through the teeth or the gums. Whether the cervical lymph nodes are infected in the same way is very doubtful. The general symptoms result in part from improper mastication of food and in part from sepsis from the local condition. There may be seen only failing nutrition, loss of appetite and anemia; or these symptoms may be accompanied by a slight but continuous fever which may persist for months. In more marked cases there may be symptoms of a pyemic character; higher temperature, joint swellings, wasting, etc. Many cases of illness diagnosticated acute rheumatism and accompanied by cardiac complications have their origin in oral

sepsis at the basis of which are carious teeth, and no treatment has any influence upon the condition until these are removed.

From the local irritation various nervous symptoms may arise. The most common are habit spasm, facial chorea, headaches, and, according to some writers, even epileptiform convulsions. The presence of carious teeth is a menace to the general health. They certainly predispose to local tuberculosis. Many persons assume that if the teeth affected belong to the first set, it matters little. However, the permanent teeth are often injured by extensive decay of the deciduous set. The treatment of this condition belongs to the dentist; but the physician should appreciate the importance of the subject and urge parents and others in charge of children to give proper attention to cleanliness and to see that carious teeth of the first set are either filled or removed.

ALVEOLAR ABSCESS

This is common in children, especially among the class of hospital and dispensary patients, in whom little or no attention is given to the care of the teeth. It causes severe pain and acute swelling, which may be limited to the gum, or it may involve to a considerable extent the periosteum of the jaw and even cause swelling of the whole side of the face. If there is retention of pus, there may be quite severe constitutional symptoms, such as chills and high temperature; but in most of the cases these are wanting. The abscess usually opens spontaneously into the mouth, but it may open externally if the molar teeth are the ones affected. It may even lead to necrosis of the jaw. If its site is the upper jaw, the pus may find its way into the nasal cavity or into the maxillary sinus.

The treatment is, in the first place, prophylactic. This requires attention to the teeth to prevent decay, and the removal of old carious fangs, which are a constant menace to the health of the child. The free use of the toothbrush and some antiseptic mouth-wash will, in the great majority of cases, prevent the occurrence of this disease. It is important that the abscess be opened early and free drainage secured. If there is a carious tooth it should be drawn.

DIFFICULT DENTITION

The place of dentition as an etiological factor in the diseases of infancy is one which has given rise to much discussion. From a very early period the view has descended, that a large number of the diseases occur-

ring between the ages of six months and two years are due to difficult dentition. The list of such diseases is a long one, but year by year it has been shortened as one after another has been shown to depend upon other causes, dentition being only a coincidence.

At the present time many good observers deny that dentition is ever a cause of symptoms in children; some even going so far as to say that the growth of the teeth causes no more symptoms than the growth of the hair. Without doubt the usual mistake made in practice is to overlook disease of the brain, ears, lungs, stomach, and intestines, because of the firm belief that the child was "only teething." The physician who starts out with the idea that in infancy dentition may produce all symptoms usually gets no further than this in his etiological investigations. Although no doubt the importance of dentition as an etiological factor in disease has been in the past greatly exaggerated, the careful and candid observer must admit that, particularly in delicate, highly nervous children, dentition may produce many reflex symptoms, some even of quite an alarming character.

Speaking from general impressions not from statistics, we should say that in our experience fully one-half of the healthy children cut their teeth without any visible symptoms, local or general; in the remainder some disturbance is usually seen, and though in most cases it is slight and of short duration, it may last for several days or even a week. The symptoms most commonly seen are disturbed sleep, or wakefulness at night and fretfulness by day, so that children often sleep only one-half the usual time. There is loss of appetite, and much less food than usual is taken. There is often, but not always, an increase in the salivary secretion, a slight amount of catarrhal stomatitis, and a constant disposition on the part of the child to put the fingers into the mouth. The bowels are often constipated or there may be slight diarrhea. The thermometer may show a slight elevation of temperature to 100° or 101.5° F. The weight often remains stationary for a week or two, and there may even be a loss of a few ounces. The duration of these symptoms in most cases is but a few days, and they require no special treatment. If the food is forced beyond the child's inclination, attacks of indigestion with vomiting and diarrhea are easily excited.

Symptoms more severe than the above, are rare in healthy children, but are not infrequent in those who are delicate or rachitic. In such susceptible children, even so slight a thing as dentition may be an exciting cause of quite serious disturbances. Often there is some other factor in the case, such as bad feeding or feeble digestion. In delicate or rachitic children there may be seen the symptoms already mentioned as occurring in healthy infants, but in greater severity; and in addition there may be severe attacks of acute indigestion. Occasionally there is

an elevation of temperature to 102° or 103° F., lasting usually only two or three days, and accompanied by no symptoms except almost complete anorexia. It is occasionally, but rarely, seen that a child will have convulsions just before or during the eruption of each tooth. Such children are almost always the subjects of latent tetany, dentition acting as any other exciting cause to determine the onset of the convulsions. In cases of eczema the symptoms often undergo a distinct exacerbation with the eruption of each group of teeth. As regards almost all the other diseased conditions which are commonly attributed to dentition, we believe that it is a delusion to ascribe them to this cause.

The physician should watch a child carefully, and examine him frequently, to be sure that he is not overlooking some serious local or constitutional disease before he allows himself to make the diagnosis of difficult dentition. Probably in ninety-five per cent of the cases in which symptoms are present, they are due to some cause other than dentition. When, however, symptoms such as any of those mentioned disappear immediately when the teeth come through, and when we see them repeated four or five times in the same child with the eruption of each group of teeth, and accompanied by red and swollen gums, we can not escape the conclusion that dentition is a factor in their production, though perhaps not the only one.

In the treatment of this condition drugs occupy but a small place. It should be remembered that infants are at this time in a peculiarly susceptible condition as regards the digestive tract, and attacks of indigestion, and even severe diarrhea, are readily excited from slight causes, especially from overfeeding. Special care should be exercised in this respect. The strength of the food should be reduced, as well as the amount given. A poor appetite indicates a feeble digestion, which should not be overtaxed. As attacks of bronchitis and acute nasal catarrh are readily induced, even slight exposure should be guarded against. The nervous symptoms, when severe, may be relieved by the use of moderate doses of the bromids or by phenacetin, better than by opiates. All soothing syrups should be discountenanced. All the various devices for making dentition easy are a delusion. In a small number of cases lancing the gums is of value. We have seen in a few rare instances marked and undoubted relief given by it. This is likely to be the case only when the gums are tense, swollen, and very red, with the teeth just beneath the mucous membrane. To press a tooth through the gum by simply rubbing gently with the finger covered with sterile gauze is frequently more effective than an incision. It seldom happens, however, that the relief expected is seen from any of the measures mentioned.

CATARRHAL STOMATITIS

This is characterized by redness and swelling of the mucous membrane, and by increased secretion of the salivary and the muciparous glands of the mouth. It usually involves a large part of the mucous membrane.

Etiology.—Catarrhal stomatitis may result from traumatism. This injury may be mechanical, or due to heat or any irritant accidentally taken into the mouth. It frequently occurs at the time of the eruption of a tooth. It complicates measles, scarlet fever, diphtheria, influenza, and many other infectious diseases. In these cases and in many others the disease is probably due to direct infection.

Lesions.—The lesions are essentially the same as in catarrhal inflammation of other mucous membranes. There is congestion with desquamation of epithelial cells and sometimes the formation of superficial ulcers. The process may be a very superficial one, or it may extend to the submucous tissue.

Symptoms.—The mucous membrane is intensely injected, all the capillaries are dilated, and small hemorrhages easily excited. The mucous membrane is swollen, this being most apparent over the gums or about the teeth. There may be some swelling of the lips. The mouth seems hot, and the local temperature is certainly increased. There is considerable pain, as shown by fretfulness, but particularly by the disinclination to take food: infants, though evidently hungry, either refusing the breast or bottle altogether, or dropping it after a few moments. The increase in secretion is sometimes marked, so that the saliva pours from the mouth, irritating the lips and face and drenching the clothing. In other cases the saliva is swallowed. On close inspection there may be seen swelling of the muciparous follicles, and even the formation of tiny cysts from the accumulation of secretion within them. The tongue is usually coated, the edges reddened, and the papillae prominent. In febrile diseases, such as typhoid, etc., we may get an accumulation of dead epithelium with the formation of cracks and fissures of the tongue, and the lips may present a similar condition. The neighboring lymphatic glands are slightly enlarged and tender. The constitutional symptoms accompanying simple stomatitis are not severe, but some disturbance is almost always present. There may be derangement of digestion with vomiting, and even a mild attack of diarrhea. In the majority of cases the disease runs a short course, recovery taking place in a few days when the primary cause is removed. In very delicate children it may be prolonged, and from the interference with nutrition may even lead to serious consequences.

Treatment.—The mouth and teeth should be kept clean. Food is more acceptable if given cold. In very severe cases, when food is refused, gavage may be resorted to three or four times daily. In all cases children may be given ice to suck. This is refreshing, both on account of the cold and from the relief to the thirst. The mouth should be kept clean with a solution of boric acid, ten grains to the ounce, or an alkaline solution, such as Dobell's, diluted with an equal amount of cold boiled water; or plain water may be used. In the severe forms, where there is much swelling and slight catarrhal ulceration, astringents are required. In our experience alum is the best; this may be applied in the form of the powdered burnt alum mixed with an equal amount of bismuth, or in solution, ten grains to the ounce, with a swab or brush. Where ulcers are slow in healing and very painful, the powdered burnt alum or the solid stick of nitrate of silver may be applied directly.

HERPETIC STOMATITIS

(Aphthous, Vesicular, or Follicular Stomatitis)

In this form of stomatitis we have the appearance first of small yellowish-white isolated spots, and subsequently the formation of superficial ulcers. These ulcers are first discrete, but may coalesce and form others of considerable size. It is a self-limited disease, usually running its course in from five days to two weeks.

Etiology.—Very little is as yet positively known regarding the cause of herpetic stomatitis. It is not common in the first year, but after that is very frequently seen throughout childhood. It occurs in the strong as well as in the delicate. It is often associated with some disturbance of the stomach, and occasionally with dentition. We have adopted the term herpetic, because the condition is analogous to herpes of the lips and face, the difference in appearance being due chiefly to location. It is apparently caused by something which acts upon terminal nerve filaments.

Lesions.—The generally accepted opinion is that there is first a vesicle, followed by a death of epithelial cells covering it, and then a superficial ulcer. The white appearance is due to the fact that the ulcers, being on a mucous membrane, are always moist. These ulcers may extend superficially, but never deeply; they heal quickly with the formation of new epithelial cells, leaving no cicatrices. Herpetic stomatitis is always associated with more or less catarrhal inflammation.

Symptoms.—The disease is characterized by local and general symptoms. The latter are quite indefinite—general indisposition, loss of

appetite, and slight fever. The local symptoms consist in the development of small, shallow, circular ulcers, usually coming in successive crops. While most frequent at the border of the tongue and the inside of the lips, they may be found upon any part of the mucous membrane of the mouth or the pharynx. There may be only half a dozen present, or the mouth may be filled with them. They are first of a yellowish color, and on an average about one-eighth of an inch in diameter. By the coalescence of several smaller ulcers there may form patches of considerable size, sometimes nearly covering the lips. The older ulcers are apt to have a dirty-gray color, and in places may look not unlike a diphtheritic membrane. The smaller ones are surrounded by a red areola, and when healing the margin is of a bright red color. Their appearance is often more like that of an exudation upon the mucous membrane than an ulceration. The other symptoms are much the same as those of catarrhal stomatitis, but usually of greater severity. The pain is particularly intense, it being often difficult to induce children to take anything in the form of food. The tongue is frequently coated, but there is never the foul breath of ulcerative stomatitis. The duration of the disease is from one to two weeks, and, if the child is in good condition, complete recovery takes place even without any special treatment. In badly nourished children the disease may last for two or three weeks; relapses may occur, and the condition may interfere very seriously with the child's nutrition.

Treatment.—This is the same as in catarrhal stomatitis, with the addition that to each one of the ulcers finely powdered burnt alum should be applied with a camel's-hair brush. If this is not effective, the solid stick of nitrate of silver may be used. The ulcers will usually yield rapidly to this treatment. In our experience, drugs given with the purpose of affecting the lesion in the mouth have been without benefit.

ULCERATIVE STOMATITIS

Ulcerative stomatitis is believed to occur only when teeth are present. It is characterized by an ulcerative process, beginning at the junction of the teeth and the gum, and extending along the teeth; it occasionally involves other parts of the mouth, but never spreads beyond the buccal cavity.

Etiology.—A form of ulcerative stomatitis is produced by certain metallic poisons, especially mercury, lead, and phosphorus; but all these are now rare. Ulcerative stomatitis also occurs in scurvy; and it seems probable that an allied disturbance of nutrition, with spongy, swollen gums, precedes some other forms of ulcerative stomatitis. Bad sur-

roundings and improper food act as predisposing causes; for the disease is quite common in institutions for children and in hospital and dispensary patients, although rare in private practice. Local causes of importance are want of cleanliness of the mouth and teeth and the presence of carious teeth. Conditions which produce a lowered vitality of the gums act as predisposing causes, and infection as an exciting cause of the disease. The constant clinical features of ulcerative stomatitis and the occasional occurrence of epidemics indicate a specific cause which is probably the same as that of ulceromembranous tonsillitis. The two conditions often exist at the same time. From the investigations of Vincent, Bernheim, Plaut and others it seems probable that noma is also produced by the same organism but represents a more virulent infection.

Lesions.—The disease may begin at any part of the mouth, but most frequently upon the outer surface of the gum along the lower incisor teeth. From this point it extends behind the teeth, and from the incisors to the canines and molars, usually of one side only; but it may involve the entire gum of both jaws. From the gums the process may spread to the lips, affecting the fold of mucous membrane between the gum and the lip, and also to the inner surface of the cheek, especially opposite the molar teeth, where large ulcers often form. In neglected cases the disease may extend into the alveolar sockets, the teeth loosening and falling out. The periosteum of the alveolar process may be involved, and even superficial necrosis of the jaw may occur, as has happened in several cases that came under our observation. These severe forms are met with in institutions chiefly and then generally follow measles or scarlet fever.

Ulcers similar in appearance may also be present in other parts of the mouth—i. e., on the soft palate or the tonsils, sometimes even when the gums are not involved.

Symptoms.—The first things noticed are the very offensive breath and the profuse salivation. It is usually for one of these symptoms that the patient is brought for treatment. On inspection of the mouth, there are seen in the mild cases, swollen, spongy gums of a deep-red or purplish color, which bleed at the slightest touch. There is a line of ulceration, usually along the incisor teeth, most marked in front, which may extend to any or to all of the teeth; sometimes it affects only the gum along the molar teeth, the incisors escaping. At the junction of the teeth and gum is seen a dirty, yellowish deposit, on the removal of which free bleeding takes place. The diseased parts are very painful, and the child cries and resists any attempt at examination. In the more severe cases and in those of longer duration the teeth are loosened, sometimes being so loose that they can be picked from the gum. There may be necrosis of the jaw, and even a loose sequestrum may be found. In

these cases the ulceration along the gums is deeper, and there may be ulcers in the cheek opposite the molar teeth, or inside the lip. The swelling may be so great that the teeth are almost covered; this is seen particularly in the scorbutic form. The saliva pours from the mouth, adding greatly to the discomfort of the patient. Beneath the jaw are felt the large, swollen lymphatic glands, which are painful and tender to the touch, but show no tendency to suppurate. The tongue is somewhat swollen, and shows at the edges the imprint of the teeth; it has a thick, dirty coating.

The disease is attended by little or no fever or other constitutional symptoms. The general condition of these patients is often poor, and there may be quite a marked cachexia. Other forms of stomatitis may be associated, and it should not be forgotten that the gangrenous form may follow.

When not recognized or not properly treated, ulcerative stomatitis may last for months. When properly treated it tends in all recent cases to recovery, usually in from five to ten days. No deformity of the mouth is left, the only untoward results being shrinking of the gum, sometimes loss of some of the incisor teeth, and more rarely a superficial necrosis of the alveolar process. All these are quite uncommon. Ulcerative stomatitis can hardly be confounded with any other form, and not only should a diagnosis of the lesion be made, but the condition upon which it depends should, if possible, be discovered; scorbutus, particularly, should not be overlooked.

Treatment.—The first thing to be done is to remove the cause. When dependent upon metallic poisoning the source should be discovered. Scorbutic cases should have the usual anti-scorbutic diet. Cleanliness of the mouth is of great importance, and this may best be accomplished by the use of peroxid of hydrogen diluted with from one to four parts of water. It should be followed by thorough rinsing with plain water, and repeated several times a day. In other cases a solution of alum, five grains to the ounce, or a mouth-wash of chlorate of potash, three grains to the ounce, may be employed. The only objection to the last mentioned is the pain which it sometimes produces. A strip of gauze between the cheek and the gums aids greatly in cleanliness. This may be left in place and affords no inconvenience, but on the contrary, comfort to the patient.

The specific remedy for ulcerative stomatitis is chlorate of potash. The best method of administration is to give two grains, or one-half teaspoonful of a saturated solution, largely diluted, every hour during the day for the first twenty-four hours and subsequently every two hours; when improvement occurs the dose may be still further reduced. Marked benefit is usually seen in one or two days even in cases that have lasted

for several weeks. If the case does not yield readily to this treatment there is probably disease at the roots of the teeth, and when loose these should be removed, and the jaw examined to see if there is necrosis. Occasionally when there is no disposition to heal, the shreds of necrotic tissue should be carefully removed, and burnt alum or nitrate of silver applied.

The constitutional and dietetic treatment in all these cases should be the same as that employed in scurvy—i. e., plenty of fruit, fresh vegetables, and sometimes the internal administration of mineral acids, especially aromatic sulphuric acid. Iron is indicated in most of the cases.

Ulceration of the Hard Palate.—This is usually seen in the first few weeks of life, but may occur in any child suffering from marasmus. The primary cause is often the injury inflicted in cleansing the mouth. In other cases it is due to the friction of the rubber nipple, or some other object which the child is allowed to suck. In still others it is apparently produced by the habit of tongue-sucking frequently observed in these young infants. The appearances are quite characteristic: there is found, rather far back upon the hard palate, usually in the middle line, a superficial ulcer, from a fourth to a half inch in diameter. There are no signs of acute inflammation. Thrush may coexist, but it has no relation to the production of the disease. Spontaneous recovery usually occurs in from one to three weeks, provided the cause can be removed. In children suffering from marasmus these ulcers are very intractable, and in many instances their cure is practically impossible. It is therefore especially important to prevent, if possible, their formation by care in cleansing the mouth, and in avoiding the other causes referred to. When ulcers have appeared they should be treated as in cases of herpetic stomatitis.

THRUSH

(Sprue; German, Soor; French, Muguet)

Thrush is a parasitic form of stomatitis characterized by the appearance upon the mucous membrane, usually of the tongue or the cheeks, of small white flakes or larger patches. It is common in infants of the first two or three months, and in all the protracted exhausting diseases of early life.

Etiology.—The exact class to which the vegetable parasite which produces thrush belongs has not yet been definitely settled. Robin's opinion was long accepted that it was the *oïdium albicans;* the view of Grawitz, that it is the *saccharomyces albicans,* is now more generally adopted. If a little of the exudate from the mouth is placed upon a slide and a

drop of liquor potassae added, the structure of the fungus is readily seen. With the low power of the microscope there can be made out fine threads (the mycelium) and small oval bodies (the spores). With a high power the threads can be seen to be made up of a number of shorter rods, at the ends of which the spore formation takes place (Fig. 28). The mycelium is produced from the spores. The spores of this fungus are of very common occurrence in the atmosphere. It is difficult or impossible for thrush to develop upon a healthy mucous membrane. Its growth is favored by slight abrasions, such as are often produced by rough methods of cleansing the mouth; also by catarrhal stomatitis, a scanty salivary secretion and want of cleanliness. The nature of the process which it produces is in all probability a sugar fermentation, the acid reaction of the mouth being the result of the growth rather than its cause. Infection may come from another patient by means of a rubber nipple or a cloth which has been used for the infected mouth, from the nipple of the nurse, or directly from the air. Its production is favored by a scanty secretion of saliva, hence it is frequent in the first two or three months of life; also by an altered secretion such as is seen in protracted wasting diseases, enterocolitis, marasmus, typhoid, tuberculosis, etc. It is very common in infants suffering from harelip or any other deformity of the mouth. The disease is frequently seen in foundling asylums, in all places where many young infants are crowded together, and where cleanliness of mouths, bottles, etc., is neglected.

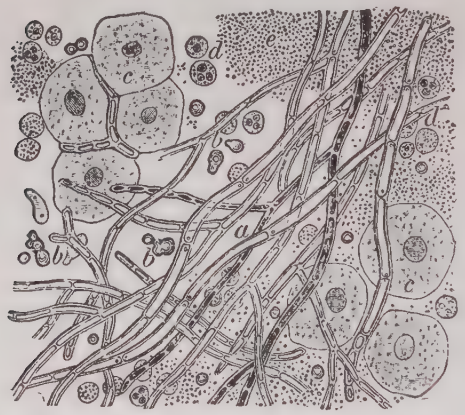


FIG. 28.—THRUSH FUNGUS (highly magnified). *a*, mycelium; *b*, spores; *c*, epithelial cells from the mouth; *d*, leucocytes; *e*, detritus. (v. Jaksch.)

Lesions.—The spores lodge between the epithelial cells and gradually separate the different layers. This occurs before the formation of the white pellicle. Later the disease spreads on the surface of the mucous membrane, and also penetrates the deeper structures. It may invade the blood-vessels and cause thrombosis or even be carried to distant parts. Although the saccharomyces albicans is commonly found upon flat epithelium, its growth is not confined to it. It usually begins at many distinct points upon the mucous membrane, and gradually spreads until coalescence takes place; a continuous membrane may be thus formed. No pus is produced by the process.

The usual seat is the margin of the tongue, the inside of the lips and cheeks, and the hard palate, but not infrequently it involves the pillars of the fauces, and the entire pharynx. Further extension in the digestive tract than this is rare, although the esophagus, the stomach, and even the intestines, may be invaded. We have seen it but once or twice in the esophagus and never in the stomach, and we know of but two reported cases in this country in which thrush has been found there. Cases involving the esophagus and the stomach appear from reports to be much more common in Europe. In a few cases in the Babies' Hospital the saccharomyces albicans has been found in the lungs of infants suffering from bronchopneumonia. There are several reported cases of general blood infection from this organism.

Symptoms.—The essential symptoms of thrush are the appearance upon the mucous membrane of the mouth—usually beginning upon the tongue or the inner surface of the cheek—of small white flakes which resemble deposits of coagulated milk, but which differ from them in the fact that they can not be wiped off. If forcibly removed, they usually leave a number of bleeding points. There may be only a few scattered patches, or the mouth and pharynx may be covered. The mouth is generally dry and the tongue coated; food may be refused on account of pain, and there may be some difficulty in swallowing. The other symptoms depend upon the conditions with which the thrush is associated.

Diagnosis.—This is rarely difficult. The deposit may be mistaken for coagulated milk, but is distinguished by the features just mentioned. When existing upon the pharynx and fauces it has been confounded with diphtheria, although this mistake can hardly be made if all the facts of the case are taken into consideration—the age of the patient, the involvement of the lips and tongue, the dry mouth, the absence of glandular enlargement, etc. In any case of doubt the examination of the deposit under the microscope at once reveals its true nature.

Prognosis.—Thrush is not in itself a dangerous disease, except in the very rare instances where it may obstruct the esophagus, and this can hardly occur except in a condition of exhaustion which is necessarily fatal. In a feeble and delicate infant, or in one with harelip or cleft palate, thrush may be a serious complication. With proper treatment most of the cases involving only the mouth are readily cured.

Treatment.—Thrush may usually be prevented by due attention to cleanliness of the mouth, rubber nipples, bottles, cloths, etc. In infants with deformities of the mouth in institutions, it frequently develops despite all precautions. All rubber nipples should be kept in a solution of boric acid and the child's mouth should be cleansed several times a day. On no account should a feeding-bottle be passed from one child to another.

In the treatment of the disease the essential things are cleanliness, and the use of some mild antiseptic mouth-wash. The best routine treatment is to cleanse the mouth carefully after every feeding or nursing with a solution of bicarbonate of soda, and to apply twice a day a one-per-cent solution of formalin. All applications should be carefully made, so as not to injure the epithelium. The best method of cleansing is by a small swab made with a wooden toothpick and absorbent cotton. Applications to be especially avoided are those mixed with honey or any syrup. In hospital cases the disease seems to be prolonged by the irritation of the rubber nipple of the feeding-bottle. In such it has been our practice to feed by gavage for two or three days, as some cases improved much more rapidly when this was done.

GONORRHEAL STOMATITIS

There has been described by Dohrn and Rosinski a form of stomatitis in the newly born, due to a gonorrheal infection. This is not likely to take place unless the epithelium has been removed. The infection in all cases occurred from the mother. The lesion consists in the formation of yellowish-white patches upon the tongue or hard palate—regions in which the epithelium is liable to be injured by rough attempts at cleansing the mouth. There may be other evidences of gonorrheal infection especially ophthalmia. The diagnosis rests upon the discovery of the gonococcus in the exudate. In all the cases cited the general health was not affected, and recovery followed in the course of a week or ten days.

The treatment consists in thorough cleanliness and in the application of a saturated solution of boric acid or of formalin, as in thrush.

SYPHILITIC STOMATITIS

The buccal symptoms of hereditary syphilis are important both from a diagnostic and a therapeutic standpoint. The most frequent lesions are fissures, ulcers, and mucous patches. Fissures are found upon the lips, most frequently at the angle of the mouth, and are usually multiple. They may be quite deep and cause frequent hemorrhages. Mucous patches are superficial ulcers developing from papules which form upon the mucous or mucocutaneous surfaces. In cases of acquired syphilis in children the primary sore may be seen upon the tongue, the lip, or the tonsil. All these symptoms are more fully considered in the chapter on Syphilis.

DIPHTHERITIC STOMATITIS

In severe cases of diphtheria the membrane is found not only upon the pharynx and tonsils, but it may appear anywhere upon the buccal mucous membrane or the lips. It is questionable whether the diphtheritic process ever begins on the mucous membrane of the mouth, or is ever limited to this part. In our own experience diphtheritic stomatitis has always been associated with deposits upon the tonsils and pharynx. It is seen only in the severest cases, and in those which, from other conditions present, are almost necessarily fatal. Bearing in mind the above points, it can hardly be mistaken for any other variety of stomatitis, although not infrequently the mistake is made of regarding as diphtheritic, cases of herpetic stomatitis in which the ulcers have coalesced. The treatment, so far as the mouth is concerned, consists in cleanliness by frequent gargling or irrigation with a hot saline solution. Forcible removal of the membrane is not to be advised.

GANGRENOUS STOMATITIS—NOMA

(*Cancrum oris*)

The term noma is used to designate all forms of spontaneous gangrene occurring in children, which involve mucous membranes or mucocutaneous orifices. The most frequent situation being the mouth, noma and gangrenous stomatitis are often used synonymously. Noma may, however, affect the nose, external auditory canal, vulva, prepuce, or anus. It is a rare disease, and usually terminates fatally.

Etiology.—Noma is seldom seen outside of institutions for children, where small epidemics are not uncommon. It is usually secondary to some of the infectious diseases, most frequently following measles, and next to this scarlet fever, typhoid, or whooping-cough. While it may occur at any age, most of the cases are in children under five years, and in those of poor general condition. Noma seldom attacks parts previously healthy. In the mouth it may be preceded by catarrhal, or more often by ulcerative stomatitis; in the auditory canal, by a chronic otitis media. There seems little doubt that the disease is contagious. We once saw five cases in a single ward, all beginning in the auditory canal, which were apparently produced by the use of the same syringe to clean the ears without proper disinfection. All these children were suffering from whooping-cough at the time.

It is now quite well established that the exciting cause of noma is the

same as that of ulcerative stomatitis (q. v.). The pathological process in one case is of a mild type occurring in patients of considerable resistance. In the other it is of a severe or malignant type occurring in patients of feeble resistance as a result of previous acute disease. In the gangrenous tissue pyogenic cocci and putrefactive bacteria are abundant. In the border zone, and extending into the adjacent healthy tissue the specific organisms of the disease are usually found.

Lesions.—The process is one of slowly spreading gangrene. In most of the cases there are thrown out inflammatory products in quite large amount, but there is little or no tendency to limitation of the disease. This usually advances steadily until death occurs. In a small number of cases a line of demarcation finally forms and the slough separates, leaving a large area to be partially filled in by granulation and cicatrization. Other infectious processes are likely to accompany the disease, particularly bronchopneumonia.

Symptoms.—The constitutional symptoms are not usually severe until the local disease has existed for several days. Then those of marked prostration and sepsis develop, sometimes quite rapidly. The temperature is usually elevated to 102° or 103° F., and sometimes to 104° or 105° F. There is dulness, apathy, feeble pulse, muscular relaxation, and very often diarrhea. Before death the temperature may be subnormal.

Of the local symptoms, often the first to attract attention is the odor of the breath; sometimes it is the dusky spot on the cheek or lip. On examination of the mouth, there usually is found upon the gum or inside of the cheek a dark, greenish-black necrotic mass, surrounded by tissues which are swollen and edematous, so that the cheek or lips may be two or three times their normal thickness. Externally the parts are tense and brawny from the swelling, this infiltration always extending for some distance beyond the gangrenous part. As the process extends, the teeth loosen and fall out; there may be necrosis of the alveolar process of the jaw and perforation of one or both cheeks or lower lip; extensive sloughing of the face may take place, usually upon one side, sometimes upon both, giving the patient a horrible appearance. In one patient the process began in the right cheek, subsequently involving the left; perforation occurred in both cheeks, and before death a large part of the face was gangrenous. The odor from a severe case is very offensive, and, in spite of all efforts at disinfection, it may fill the ward or even the house. Pain is rarely severe, and in many cases it is absent. Extensive hemorrhages are rare.

We have notes of seven cases in which noma affected the ear, being preceded by chronic otitis media in every instance. The disease began in the deeper structures of the canal, the first symptom noticed usually

being a nodular swelling just beneath the ear, crowding the lobe upward. Shortly afterward there appeared the dirty brown discharge with a gangrenous odor. Later, the gangrenous circle surrounded the meatus, which gradually extended, until in some cases the whole side of the face and head were involved. A probe could readily be passed into the cranial cavity. All these cases ended fatally.

The usual duration of the disease is from five to ten days. If recovery takes place, there is first seen a line of demarcation; then the slough is thrown off, and granulation and cicatrization begin, but require a long time, usually leaving an unsightly deformity.

The prognosis is grave, fully three-fourths of the cases proving fatal. The results depend not only upon the disease itself, but upon the condition of the patient with which it is associated.

Gangrenous stomatitis can hardly be mistaken for any other form of disease occurring in the mouth, and early recognition is of great importance, since only early treatment is likely to be successful.

Treatment.—Much can be done to prevent the disease by careful attention to all the milder forms of stomatitis, particularly to the ulcerative variety. Frequent and thorough cleansing of the mouth in all acute infectious diseases is a part of the treatment which is too often neglected. This should be a matter of routine in every severe illness in a young child. Recognizing the malignant nature of gangrenous stomatitis, its treatment should be radical from the very outset. Of the measures which have been proposed, that which seems to offer the best chance of arresting the process is excision with cauterization. This should be done under anesthesia. In excising, one should go some distance into tissues apparently healthy, for the reason that the process has always advanced farther in the subcutaneous tissues than in the skin. The edges of the wound should then be thoroughly cauterized, best by the Paquelin cautery. Of the other means employed, the use of strong carbolic acid immediately followed by alcohol is probably the best. This is to be used after excising or curetting the necrotic tissue. The mouth should be kept as clean as possible by the use of peroxid of hydrogen. The general treatment should be supporting and stimulating. As the possibility of contagion exists, every case should be isolated.

CHAPTER II

DISEASES OF THE PHARYNX

ACUTE PHARYNGITIS

Acute pharyngitis may exist as a primary disease, or with any of the infectious diseases, particularly scarlet fever, measles, diphtheria, or influenza. Secondary pharyngitis will be considered in connection with these different diseases.

Certain children have a constitutional predisposition to attacks of acute pharyngitis, and contract it upon the slightest provocation. In some of them there is a strongly marked rheumatic diathesis. Attacks of acute pharyngitis often follow exposure. In many cases they are associated with acute disturbances of digestion. All of the above causes probably act by producing local and general conditions favorable to the development of microörganisms already present in the mouth. The bacteria most frequently associated with severe attacks are the staphylococcus, the pneumococcus, the streptococcus, and less frequently, the influenza bacillus.

In acute catarrhal pharyngitis the inflammation may involve the entire mucous membrane of the tonsils, fauces, uvula, posterior and lateral pharyngeal walls, or any part of it. It may exist alone, or in connection with a similar inflammation in the rhinopharynx or in the larynx. In the beginning there is seen an acute redness, usually involving the entire pharynx. This may entirely subside after twenty-four hours, or it may be followed by the usual changes of acute catarrhal inflammation—dryness, swelling, and edema. Later there is increased secretion of mucus, and finally there may be muco-pus. Occasionally slight hemorrhages are present.

There is pain at the angle of the jaws, which is increased by swallowing, also a sensation of dryness and roughness in the pharynx, and often an irritating cough. There may be slight swelling of the neighboring lymphatic glands. The constitutional symptoms in young children are often severe. Not infrequently there is a sudden onset with vomiting, and a rise of temperature to 102° or even 104° F. These symptoms are usually of short duration, frequently less than twenty-four hours, and in two or three days the patient may be entirely well. In other cases the pharyngitis may be accompanied or followed by laryngitis.

Acute primary pharyngitis is to be distinguished from scarlet fever, diphtheria, measles, and influenza. A positive diagnosis from scarlet fever is impossible until a sufficient time has elapsed for the eruption to

appear, and the patient should be closely watched for the first sign of this. If scarlet fever is prevalent, a child with the symptoms of severe pharyngitis should at once be isolated while waiting for the diagnosis to be settled. There is commonly less difficulty in excluding measles because of the absence of Koplik's sign on the buccal mucous membrane, and of the accompanying catarrh of the eyes and nose. Catarrhal diphtheria can be excluded only by culture.

The first step in the treatment of acute pharyngitis is to open the bowels freely by means of calomel, castor oil, or magnesia. The child should be kept in bed, and the diet should be fluid, or, in the case of infants, the amount of food should be much reduced. Pieces of ice may be swallowed frequently for the relief of pain and thirst. Internally there may be given two grains of phenacetin every four hours to a child of three years. It is important at the outset to induce free perspiration. The disease is not serious, and the indications are to make the child as comfortable as possible during the short attack.

UVULITIS

Acute inflammation of the uvula, with swelling and edema, occurs as a part of the lesion in acute pharyngitis. In rare instances the uvula may be the principal or the only seat of inflammation. Huber (New York) has reported two cases, one of which is unique. An infant ten months old was apparently well until two hours before he was seen, when there was noticed a constant irritating cough, accompanied by considerable gagging. Later there could be seen in the mouth a prominent red mass, the enlarged and elongated uvula. There were also paroxysms of coughing, which interfered both with nursing and deglutition. The general symptoms were quite alarming. The uvula was found to be fully one inch long and half an inch wide, red and edematous; in other respects the throat was normal. The symptoms were relieved by multiple needle punctures and the use of ice. In such conditions the greatest relief is often afforded by the application of epinephrin or its use as a spray or gargle.

ELONGATED UVULA

Probably this is primarily a congenital condition. It is increased by repeated attacks of acute or subacute inflammation. The degree of elongation varies in different cases; in some it may be an inch in length. Only the mucous membrane is involved in the elongation. The symptoms are those of local irritation, especially a cough upon lying

down, and the sensation of a foreign body in the pharynx. In some cases it may be a reflex cause of asthma, or, more frequently, of catarrhal spasm of the larynx. The diagnosis is very easily made by inspecting the throat. The treatment consists in grasping the tip of the uvula with forceps and cutting off the excess with the scissors, or a uvulotome. Care should be taken not to cut off too much of the uvula, or severe hemorrhage may occur.

RETROPHARYNGEAL ABSCESS

Two distinct varieties are seen: (1) The so-called idiopathic abscesses which belong to infancy, and (2) abscesses secondary to caries of the cervical vertebrae.

Retropharyngeal Abscess of Infancy.—All of the later investigations regarding this disease indicate that primarily it is not a cellulitis, but a suppurative inflammation of the lymph nodes (lymphatic glands) with a surrounding cellulitis. The retropharyngeal lymph nodes form a chain on either side of the median line between the pharyngeal and the prevertebral muscles. These nodes are said to undergo atrophy after the third year, and in some cases to disappear entirely. Retropharyngeal abscess—or, more properly, retropharyngeal lymphadenitis, since the process does not invariably go on to suppuration—is probably never primary, but secondary to infectious catarrhs of the pharynx, and is set up by the entrance of pyogenic bacteria, usually the staphylococcus or streptococcus. Its pathology is the same as the more frequent suppurative inflammation of the external cervical lymph nodes, with which it is sometimes associated. Usually only a single node is involved, but sometimes two or three are affected, and these may be situated upon opposite sides. We have frequently seen retropharyngeal lymphadenitis so severe as to give rise to marked local symptoms, although it did not go on to suppuration. Kormann's observations, however, show that swelling of these glands in diseases of the mouth and throat is very much more common than is generally supposed. Similar abscesses from suppurative inflammation of other lymph nodes in the neighborhood of the pharynx may occur. We have seen one situated between the epiglottis and the base of the tongue.

Etiology.—These cases almost invariably occur in infancy. Fully three-fourths of those that have come under our observation have been in patients under one year. Bokai (Buda-Pesth) reports that of sixty cases observed, forty-two occurred during the first year, eleven during the second year, and only seven at a later period. The primary disease is usually a severe rhinopharyngitis, or an attack of epidemic catarrh,

but rarely it occurs as a sequel of scarlet fever or measles. In six hundred and sixty-four cases of scarlet fever, Bokai noted retropharyngeal abscess in seven cases. After measles it is even more rare. Retropharyngeal abscess usually occurs in winter or spring, on account of the prevalence of the diseases upon which it depends. It is seen quite as frequently in children who were previously robust as in those who are delicate, but is more common in those who are prone to severe catarrhal affections.

Symptoms.—The early symptoms in most cases are merely those of an ordinary rhinopharyngeal catarrh. After this has subsided the temperature may remain slightly elevated, often for a week or more, before local symptoms are noticeable. Sometimes, without any distinct history of previous catarrh, there are seen quite high temperature, from 102° to 104° F., loss of flesh, and prostration. A careful examination may be required, and sometimes observations for a day or two, before the explanation of these constitutional symptoms is discovered. In other cases the early constitutional symptoms are so slight as to escape notice, and the local symptoms are the only ones present. Although usually these are not severe, retropharyngeal abscess may cause dyspnea, which in a short time assumes an alarming character. The duration of the inflammatory process before abscess forms is generally five or six days, but it may be several weeks. The temperature is invariably elevated, usually from 100° to 103° F.; occasionally it may be 104° or 105° F., with symptoms of prostration seemingly out of all proportion to the local disease, but which are to be explained by the tender age and feeble resistance of the patient.

The most characteristic local symptoms are the posture, the head being drawn far backward to relieve pressure on the larynx, the noisy respiration with the mouth open, difficulty in deglutition and some external swelling. Sometimes the first thing to attract notice is a sudden attack of dyspnea severe enough to cause asphyxia. This is due to the pressure forward of the abscess encroaching upon the larynx. The mouth may be dry, or there may be a copious secretion of pharyngeal mucus. The dyspnea is in most cases greater on inspiration, and in some it is noticed only then, expiration being normal. The difficulty in swallowing is greater when the tumor is low. The child may find it impossible to swallow, and in consequence may refuse to nurse; or the difficulty in nursing may depend upon the nasal obstruction. Sometimes there is regurgitation of food through the nose or mouth. The voice is usually nasal. Generally there is no hoarseness, but a peculiar short cry which is quite characteristic. There may be, although rarely, aphonia. Usually there is some swelling to be seen externally, just below the angle of the jaw in front of the sternomastoid muscle; exceptionally this may be

more prominent than the internal swelling. Occasionally torticollis is an early symptom.

On inspection of the throat there is seen a distinct bulging of the lateral wall of the pharynx, usually a little above the base of the tongue. The swelling may be so great as to crowd the uvula to one side and nearly fill the pharynx. It is rarely, if ever, in the median line. There is usually redness of the mucous membrane and edema of the uvula and of the adjacent parts. On digital examination the swelling is made out even better than by inspection. It may be situated so low down as not to be visible at all. In the early stage there may be felt only a localized induration or a somewhat diffuse swelling, but by the time the swelling is large enough to produce marked symptoms, fluctuation can generally be discovered.

Prognosis.—When left to itself the abscess may open into the pharynx, the pus being swallowed or expectorated. The cavity may close rapidly by granulation, and in a few days the patient be entirely well; or the abscess may refill. External opening almost never takes place. It is rare for much burrowing to occur. In young or very delicate infants the constitutional symptoms may be so severe that the child continues to fail even after the evacuation of the abscess, and dies usually from bronchopneumonia.

Death may occur from asphyxia due to pressure upon the larynx, to edema of the glottis, or from rupture of the abscess into the air passages, especially if this occurs during sleep. Carmichael, Bokai, and others have reported deaths from ulceration into the carotid artery, or one of its large branches. Carmichael's patient was only five weeks old. The general mortality is from five to ten per cent; many deaths are due to a failure to make the diagnosis. Gautier has collected ninety-five cases, with forty-one deaths. In our experience death has most frequently resulted from late bronchopneumonia; in one case it was due to a secondary retro-esophageal abscess.

Diagnosis.—Retropharyngeal abscess is to be suspected if in an infant there is difficulty in swallowing, noisy dyspnea, mouth-breathing, and the head drawn backward. A positive diagnosis is possible only by a digital examination of the pharynx. The mistake most often made is, that the physician, called to a young child suffering from great dyspnea, has jumped at a diagnosis of laryngeal stenosis, and forthwith performed tracheotomy or intubation, without taking the trouble to get the history or to make a careful examination of the pharynx. Many such cases are reported in which the child has died during the operation or immediately afterward, the autopsy first revealing the nature of the disease. A sudden attack of dyspnea like that caused by the rupture of an abscess might be produced by the lodgment of a foreign body in the pharynx

or larynx. A digital examination would aid in the diagnosis. We once saw in an infant a sarcoma of the pharyngeal lymph nodes which gave an external and internal tumor exactly like that of a retropharyngeal abscess.

Treatment.—Before the abscess has pointed, hot applications may be made to the throat to relieve the symptoms and to hasten the formation of pus, since resolution is not to be expected. Spontaneous opening should never be waited for, on account of the danger of the rapid development of serious symptoms from pressure or edema, or of suffocation from an opening into the air passages, especially during sleep.

As soon as the diagnosis is made the case should be carefully watched, and as soon as a point of superficial fluctuation is detected, but not before, the pus should be evacuated. External incision has been advocated, but the internal opening is much to be preferred. In opening through the mouth the patient should be seated in an upright position and the head firmly held. The use of a mouth-gag may cause asphyxia. The abscess may be opened with a bistoury which has been guarded to its point by winding with rubber plaster, or better with a pair of blunt pointed scissors or with an artery clamp. Often a finger-nail sharpened to a point is all that is necessary. After opening it is well to insert the finger into the cavity to enlarge the opening and break down any septa; for after a simple puncture the abscess may refill. The head should then be bent forward, to allow the pus to escape through the mouth. The amount of pus evacuated varies from one dram to half an ounce. In the majority of cases no after-treatment is required. The relief of the dyspnea and dysphagia is immediate, and, except in young infants, recovery usually rapid. Occasionally there is so much edema that even after evacuation tracheotomy may be necessary.

Retropharyngeal Abscess from Pott's Disease.—This form is rare in comparison with that just described, and under three years of age it is extremely so. These abscesses are usually larger, and the amount of pus contained may be from four to eight ounces. They form very much more slowly, often lasting for months, and as with other secondary abscesses, the constitutional symptoms are seldom severe. The swelling is frequently in the median line, and is not so circumscribed as in the idiopathic cases. The pus often burrows along the spine for several inches.

The symptoms of Pott's disease of the cervical region are usually present for several months before the appearance of the abscess. Sometimes the abscess precedes the deformity, and it may be the first intimation of the existence of bone disease. The local symptoms resemble those of the idiopathic cases, but they develop more slowly, and sudden attacks of fatal asphyxia are very rare. External swelling is usually

seen, and it may be quite large, extending almost from one ear to the other, forming a distinct collar. On digital exploration there may be found an irregularity of the anterior surface of the cervical vertebrae, and occasionally a marked angular prominence.

When left to themselves these abscesses may open externally in front of the sternomastoid muscle just below the jaw, sometimes nearly as low as the clavicle; they may rupture internally into the pharynx, the esophagus, or the air passages; or they may burrow a long distance in front of the spine. Death may result from pressure upon the larynx, or from rupture into the larynx, trachea, or pleura; all these, however, are rare. The abscesses not infrequently refill after they are evacuated, and occasionally a discharging sinus is left for many months.

Treatment.—These abscesses should be opened or aspirated as soon as they are large enough to give rise to local symptoms. The external incision just in front of the sternomastoid muscle is generally to be preferred to opening through the mouth, since it gives better drainage, and the after-treatment is more easily carried on; and a sinus opening externally is less objectionable than one opening into the pharynx.

ADENOID GROWTHS OF THE VAULT OF THE PHARYNX

This is a very common condition and one formerly much neglected by the general practitioner. It is the source of more discomfort and the origin of more minor ailments than almost any other pathological condition of childhood.

There is a mass of lymphoid tissue situated at the vault of the pharynx which in structure closely resembles the tonsils. It is often spoken of as the "pharyngeal tonsil." Like the faucial tonsils, this may become greatly hypertrophied, so as to form a tumor large enough to fill the rhinopharynx completely. Those tumors have a broad attachment which is sometimes more to the roof, and sometimes more to the posterior wall of the pharynx. The term *adenoid vegetations* was given to them by Meyer, who first described them in 1868. In infancy these growths are soft, vascular, and spongy; in older children they become firm, dense, and more fibrous. Their appearance is well shown in Fig. 29. Adenoid vegetations are associated with hypertrophy of the faucial tonsils in about one-third of the cases. Growths large enough to cause decided nasal obstruction may in time produce changes in the facial bones amounting to positive deformity. The bony palate may be dome-shaped or even acutely arched; the dental arch of the upper jaw becomes almost V-shaped. Deformities of the thorax also occur, which will be described with the symptoms.

Etiology.—Hereditary influences certainly play some part in the production of this condition. Frequently every one of a large family of children may be affected, and often the parents have suffered from the same condition. While infants are born with adenoid tissue in the nasopharynx, it is in almost all instances small in amount and seldom increases markedly in size until after several months. What causes the

FIG. 29.—ADENOID VEGETATIONS, NATURAL SIZE. (1) From child eight months old; (2) from child twenty-two months old; (3) from child two and one-half years old; (4) from child two and one-half years old; (5) from child three years old. With the exception of (5) all were removed with a single sweep of the curette. Although the growths represented are somewhat larger than the average for the ages mentioned, just such ones are constantly met with in practice.

abnormal development of this tissue it is hard to say. Adenoid growths are most common in damp, changeable climates. Their first symptoms often follow an attack of measles, scarlet fever or diphtheria. The repeated attacks of rhinopharyngitis associated with adenoid growths are more often a result than a cause of the condition.

Czerny believes that the excessive growth of tissue in the rhinopharynx is in many instances the result of overfeeding. It is certainly true that adenoid growths are much more common in well nourished than in poorly nourished children. Much interest has lately been awakened regarding the relation of adenoid growths to tuberculosis. Of 945 cases collected by Lewin in which specimens of adenoids were ex-

amined, tuberculosis was present in five per cent. Though this proportion is no doubt much higher than will be found in private practice, the fact is an important one; for it is highly probable that this is the channel of infection in not a few cases of tuberculosis.

Symptoms.—The symptoms of adenoid growths are usually first noticed when children are from eighteen months to three years old; but they may be present almost from birth. We have in several instances seen them to a marked degree in infants only a few months old. The symptoms generally increase in severity as age advances, being always better in summer and worse in winter, until the age of six or seven is reached. The chief symptoms are those which relate to (1) chronic rhinopharyngeal catarrh, (2) mechanical obstruction, (3) otitis and other aural conditions, (4) general malnutrition and anemia, (5) reflex nervous phenomena.

The rhinopharyngeal catarrh shows itself by a persistent nasal discharge, or frequently recurring acute attacks of head-colds during the winter season. In susceptible children these attacks are often the beginning of a bronchitis, which may keep a young child indoors almost the entire winter.

The obstructive symptoms are inability to blow the nose, mouth-breathing constantly or only during sleep, and a nasal voice. The difficulty in breathing is increased when the child lies upon the back. In consequence of this, children sleep in all sorts of positions—lying upon the face, sometimes upon the hands and knees, and often toss restlessly about the crib in the vain endeavor to find some position in which respiration is easy. The attacks of dyspnea at night may amount almost to asphyxia, and are the explanation of many of the so-called night-terrors from which children suffer. When the obstruction has existed from infancy there are often deformities of the chest; these are most marked in rachitic subjects. The most frequent one consists in deep lateral depressions of the lower part of the chest, with a prominence of the sternum. The deformity is due to interference with pulmonary expansion. There is often seen a flattening at the root of the nose, and sometimes a prominence of the transverse vein in this region.

Some impairment of hearing exists in a large proportion of the cases. Blake (Boston) found this to be true in 39 out of 47 cases examined; in 35 of these marked improvement in the hearing followed removal of the adenoid growths. Deafness may be due to tubal catarrh or to otitis. Often a history is given of several attacks of suppurative otitis.

Many young children who are subject to attacks of spasmodic croup have adenoid growths, the removal of which is frequently followed by the complete cessation of such attacks. Other respiratory symptoms associated with adenoid growths are intractable cough without bronchial

symptoms or signs, and persistent hoarseness lasting for months, or even for years and recurring every cold season. These symptoms are the result of the chronic inflammation in the rhinopharynx, sometimes extending to the larynx, with an increased secretion of thick mucus. Both these conditions are often cured by the removal of the adenoid growths after all other treatment has been without effect. Bronchial asthma seems at times to be dependent upon these growths.

The reflex symptoms ascribed to adenoid growths have been greatly exaggerated. Children become nervous if they have obstructive symptoms with disturbed sleep, or if they spend much of the time in bed or in the house. Such children present a number of nervous manifestations that may be due to other factors producing nervousness, quite as much as to adenoid growths. Incontinence of urine is very rarely cured by the removal of such growths. Headaches with them are, however, common. Stammering, chorea and even epileptiform seizures have been attributed to adenoid growths, but without sufficient justification.

The general health of patients suffering from adenoid growths may be impaired from loss of sleep and from confinement to the house necessitated by attacks of bronchitis or rhinopharyngitis. Anemia is often present. In old cases of a severe character, children may have a dull and stupid facial expression. They are languid, listless, often depressed and this associated with deafness frequently causes them to be regarded in school as children who are somewhat deficient mentally.

These patients are always better in summer and worse in winter. The natural course of the growths if left to themselves is to increase up to a certain point, and then to remain stationary until puberty, when they usually undergo some degree of atrophy. This, with the marked increase in the capacity of the rhinopharynx which occurs at this time, results in a disappearance of the most aggravated symptoms. The removal of the patient to an elevated region with a dry atmosphere will often result in a relief from all the symptoms, and a diminution in the size of the growth, but unless such a change in residence is permanent the symptoms are liable to return. Under ordinary conditions there is little or no tendency to spontaneous recovery. In children with adenoid growths attacks of diphtheria, scarlet fever, measles, and whooping-cough are all likely to be more severe.

Diagnosis.—In a well-marked case the condition is usually evident from the history, and can scarcely be overlooked. The intractable nasal catarrh, upon which no treatment, local or general, has more than a temporary influence, the mouth-breathing, the disturbed sleep, and the slight deafness—all are characteristic. At other times the patients come for treatment on account of the general symptoms—the nervous depression, the headaches, or the anemia. In rare cases the leading symptom may

be epistaxis. The symptoms do not always depend upon the size of the growth, for in a small throat quite a small growth may cause very marked symptoms.

Although the history is in most cases clear, only an examination can make us certain that an adenoid growth exists. The growth is ordinarily felt as an irregular, granular, soft, velvety mass, or sometimes as a firm tumor completely blocking the passage; and the finger, when withdrawn, is frequently covered with blood. By posterior rhinoscopy, the growth in older children can be seen.

Treatment.—The disappearance of adenoid growths is possible only when they are small. This is aided by removal to a warm, dry climate for the winter season. All possible means should be employed to prevent these patients from taking cold. With the larger growths these methods may improve the catarrhal symptoms, but can hardly affect the obstructive ones. The reduction of tumors of any considerable size by local applications is a delusion; every marked case that has come to our notice has been relieved only by operation.

Removal of adenoid growths is indicated: (1) When the obstructive symptoms—habitual mouth-breathing, disturbed sleep, nasal voice, chest deformities, etc.—are marked; (2) for a chronic nasal discharge, constantly recurring attacks of rhinopharyngitis, particularly when these tend to develop into bronchitis or laryngitis; (3) when there is asthma or repeated attacks of catarrhal spasm of the larynx; (4) with deafness, chronic otitis, or repeated attacks of acute otitis. Although striking improvement is not infrequent, one should be cautious about promising too much from operation, especially as regards the nervous conditions; also in older children when there is deafness or asthma.

The preferable time for operation is the late spring or early summer, in order that during the warm months the mucous membranes may have an opportunity to regain their normal condition; however, operation may be done at any time except during attacks of acute catarrh. Unless the symptoms are very marked, it is desirable to defer operation until a child is at least two years old.

Removal of adenoids by scraping with the finger nail is at best a very uncertain method, and is not to be advised. Operation for the removal of adenoids is preferably done with general anesthesia. So many deaths from operations done under chloroform have now been reported, and so many narrow escapes have occurred that have not been reported, that chloroform anesthesia should be given up altogether. Deep anesthesia is not usually necessary, and if the semi-erect position is assumed it increases the danger of the entrance of blood or portions of the growth into the larynx, which might cause asphyxia. The operation should only be done by one skilled in its performance.

Hemorrhage is always abundant, and seems alarming to one who sees it for the first time, but it generally ceases in a few minutes. There is evidence that the administration of fifteen or twenty grains of calcium lactate during the twenty-four hours preceding the operation materially lessens the bleeding. A child should not pass from the physician's observation until all hemorrhage has stopped. He should be kept quiet, preferably in bed, for twenty-four hours; and in the house for five or six days, unless the weather is warm. No after-treatment is necessary. Recurrences are occasionally seen even after a thorough operation by an experienced surgeon; but many of them are due to the fact that the primary operation was incomplete. The improvement generally begins in a few days, sometimes at once, though the full benefit may not be seen for a month. The breathing becomes freer, the sleep more quiet; the mouth may soon be habitually closed; voice and hearing improve, and the benefit to the general health is soon apparent. The pallor, listlessness, and inattention disappear, and a rapid increase in weight often follows. The entire appearance of the child may in a few months be transformed.

Dangers and Accidents from Operation.—While it is rare that any accidents of a serious nature are met with, it should not be forgotten that they may occur. Undue laceration of the parts may result from a bungling operation, particularly with too large curettes or with the forceps. Hemorrhage may be excessive or even fatal. We have seen but one case of fatal hemorrhage, this in a bleeder, and but two other instances of serious hemorrhage. A fatal result is exceedingly rare. Hemorrhage may be continuous after operation, or secondary, in which case it almost invariably occurs within twenty-four hours. It is important, therefore, that the patient be kept under observation for that time. Bleeding is best controlled by injecting into the rhinopharynx through the nostrils one or two drams of hydrogen peroxid, full strength, or, this failing, a solution of epinephrin (1–1000) may be used in the same manner. If this is not effective, plugging of the rhinopharynx and posterior nares may be resorted to. In all cases the patient should be kept absolutely quiet.

Occasionally an acute attack of bronchitis or otitis occurs after operation; and in a few recorded instances acute meningitis has followed. The danger of asphyxia from the entrance of blood or the tumor into the larynx has already been mentioned.

The danger from chloroform anesthesia is due not so much to the nature of the operation as to the condition of the patient. It is now well established that all children in whom the condition known as status lymphaticus is present, bear chloroform very badly.

CHAPTER III

DISEASES OF THE TONSILS

The tonsils are lymphoid structures closely resembling Peyer's patches, but, instead of having a flattened surface, the lymphoid tissue in the tonsils is folded upon itself, forming quite deep depressions—the tonsillar crypts. These crypts, like the surface of the tonsils, are lined by epithelial cells. They contain lymphoid cells, desquamated epithelium, particles of food, and bacteria. Under normal conditions the tonsils take no part in absorption from the mouth. When, however, their epithelium is diseased or removed, the tonsils absorb with very great facility every sort of poison which the mouth may contain.

The most important chronic infection which takes place through the tonsils is that of tuberculosis; the most important acute or subacute infection is probably that of pyogenic organisms. Poisons absorbed by the tonsils are taken up by the lymphatic vessels and through them reach the cervical lymph nodes and finally may be carried into the general circulation.

Acute inflammation of the tonsils, like that of the pharynx, occurs regularly in diphtheria, scarlet fever, and measles, less frequently in the other infectious diseases. The secondary forms will be considered with the diseases with which they are associated.

Acute catarrhal tonsillitis, or inflammation of the mucous membrane covering the tonsils, occurs as part of the lesion in acute pharyngitis, but very rarely is seen alone.

MEMBRANOUS TONSILLITIS

(Pseudodiphtheria; Streptococcus Angina; Croupous Tonsillitis; Septic Sore Throat)

This occurs both as a primary inflammation and secondary to the acute infectious diseases, especially scarlet fever and measles. The angina of scarlet fever is essentially a part of that disease and is more fully considered in connection with it.

Etiology.—As was first shown by Prudden in 1888, and abundantly confirmed by others since that time, this inflammation is usually due to the streptococcus; it may be found alone, or associated with the staphylococcus aureus, and occasionally the staphylococcus may be found alone.

The streptococcus is very frequently found in the throats of healthy

children, particularly in winter and in cities, and more often in those who live in tenements or who are inmates of hospitals or other institutions. The local conditions in the mucous membranes during an attack of measles, scarlet fever, and other infectious diseases, are especially favorable for the development of these germs, which at such times are very often present in great numbers even when no membrane is seen. There are seen occasionally, especially in cities, epidemics of great severity in which many persons, adults as well as children, but the latter chiefly, are attacked. Such epidemics have in recent years broken out in Boston, Chicago and Baltimore. Several of these have been carefully studied epidemiologically and have been traced to the milk supply. The milk has been infected from one or more cows suffering from septic infection of the udder. The organism has been found to be a hemolytic streptococcus with rather distinct cultural characteristics.

In the presence of an epidemic of severe tonsillitis, the milk supply should always be suspected.

Lesions.—In the primary cases the membrane is generally confined to the tonsils or is chiefly there, only small deposits appearing elsewhere. In the secondary cases, the entire pharynx may be covered and the disease may extend to the nose, the mouth, the middle ear, and rarely to the larynx, trachea, and bronchi.

The structure of the membrane resembles that of true diphtheria, and it may be impossible by a microscopical examination to separate the two diseases.

In the mild cases the inflammation of the mucous membrane is a superficial one and the pseudomembrane is not very adherent. In the severe cases, chiefly the secondary ones, the process extends much deeper. Besides the pseudomembrane upon the surface, there is intense congestion, edema, and cell-infiltration of all the lymphoid and cellular tissue of the pharynx. It may involve the tonsils, soft palate, uvula, epiglottis, adenoid tissue of the vault and the entire pharyngeal ring, and also extend to the external lymph nodes and surrounding cellular tissue. The process both in the throat and externally in the neck may terminate in resolution, suppuration, or in necrosis. In severe cases, especially in the epidemic form, there are found the lesions of general septicemia or pyemia. There may be peritonitis, endocarditis, pericarditis, meningitis, arthritis and erysipelas.

The streptococci are found in the false membrane, in the underlying mucous membrane, in the lymph spaces, in the lymph nodes, and in the visceral lesions.

Symptoms.—1. *The Primary Cases.*—The onset is usually abrupt, with well-marked symptoms: there are frequently chilly sensations, headache, vomiting, general pains, and in most cases the child complains of

soreness of the throat and pain on swallowing. There are first seen a general redness and swelling of the tonsils, sometimes of the entire pharynx; shortly afterward membranous patches appear upon the tonsils. These vary greatly in appearance. In color they are yellow or gray, often changing later to a dirty olive tint. The membrane seems loosely attached and can frequently be wiped off with a swab. It is often irregular in its outline, which is not sharply defined. The membrane usually remains but three or four days and disappears rapidly. As a rule, it is limited to the tonsils, and does not spread after it first forms. Occasionally, however, small patches are also seen upon the fauces or the pharynx. The constitutional symptoms are generally severe during the first two days, and the temperature may be 103° or 104° F., but by the third day it falls, and most of the symptoms subside. It is rare for the disease to extend either to the nose or the larynx.

The epidemic cases are usually more severe and the course prolonged. After the first few days, the throat symptoms may nearly disappear, but the fever continues at times for many weeks. The enlargement of the cervical glands is a striking feature, especially of those cases that recover, and this enlargement may persist for a considerable time after the establishment of convalescence. Suppuration of the glands is infrequent. Eruptions are quite common. They may be small, punctate and hemorrhagic or erythematous. If of the latter type, they may be mild or intense, at times closely simulating scarlet fever.

The tendency to complications is great. One of the most common is peritonitis, which is almost uniformly fatal. Endocarditis and pericarditis are frequently seen. There may be septic arthritis, erysipelas or localized abscesses. Otitis media is often associated. Death may be due to the complications or to the septicemia. It is a very severe form of disease. Except in the epidemic cases, the complications and sequelae are infrequent.

2. *The Secondary Cases.*—Some of these are mild, but the majority are severe. The clinical picture of the latter is that of *scarlatina anginosa,* as given by the older writers.

In measles the throat symptoms are somewhat later than in scarlet fever; they may begin at the height of the primary fever, and increase while the eruption fades. The process is almost invariably complicated by bronchopneumonia.

Secondary cases as a class are characterized by high temperature (Fig. 30), rapid, feeble pulse, great prostration, delirium, apathy or stupor, and often albuminuria. In fatal cases death usually occurs at the height of the disease, from asthenia, bronchopneumonia, or nephritis. If none of these complications develop, patients may withstand the toxic symptoms even when they are very severe.

There may be in connection with the local process in the throat, deep sloughing of the tonsils or adjacent structures, suppuration of the lymphatic glands or in the cellular tissue of the neck, occasionally followed by serious hemorrhage. However, these complications are rare, and if the patient survives the danger of the acute stage of the disease, he usually recovers.

Diagnosis.—The clinical features which distinguish membranous tonsillitis from diphtheria are considered under the latter disease. It is

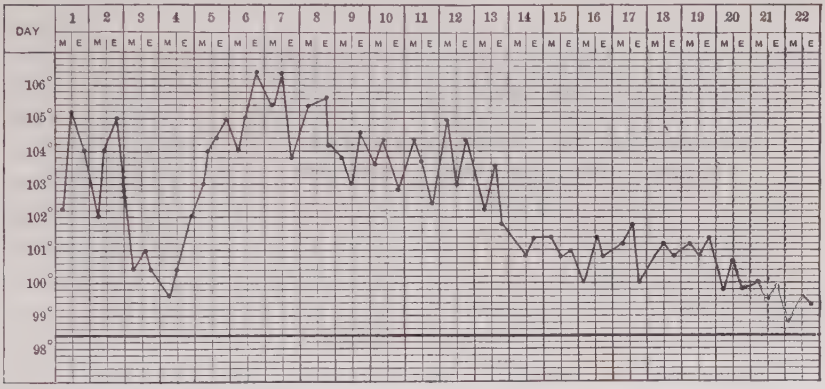


FIG. 30.—STREPTOCOCCUS ANGINA, FOLLOWING MEASLES. The chart begins at the time of the full eruption in a severe case of measles. On the third day the temperature fell, with fading eruption, and child seemed convalescent. With secondary rise in temperature, the tonsils, which before had been only red, showed membranous patches, the exudation rapidly spreading until the entire pharynx was covered; throat symptoms very severe, with great swelling of cervical glands, but the membrane did not extend beyond the pharynx. From sixth to twelfth day a most profound septicemia, so that life was despaired of. The patient was a vigorous child, and, escaping both nephritis and pneumonia, made a good recovery. Convalescence quite rapid; no sequelae. Repeated cultures were made from the throat, but all showed only streptococci. Patient a girl four years old. Case observed in private practice.

impossible in any case to be certain of the diagnosis except by cultures; for, although by clinical symptoms alone one may in the great majority of cases be certain that a given case is one of true diphtheria, to say that any membranous inflammation of the throat is not diphtheria is impossible.

A membrane which appears in the throat early in the course of measles or scarlet fever, or at the height of the primary disease, is usually due to the streptococcus; while one which develops late or after the primary fever has subsided, is frequently due to the diphtheria bacillus. When an eruption is present the diagnosis from scarlet fever may be very difficult, at times well nigh impossible.

Prognosis.—In a child previously healthy, primary membranous tonsillitis, except the epidemic form, is not a serious disease. In the sec-

ondary cases, we find very different conditions. From the best available statistics it would appear that the usual mortality, when it is secondary to scarlet fever and measles, is from fifteen to twenty per cent. However, when these diseases prevail epidemically in institutions, the mortality is often higher than this.

Treatment.—Every child with a membranous patch on the tonsils requires close watching; strict quarantine should be enforced until the diagnosis is positively settled, and even if it is not diphtheria, close contact with other people should be prevented. If under three years old, unless the case can be seen frequently, diphtheria antitoxin should be administered, pending the result of a bacteriological examination. The primary cases require only the treatment of an attack of tonsillitis.

In the severe secondary and septic cases the nose and pharynx should be syringed with a warm saline solution every two hours by day and every four hours by night. Where the swelling and edema are great, benefit may result from frequent spraying with solutions containing epinephrin, also from inhaling hot vapor impregnated with eucalyptol, benzoin, etc. As an external application, whenever there is great adenitis and cellulitis, nothing is so beneficial as the ice-bag.

The general management of these cases as to feeding, stimulants, etc., is the same as in diphtheria. Aside from stimulants no internal medication should be attempted with young children. Those who are older may take with advantage tr. ferri chlor., gtt. v to x, with glycerin, every three or four hours. All milk should be boiled when there is an outbreak of several cases of severe tonsillitis in a community or family.

ULCEROMEMBRANOUS TONSILLITIS

(*Vincent's Angina*)

This is an inflammation somewhat resembling croupous tonsillitis, but it is often unilateral and associated with superficial ulceration. The tonsil is covered with a dirty yellowish exudation, which may be mistaken for diphtheria. There is superficial necrosis, and when this tissue is wiped away with a swab, bleeding occurs. The disease is further distinguished by the swollen lymph nodes at the angle of the jaw, and by the fact that the constitutional symptoms which accompany other forms of tonsillitis are either very slight or absent altogether. The etiology is similar to, if not identical with that of ulcerative stomatitis, with which it is sometimes associated. At such times the breath is foul and there is often profuse salivation.

Ulceromembranous tonsillitis was first described by Vincent, and

by him attributed to a fusiform bacillus which he described, although a spirillum was found associated with it. Vincent's observations have been confirmed, and it has been shown that the spirillum is a degenerative form of the bacillus.[1]

The chief interest in ulceromembranous tonsillitis lies in the diagnosis, although it is not an infrequent disease. It is to be treated, like ulcerative stomatitis, by the internal administration of chlorate of potash, combined with the local application of some antiseptic, such as peroxid of hydrogen or a ten-per-cent solution of nitrate of silver.

FOLLICULAR TONSILLITIS

This is the most frequent and most characteristic form of inflammation of the tonsil. It is essentially an inflammation of the tonsillar crypts, and secondarily of the whole glandular structure.

Etiology.—There is seen in certain children a predisposition to attacks of tonsillitis, so that from very slight exciting causes these occur—sometimes from exposure, sometimes possibly from derangement of the stomach, and sometimes without any evident reason. Children with a rheumatic inheritance appear to be more susceptible than others. One attack predisposes to a second. Patients suffering from chronic hypertrophy of the tonsils are exceedingly prone to acute tonsillitis. It is not very common in infancy, but after this period it is very frequent throughout childhood. The disease, in all probability, begins as an infectious inflammation at the bottom of the crypts, due to the presence of streptococci or staphylococci, which readily enter from the mouth, and excite an attack whenever favorable conditions are present.

Lesions.—As a result of the inflammation, the tonsillar crypts are filled with epithelial cells, pus cells, mucus, and bacteria. These form masses which appear at the mouth of the crypts as small yellow dots, often miscalled ulcers. Sometimes, in addition, fibrin is poured out, and forms, with the other inflammatory products, little plugs which project somewhat from the surface of the mucous membrane, and which can easily be pressed out. Accompanying the changes in the mucous membrane above mentioned, there are acute congestion and swelling of the whole tonsils, with more or less proliferation of the lymphoid tissue.

[1] Vincent's bacillus is about twice as long as the Klebs-Loeffler bacillus. It is thin, with pointed ends, and sometimes bent; it is negative to Gram's stain. The fusiform bacillus is occasionally found alone; the spirillum, never alone. The bacillus is found in smears from the affected tonsil, in making which it is recommended to go deeply into the necrotic tissue, since the superficial parts are crowded with other bacteria. It is grown with difficulty and only upon special culture media.

Follicular tonsillitis is almost always bilateral. Although the pathological process is generally limited to the tonsils, there may be more or less pharyngitis associated.

Symptoms.—The general symptoms usually appear before the local ones, and are often quite severe. The onset is abrupt with chilly sensations, occasionally a distinct rigor. In infants there is often vomiting, and sometimes diarrhea. There is pain in the back, in the muscles of the extremities, and in the head. Sometimes there is pain in the lateral cervical muscles. The temperature rises rapidly to 102° or 103° F., often it touches 104° or 105° F.

The first local symptoms are some swelling of the tonsils and the appearance upon them of isolated yellow spots a little larger than a pin's head. Often these can be wiped off with a swab, or the little plugs can be squeezed out, leaving slight depressions. Later there is acute congestion of the tonsil, with more swelling. Even when the disease is at its height the local pain and discomfort may be only moderate, and in many cases scarcely noticeable. The swelling and tenderness of the lymph glands behind the angle of the jaw are not great, and may be absent.

The constitutional symptoms, as a rule, last three days, and are most severe upon the first day. The local symptoms last somewhat longer, but usually by the end of the fourth day the exudate has disappeared, although enlargement of the tonsil may persist for a week or even longer. On account of the connection of tonsillitis with rheumatism, the heart should be watched during attacks, especially in those who are subject to them.

Diagnosis.—Tonsillitis may be confounded at its onset with scarlet fever. The great frequency of tonsillitis makes inspection of the throat imperative in every case of acute illness in children. The diagnosis from diphtheria is considered in connection with that disease.

Treatment.—Follicular tonsillitis is a mild disease without danger to life, and one which runs a short, self-limited course. The indications are, therefore, to make the patient as comfortable as possible by the relief of individual symptoms. Older children, particularly those who are rheumatic, should be treated with sodium salicylate, or aspirin, four or five grains every three hours being given for the first twenty-four hours, and later less frequently. To infants these drugs must be given in smaller doses and with care, lest they upset the stomach. The general muscular pains of the first day are best relieved by phenacetin, two grains every four hours to a child three years old. Later it may be used in smaller doses, but enough should be given to make the patient comfortable.

Local treatment is better omitted with infants. Older children may gargle with a solution of boric acid or may use a spray of Dobell's solu-

tion. Benefit often follows painting the tonsils with tincture of iodin or a ten-per-cent solution of silver nitrate. In all doubtful cases the patient should be isolated and the same general treatment adopted as in diphtheria.

PHLEGMONOUS TONSILLITIS—PERITONSILLAR ABSCESS—QUINSY

This is an inflammation of the cellular tissue surrounding the tonsil, sometimes invading the tonsil itself. It may terminate in resolution, but usually goes on to the formation of an abscess. Phlegmonous tonsillitis is much less common in children than in adults, and, compared with the other forms, it is a rare disease in early life. It is the only variety which is regularly unilateral. In most cases the inflammatory process is circumscribed, but in rare instances there is seen a diffuse phlegmonous inflammation of the pharynx.

In certain patients there exists a constitutional predisposition to the disease, which may be associated with rheumatism. The exciting cause may be exposure, or anything which may reduce the patient's general health, to which there is added local infection. Catarrhal pharyngitis predisposes to this disease.

Symptoms.—The onset resembles that of follicular tonsillitis, the temperature is often high, and the muscular pains and prostration severe. There is acute pain in the throat, which is increased by deglutition, and finally may be so great that swallowing is almost impossible. It is difficult to open the mouth. There is pain in the lateral muscles of the neck, and often tenderness. In the beginning but little can be seen on inspection, even though the patient complains of a very sore throat. This is always a suspicious circumstance, and should lead one to look out for quinsy. It is due to the fact that the inflammation begins in the deeper tissues, and that the mucous membrane is affected later. After twenty-four or forty-eight hours there is usually quite marked swelling, which is rather more behind the tonsil than elsewhere, pushing it upward and forward; sometimes it is more in front of the tonsil. A little later there is intense inflammation of the mucous membrane covering the tonsil, fauces, and uvula, and not infrequently a fibrinous exudate; the uvula may be pushed to one side, and the isthmus of the fauces diminished to barely one-half its natural size. In one of our own cases marked torticollis was present, and existed for two or three days before the diagnosis of quinsy could be made by the other symptoms.

In most cases the recognition of quinsy is quite easy by attention to the symptoms above mentioned. By inspection of the throat less information is sometimes obtained than by palpation; by this means a

fulness, and later a point of fluctuation, can readily be made out. Acute phlegmonous tonsillitis generally involves no danger to life. In very young infants serious results may follow spontaneous rupture during sleep; and in older children occasionally there may be edema of the glottis. If not treated, abscess usually forms in from five to seven days, and opens spontaneously.

Treatment.—Many drugs have been advocated, but to our minds the best is salol, which should be given in doses of two grains every two hours to a child of five years. In some patients larger doses may be used. This may be combined with small doses (gr. ¼) of Dover's powder. Relief may be afforded by very hot or cold applications, according to the sensations of the patient. The holding of ice in the mouth and the application of an ice-bag externally, often give great comfort. In other cases, gargling with very hot water and the application of hot flaxseed poultices externally, will be preferred. As soon as fluctuation is detected an incision should be made with a guarded bistoury. If made too early, only a small amount of pus is evacuated and the abscess may refill. After spontaneous rupture the relief to symptoms is usually immediate.

CHRONIC HYPERTROPHY OF THE TONSILS—CHRONIC TONSILLITIS

The condition known as chronic hypertrophy is a permanent enlargement due to a proliferation of the lymphoid tissue of the tonsils, and an increase in the connective-tissue stroma. If the increase in the connective tissue is slight, the tonsil is soft; if it is great, the tonsil is firm and hard, almost like a fibrous tumor. All degrees are found. Associated with hypertrophy of the tonsils there are usually found adenoid growths of the pharynx, both of these depending upon similar local and constitutional conditions. There is in nearly all marked cases a chronic pharyngeal catarrh which may involve the Eustachian tubes.

Etiology.—Hypertrophy of the tonsils is an exceedingly common condition in the cities of the seacoast and lake districts of the temperate zone. In a routine examination of 2,000 New York school children, Chappell found enlargement of the tonsils sufficiently marked in 270 cases to be considered pathological. The causes are constitutional and local. The condition frequently exists in certain families for several generations. It occurs in children who are in other respects healthy. According to Czerny, overfeeding may produce tonsillar enlargement just as it does enlargement of the adenoid tissue of the rhinopharynx. The most important of the local causes are attacks of acute or subacute pharyngitis. While it is true that attacks of acute inflammation are

often the cause of hypertrophy, it is also true that hypertrophy is one of the most frequent predisposing causes of acute attacks, and that it may be seen in children who have never had acute tonsillitis.

Symptoms.—Hypertrophy of the tonsils is rarely marked enough to cause any decided symptoms before the end of the second year, although occasionally in younger children enlargement sufficient to bring the two tonsils into contact may be seen. The most important local symptoms, formerly ascribed to hypertrophied tonsils, are now known to depend upon adenoid growths of the pharynx. As these conditions are so frequently associated, it is somewhat difficult to determine which symptoms are due to the tonsils alone. In a marked case, the most prominent symptoms are mouth-breathing, disturbed sleep accompanied by snoring, and nasal voice—the patient in some cases talking as though he had food in his mouth. There may be some difficulty in swallowing solid food. Enlarged tonsils may often be felt externally. As a consequence of the obstruction of the Eustachian tubes there may be deafness. Deformities of the chest, such as pigeon-breast, are occasionally seen, but probably depend more upon obstructed respiration by adenoids than by the tonsils.

There are seen in certain children tonsils which show only a very moderate amount of enlargement, but are of unhealthy appearance and are accompanied by low fever and other indefinite symptoms of illness which may persist for months. The tonsils appear to act in such cases as the avenue of absorption which results in a general infection.

The soft tonsils may diminish somewhat in size spontaneously. They sometimes shrink very decidedly after an attack of acute tonsillitis, scarlet fever, or diphtheria. As a rule the tonsils become firmer and harder as time passes. They usually increase in size up to a certain point, and then remain nearly stationary until about puberty, when they may diminish considerably. During intercurrent attacks of inflammation, the swelling is much increased, and the symptoms are proportionately aggravated. In cases of marked enlargement very little spontaneous improvement is to be looked for during childhood.

Treatment.—Very large tonsils are a source of continued danger to the patient, and in every case of marked hypertrophy treatment should be advised. The danger may be from Eustachian catarrh and deafness, or from repeated attacks of acute tonsillitis. But quite as important as these is the fact that they increase the liability to contract diphtheria, and add to the dangers both from diphtheria and scarlet fever. If the patient is removed from the locality in which acute tonsillitis is liable to occur, to a dry climate, considerable improvement is likely to result in a young child in whom the tonsils are soft, but not much is to be

expected in older children with hard, fibrous tonsils, except, perhaps, a cure of the accompanying pharyngeal catarrh.

No internal remedy offers much chance of benefit. Astringent applications may accomplish something in recent, but practically nothing in old cases. In every marked case, operation is the only thing which can be relied upon to effect a cure. For convenience of consideration, the cases may be divided into four groups: (1) Those in which the tonsils are nearly or quite in contact; (2) those in which they project only slightly beyond the faucial pillars; (3) those in which the tonsils, although large, are deeply imbedded; (4) diseased tonsils, though showing only moderate enlargement, especially when associated with tuberculous glands of the neck. All of the first group should unquestionably be operated upon, unless the patient's general condition is such as to forbid operation of any kind. In the second group operation is not indicated unless there are repeated acute attacks of inflammation. Whether an operation is done in the third group will depend upon the individual case. If there are frequent attacks of acute tonsillitis or evidence of involvement of the ears operation should be performed. In the fourth group operation is indicated if general symptoms are present.

Of the various operations proposed for the removal of hypertrophied tonsils complete enucleation is clearly to be preferred. It is a painful operation, some preliminary dissection is usually required, and hence general anesthesia is necessary. The risk of serious hemorrhage in children is slight, but preparations should always be made to control it as even with non-bleeders one can never tell how severe it may be. Enlargement of the tonsil subsequent to simple amputation is quite frequently seen, especially if the patient operated on is under two years old.

We have more than once seen physicians greatly alarmed at the gray wound on the day following tonsillotomy, the appearance being such as to lead in several cases to the diagnosis of diphtheria. It is seldom that any but good results follow the operation of tonsillotomy if properly performed. When adenoids of the pharynx are also present, the symptoms may depend more upon them than upon the enlarged tonsils, and little benefit is seen unless the adenoids also are removed.

CHAPTER IV

DISEASES OF THE ESOPHAGUS

MALFORMATIONS

Congenital anomalies of the esophagus are often associated with those of the lower part of the respiratory tract.

There may be, (1) Congenital fistula of the neck, due to a want of closure between the second and third branchial arches. This gives an external opening just above and to the outside of the sternoclavicular articulation, which communicates with the upper part of the esophagus or the lower part of the pharynx. (2) The esophagus may be absent, the pharynx ending in a blind pouch. (3) The esophagus may be obliterated in certain portions, being represented only by a fibrous cord. (4) There may be stenosis and dilatation or diverticula. (5) There may be fistulous communication with the trachea, existing either alone or associated with some of the other deformities mentioned. This is the variety which we have most frequently met with. From above, the esophagus usually terminates in a blind pouch. From below, it communicates with the trachea a short distance below the larynx. The two parts of the esophagus are usually connected by a fibrous cord.

Congenital narrowing of the esophagus and fistula of the neck are amenable to surgical treatment. The cases of complete obstruction in the esophagus are almost of necessity fatal, the patients dying from inanition four or five days after birth.

The symptoms of esophageal obstruction are regurgitation on attempts at swallowing and the impossibility of passing the stomach tube. An X-ray picture after the administration of bismuth often gives valuable information.

ACUTE ESOPHAGITIS

It is quite remarkable, considering the frequency of pathological processes in the pharynx, that these so rarely extend to the esophagus. Thrush, when very extensive in the pharynx, may involve the upper part of the esophagus; but there it gives rise to no new symptoms. Diphtheria of the pharynx may invade the esophagus, but this is seen only in rare instances. Diphtheria of the esophagus produces no symptoms by which it can be diagnosticated during life.

Catarrhal Esophagitis.—Catarrhal esophagitis is very rarely met

with. It may be caused by lacerations due to swallowing a foreign body, which may excite a simple catarrhal inflammation, or, if the foreign body is sharp and angular, lacerations may be produced which result in ulcerations of variable depth. The chief symptoms of catarrhal esophagitis are soreness and pain on swallowing. These lacerations, when slight, are healed in a few days, and are rarely followed by any after-effects.

Corrosive Esophagitis.—This is altogether the most frequent form, and the only one which is of clinical importance. The usual causes are the same as of corrosive gastritis, viz., the swallowing of caustic alkalis or strong acids. It is often in the esophagus that the most extensive injury is done. The effects are superficial or deep, according to the amount of the irritant swallowed and its degree of concentration. There may be simply a destruction of the epithelial layer, which is followed by no serious consequences, or the mucous membrane may be destroyed and the submucous coat invaded; rarely, however, does the injury extend to the muscular layer. If the patient survives the dangers incident to the irritant poisoning and the acute inflammation which follows, healing by granulation and cicatrization takes place, the contraction of the cicatrix gradually narrowing the lumen of the esophagus until stricture is produced.

The early symptoms of corrosive esophagitis are mingled with those of inflammation of the mouth, pharynx, and stomach. There is a burning pain in the parts, great thirst, and spasm of the esophagus on attempts at swallowing. There follows a period of acute inflammation of several days' duration, with great dysphagia and pain, in which the principal danger is edema of the glottis. After this the patient may be comparatively well until the symptoms of stricture begin, usually in from three to six months after the injury.

The indications for treatment in the early stages are, to neutralize the caustic in order to prevent if possible its deep action, to give oils, demulcent drinks and ice for the local effect, and morphin for the pain.

The treatment of esophageal stricture is purely surgical.

RETRO-ESOPHAGEAL ABSCESS

Acute retro-esophageal abscess occurs in infancy, though very rarely, the pathology being the same as in acute retro-pharyngeal abscess, the difference being merely one of location. A striking case of this kind occurred in the New York Foundling Hospital. An infant six months old was admitted with no loss of voice but with high fever (104° F.) and severe dyspnea which were the prominent symptoms until death occurred four days later. There was a leucocytosis of 100,000. At

autopsy an abscess was found containing about three ounces of pus between the esophagus and the spine, extending from the larynx to below the bifurcation of the trachea. Shortly afterward a very similar case occurred at the Babies' Hospital, following a retro-pharyngeal abscess which had been opened two weeks before. Similar abscesses have also been observed after acute pharyngitis with the acute infectious diseases.

Retro-esophageal adenitis, or enlargement of the lymph nodes in this situation without suppuration, is also rare. We once met with a case of this sort in which the gland formed a tumor nearly an inch in diameter at the upper part of the esophagus, causing pressure symptoms necessitating tracheotomy. The growth was at first thought to be malignant, but completely disappeared after a summer in the country.

Retro-esophageal abscess may result from the breaking down of tuberculous lymph nodes in the posterior mediastinum, and may give rise to symptoms like those which result from an abscess due to Pott's disease.

Perforation of the esophagus and a food-fistula connecting the esophagus and the trachea may result from ulceration caused by a tracheal canula or by a foreign body. This may be accompanied by abscess.

The most common variety of retro-esophageal abscess is that due to Pott's disease of the lower cervical or upper dorsal region. The symptoms are obscure, and an exact diagnosis is not often made during life. Death may occur quite suddenly when the previous symptoms have been so slight as to be easily overlooked. The following is a fair example:

A girl two years old was admitted to the Babies' Hospital with caries of the upper dorsal region of two months' duration. The patient was kept in bed and a plaster-of-Paris jacket applied. About a month later dyspnea was first observed; this was at times quite intense, and again almost absent. It was always on inspiration, expiration being easy. No explanation for this was found in the lungs. There was no difficulty in swallowing, and very little cough. After these symptoms had lasted for about a week, the child while eating was suddenly seized with violent dyspnea, and in a few moments became completely asphyxiated. Tracheotomy was immediately done, and by means of artificial respiration the patient was restored to comparative comfort. About two hours later a second attack occurred, and the patient died in an hour. At the autopsy there was found an abscess a little larger than a hen's egg, containing about two ounces of curdy pus, overlying the bodies of the first three dorsal vertebrae and communicating with them. These vertebrae were carious. The right pneumogastric nerve, an inch and a half above the bifurcation of the trachea, was compressed between the abscess and a large tuberculous lymph node, with the capsule of which it was blended. In the lungs were a few small tuberculous deposits and the usual conditions found in death by asphyxia. The dyspnea seems to have been of

nervous and not of mechanical origin, and caused by irritation of the pneumogastric. The fatal issue was apparently from an increase of the pressure upon the nerve.

We have seen but one other case, and this closely resembled the one reported. In the thirteen cases collected by Griffith the symptoms in all were much alike. Dyspnea, usually of a spasmodic character, was prominent in nearly all, and generally it was the most prominent symptom. It was more marked on inspiration, and often accompanied by a spasmodic cough, suggesting laryngeal stenosis. The voice was affected in but two cases, in one complete aphonia being present. It is striking that in no case was there any difficulty in swallowing, in marked contrast to retropharyngeal abscess. Swelling in the neck was noted in but three cases. Spinal caries was stated to be present in seven cases and absent in two. The final attack of asphyxia sometimes came without warning, sometimes was preceded for several days or longer by milder attacks.

The diagnosis of this condition is very difficult, and a positive diagnosis almost impossible. It may be suspected in cases of Pott's disease of the lower cervical or upper dorsal regions, when there is spasmodic inspiratory dyspnea, especially if accompanied by irritative cough. It should, however, be remembered that precisely similar symptoms may depend upon the irritation of a tuberculous node, and that the sudden asphyxia is exactly like that caused by the ulceration of such a node into the trachea or a large bronchus. The latter, however, may occur without the presence of Pott's disease. If the abscess is higher up, there may be a swelling on either side of the neck, just above the clavicle. In most of the cases there are no external signs of disease. Such abscesses are too low to be reached by digital examination of the pharynx. The attack of asphyxia may also be confounded with that due to the presence of a foreign body in the larynx.

The prognosis in cases of retro-esophageal abscess is exceedingly bad. Death usually results from pressure upon the pneumogastric, as in the cases reported. The abscess may rupture into the esophagus and recovery follow. This termination is very rare, but such a case has been reported by Knight. A fatal one is reported by Löschner and Lambl. The abscess may burrow along the esophagus into the abdominal cavity and excite peritonitis; finally, it may open externally.

But little is to be said under the head of treatment. The symptoms are rarely definite enough to justify a radical surgical operation. Tracheotomy gives but temporary relief to the asphyxia. This operation should be performed, however, in every case, because of the impossibility of making a diagnosis of retro-esophageal abscess from other conditions in which the operation might be curative.

CHAPTER V

DISEASES OF THE STOMACH

It is difficult wholly to separate diseases of the stomach from those of the intestine. Although in older children they are often quite distinct, in infancy they are more frequently associated; but at one time the gastric symptoms may be prominent, and at another the intestinal symptoms. Functional disorders particularly are likely to involve the whole tract. Serious organic lesions are more frequently limited in their extent either to the stomach or to the intestine. The former are rare, while the latter are very common. The diseases in which the stomach is alone or chiefly involved will be considered by themselves. Those in which both the stomach and intestine are involved are classed with the intestinal diseases, as the intestinal symptoms usually predominate.

DIGESTION IN INFANCY

The first step in the process of digestion in the newly-born infant is sucking. During this act the nipple is grasped between the lower lip and tongue below, and the upper lip and jaw above. The back of the mouth is closed by the palate. A strong downward movement of the lower jaw causes a partial vacuum in the mouth, and produces the suction force which causes the milk to flow. Sucking can be carried on only when the nose is free for respiration and the palate and upper jaw intact. Children with deformities of the mouth, like cleft palate and harelip, suck only with the greatest difficulty, and complete nasal obstruction prevents nursing.

The Saliva.—This is present at birth only in very small amount, and the part which it plays in digestion in early infancy is an insignificant one. During the third and fourth months it increases markedly in quantity, and at this time it possesses quite actively the power of transforming starch into sugar. This property is present only to a very slight degree during the early weeks.

The Stomach.—Our knowledge of the anatomy and physiology of the infant's stomach has been greatly increased through the use of the X-ray. The position varies considerably in normal conditions and very greatly in pathological conditions. The stomach is usually somewhat obliquely situated in the abdomen, not only from side to side, but from before backward, as the cardiac orifice is quite near the spine while the pylorus is much anterior. The pylorus is usually considerably to the right of

the median line and generally situated somewhat behind the pyloric third of the stomach.

When inflated after death the normal infant's stomach resembles a curved cylinder with a greatly shortened superior border. After the first year the great development of the fundus occurs and the shape is much like that of the adult stomach. During life the shape of the stomach varies greatly with the amount of food and gas it contains and with the condition of its muscular walls, whether relaxed or contracted. It enlarges with great facility with the introduction of food. In conditions when there is a lowered muscular tone, as in rickets or malnutrition, great changes in size, shape and position are met with. In some cases the stomach is almost entirely to the left of the median line. The abnormal shapes are temporary or permanent, according to circumstances, and no doubt have much to do with the facility with which the stomach empties itself during digestion.

In the nursing infant, food begins to leave the stomach almost at once, and within five minutes a very considerable proportion of the amount taken has often reached the intestine. At the end of half an hour the greater part of the food has usually left the stomach. In infants taking cow's milk, the food passes out more slowly but after the first few minutes food is seen in the intestines. The opening of the pylorus is much influenced by the reaction of the gastric contents. It normally opens when a certain degree of acidity is reached. The addition of alkalis to cow's milk markedly delays the emptying of the stomach. This is also influenced by the composition of the food; when the food contains a high fat percentage, emptying of the stomach is much delayed. The whey first reaches the intestine, afterwards the casein, and lastly the fat. Solid food is retained in the stomach a longer time than milk.

The stomach always contains gas, and, by the X-ray, after every feeding a large bubble of gas is seen above the food, often half filling the stomach. Most of this gas is air that has been swallowed. In conditions of disordered digestion the amount may be very great. There is a natural tendency for the stomach to contract and expel this gas after taking food; but if the infant is placed upon his back and kept there, this is mechanically impossible, as has been well shown by the investigations of C. H. Smith.

Gastric Digestion.—The rôle of the stomach in digestion is not so important in infants as in adults. The gastric part of digestion is only preliminary and partial; the major part of digestion takes place in the intestines. While the function of the stomach is largely that of a reservoir into which the milk is received and from which it is allowed to pass gradually into the intestines, certain definite changes take place

there chiefly owing to the activity of the rennet ferment and the gastric lipase. It was until recently believed that the action of the gastric juice was chiefly upon the protein of the food by virtue of the pepsin and hydrochloric acid contained in it. It has been shown, however, that for each gastric ferment a certain concentration of acid is necessary for its activity. In a large series of cases, different observers have determined that the concentration of acid in the gastric juice of normal infants fed upon cow's milk is low, much less than that of adults. Pepsin is inert in a solution of such weak concentration. It is therefore altogether probable that gastric digestion by pepsin is practically negligible. Nevertheless, pepsin is found in the stomach at birth and may even be demonstrated in the fetus as early as the fourth month.

The concentration of acid in the stomach, although insufficient for the action of pepsin, is sufficient for the activity of the rennet ferment and the lipase. Coagulation is the first change which milk undergoes in the stomach. Woman's milk coagulates in loose flocculi and quite imperfectly, while cow's milk coagulates in much firmer, more compact masses, owing to the larger amount of casein. The motility of the stomach plays an important part in digestion. The churning movements soon break up these casein masses into much smaller particles. Rennet has a feeble digestive action upon protein. Many good authorities consider that rennet is not a separate substance but that coagulation is one of the properties of pepsin. The question is as yet undecided but pepsin and rennet are always present in coresponding amounts. It has been shown that a lipase or fat-splitting ferment is present in the stomach even of infants and that it increases the activity of the pancreatic lipase. Its importance in the stomach is not clearly known.

Pepsin is found in the stomach at birth, and even in the fetus as early as the fourth month. In fifteen minutes after feeding the reaction of the stomach contents is always acid. Free hydrochloric acid can not usually be demonstrated until about an hour after feeding, then only in small quantities, and in very many cases not at all. The reason for this is, that the acid combines with the casein and the salts of milk, those of cow's milk in particular having a great power of combining with hydrochloric acid.

The duration of gastric digestion varies with the age of the infant and with the food. During the first month the stomach of healthy nursing infants is usually found empty in an hour and a half after feeding, often in one hour. In those taking cow's milk the average is at least one hour longer. In infants from two to eight months old the average is two hours for those receiving breast milk, and two and a half to three and a half hours for those fed upon cow's milk. The time is influenced by the size of the meal taken and by the composition of

the food. The higher the proportion of fat in the meal, the longer the food is retained in the stomach, and also the smaller the amount of gastric juice secreted. Very little absorption takes place from the stomach. There is here absorbed a certain proportion of sugar and peptones, but practically no water, fat, or salts. The amount of gastric juice secreted is very large. In experiments upon animals it has been shown to be nearly as great as the volume of milk taken.

The bacteria of the stomach are very few as compared with those of the intestine, and no varieties are constantly present.

The Intestines.—The length of the small intestine at birth is about nine feet; that of the large intestine about eighteen inches. The great length of the sigmoid flexure is the most striking peculiarity, this being nearly one-half the length of the large intestine.

Intestinal Digestion.—All the important elements of food—protein, carbohydrates, and fat—are acted upon by the pancreatic juice. The protein is converted into peptones by trypsin. The digestion of protein is completed by the erepsin of the intestinal juice, which converts peptones and albumoses into amino acids. In this form the nitrogenous portion of the food is finally absorbed.

The amylolytic ferment of the pancreas has the power of converting starch into maltose. This action is feeble during the first four or five months, but is present even in early infancy. Milk sugar is changed into galactose and glucose, and cane sugar and maltose into glucose through the agency of the intestinal and pancreatic juices. Fats are partly emulsified and partly saponified by the pancreatic juice in connection with the bile.

Absorption.—From the small intestine absorption takes place very rapidly. The protein is absorbed in the form of peptids and amino acids. Sugars of all varieties are changed to glucose during absorption. Fat is absorbed in the form of fatty acids and soaps; but in their passage through the wall of the intestine the fatty acids are converted into neutral fats. Absorption from the large intestine, except of water, is quite imperfect. Fat absorption is very slight. Sugar, salts, and peptones, however, may be absorbed with moderate facility.

Intestinal Bacteria.—For the fundamental work upon this subject we are indebted to the researches of Escherich. Bacteria are absent from the entire gastro-enteric tract at birth. They quickly enter by the mouth and rectum, and by the end of twenty-four hours they are usually found in all parts of the intestinal tract. The meconium bacteria are derived from the inspired air, and hence vary somewhat with surroundings. As soon as the ingestion of milk begins these varieties are displaced, and throughout the period in which the infant has this food exclusively, there have been found in healthy conditions but few varieties which are con-

stantly present. These are the *b. lactis aerogenes* the *b. coli communis*, and the *b. bifidus*. The number of bacteria varies in different parts of the intestine. They are found in greatest numbers in the cecum and colon, and are relatively few in the small intestine. The *b. lactis aerogenes* is found most abundantly in the upper part of the small intestine, in small numbers only in the colon, and usually there are none in the feces.

The *b. coli communis* is found in but small numbers in the upper small intestine, becoming more abundant lower down. In the colon and in the feces it is present in considerable numbers. The most abundant organism in the large intestine, however, is the *b. bifidus*. A change from a milk diet to a mixed diet of meat and farinaceous food produces a marked change in the character of the intestinal bacteria.

Feces.—The first discharges after birth consist of meconium; this is of a dark brownish-green color, semi-solid, and usually passed from four to six times daily during the first two or three days. On the third day the stools begin to change in character, and by the fourth or fifth day they have usually assumed the appearance of normal milk-feces. Under many abnormal conditions the stools may continue to have the character of meconium for a week or more. Meconium is composed of intestinal mucus, bile, the vernix caseosa, epithelial cells from the epidermis, hairs, fat-globules, and cholesterin crystals. For its formation there are necessary the secretions of the intestine and the liver and the swallowing of a considerable amount of amniotic fluid.

Milk-feces.—The amount of feces discharged daily by a healthy nursing infant is from two to three ounces (60-90 gms.). Such stools have the color of the yolk of egg but may be paler, and from time to time even slightly greenish. They are seldom entirely smooth and homogeneous but usually contain a large number of small light-yellow particles. The consistency is butter-like but often rather looser than this. Under normal conditions the stools are never watery. The reaction is acid, and there is a slightly sour but not unpleasant odor. The reaction is due to the presence of fatty acids or lactic acid. The color depends upon bilirubin. The stools of an infant fed upon cow's milk may, in conditions of perfect digestion, differ in no respect from those just described; usually, however, they are firmer, rather more homogeneous, of a paler yellow color, and may be neutral or even alkaline in reaction. The normal stool of a nursing infant contains about 85 per cent of water and 15 per cent of solids; that of one taking cow's milk has about 80 per cent water and 20 per cent solids.

The only gases present are hydrogen and carbon dioxid. Sulphuretted hydrogen and marsh gas, to which the odor of adult stools is largely due, are not present.

The solids of the stools are chiefly fat, salts and nitrogenous matters. Sugar is not found, but its derivative, lactic acid, may be present in small amount. The fat makes up from 20 to 40 per cent of the dried matter, and is in the form principally of soaps and fatty acids, with a smaller proportion of neutral fats. The inorganic salts form about 10 per cent of the solids of the breast-milk stool, and from 20 to 35 per cent of the solids of the cow's-milk stool, about three-fourths of this being calcium phosphate. The nitrogenous elements of the cow's-milk stool make up about 25 per cent of the dried residue, but only a small proportion of this represents unabsorbed protein. They are chiefly derived from the intestinal secretions and the bodies of bacteria. Amino acids, representing unabsorbed food protein, make up from 2.4 to 24 per cent of the nitrogen of the stool. The protein of woman's milk is almost entirely absorbed, and that of cow's milk largely so, under almost all conditions.

A healthy nursing infant absorbs about 85 to 90 per cent of his ingested fat, about 95 per cent of his protein, and about 80 to 85 per cent of his salts. A healthy infant taking cow's milk absorbs about 85 to 90 per cent of his ingested fat, about 90 to 95 per cent of his protein, and about 60 per cent of his salts.

The biliary elements present in the stool are hydrobilirubin, unchanged bilirubin, and cholesterin. The presence of biliary acids is doubtful. Mucus is always present in considerable quantity.

Microscopically there are seen epithelial cells, chiefly of the columnar variety, a few round cells, mucous corpuscles, fat globules and crystals of fatty acids, cholesterin, mucin, crystalline inorganic salts, sometimes bilirubin in crystals, yeast fungi, and bacteria in immense numbers.

If the infant is taking a food containing starch, this may appear to a greater or less extent in the stools, a larger amount in the case of very young infants.

The number of stools of breast-fed infants during the early weeks is from two to six daily. After the first month two stools a day are the average; many infants have three, many others but one. With modified cow's milk the stools are seldom more than one or two a day and there is frequently constipation.

As soon as an infant is put upon a mixed diet, the peculiar characters of the stools disappear, and they come to resemble more closely those of the adult, though remaining softer throughout infancy. They become darker in color and assume the adult odor, while retaining their acid reaction. The bacteria, while still in great numbers, are more varied than are met with in milk-feces.

MALPOSITIONS AND MALFORMATIONS OF THE STOMACH

The stomach is sometimes in the thoracic cavity in cases of diaphragmatic hernia. It may be found in a vertical (fetal) position, variously adherent to the colon and small intestine. Malformations are much less frequent than those of other parts of the alimentary tract. There may be atresia or stenosis at either orifice, and very rarely a constriction is found near the middle of the organ, dividing it into compartments. The symptoms of atresia at either orifice are persistent regurgitation or vomiting, and death in a few days from inanition.

HYPERTROPHIC STENOSIS OF THE PYLORUS

This condition known also as *congenital stenosis of the pylorus,* or simply as *pyloric stenosis of infancy,* is not an uncommon one. It is characterized by persistent vomiting, constipation, wasting, marked visible gastric peristalsis, and usually a palpable tumor. It is a serious condition, and unless recognized early and properly treated it has a high mortality. It is seen in early infancy, usually in the first two months of life but seldom in the first two weeks. Fully four-fifths of the cases occur in male infants. It has no relation to feeding, the large proportion of recorded cases having been seen in nursing infants.

The pathology of stenosis of the pylorus in early infancy is somewhat obscure and at the present time quite diverse views are held. It is believed by some that the primary and essential condition is one of spasm; that the hypertrophy when it is present is secondary; that in a very considerable proportion of the cases there is only pylorospasm without hypertrophy; that with proper medical treatment most cases recover, and that surgical intervention is rarely called for. The other view and that which seems to harmonize best with the clinical symptoms and the pathological findings is that the primary condition is one of hypertrophy which is congenital; that to this, spasm is added; that in all cases both factors—hypertrophy and spasm—are present; that while the cases differ in degree they are the same in kind; that while many of the milder ones may recover without operation the severer ones should be treated surgically. Spasm certainly plays an important part in the production of symptoms; but to regard this condition as one essentially of muscular spasm seems to us erroneous.

The appearance of the pylorus when seen at autopsy or operation is remarkably uniform. It forms a hard, whitish tumor about the size of

a peanut, of almost cartilaginous consistency. Its lumen may be so narrowed as barely to admit a fine probe, while the normal pylorus will usually admit a No. 21 sound, French scale. Frequently water can not be forced through the stenosis owing probably to the fact that the mucous membrane is thrown into folds. The walls of the stomach are often hypertrophied, especially toward the pyloric end. The stomach is usually much dilated; its lower border may be below the navel. On section the pylorus is found to be much thickened and by microscopical examination this is seen to be chiefly of the circular muscle fibers. This coat appears to be two or three times the normal thickness. The other coats —submucous, mucous and longitudinal muscular—are thickened but to a much less degree.

Symptoms.—The clinical picture is a striking one. Symptoms rarely begin in the first week of life. An infant who for the first two or three weeks has usually shown no signs of gastric disorder, and often has been nursing and gaining regularly in weight, begins, without evident cause, to vomit; at first occasionally, but soon habitually. This vomiting in a short time becomes forcible, projectile. It may be of this type almost from the outset. Changes in diet have but a temporary effect upon it, or none at all. The bowels are constipated. The infant wastes steadily, the scales often showing a loss of one or two ounces a day. There is no fever and little or no evidence of pain. There is progressive failure in nutrition and death may occur from exhaustion in from four to six weeks from the beginning of marked symptoms.

Vomiting.—The manner of vomiting is characteristic. It is more forcible than that seen under any other condition. An infant will often fairly shoot out the contents of the stomach to a distance of three or four feet. Food frequently comes through the nose. The vomiting usually has a relation to the taking of food. It most frequently comes directly after feeding, sometimes even while the child is still at the breast. After an attack of vomiting, nursing is sometimes resumed with avidity, showing a distinct absence of the usual symptoms of gastric indigestion. All the food is generally expelled at one time. The frequent regurgitation of small amounts is unusual. Generally vomiting does not occur at night unless the child is nursed at that time. The vomited matters at first consist only of food, often but little changed. After a time there is mucus, sometimes in large quantities. The amount vomited at one time is often considerably greater than the feeding just taken, indicating a considerable retention of food in the stomach. Some of these children vomit regularly after every feeding; others retain two or three feedings and then expel the whole amount. The frequency of vomiting varies from once or twice to six or eight times a day. Owing to the loss of fluid by vomiting the urine is usually very scanty. There

is no uniform change in the gastric secretions, but there is frequently hyperacidity present.

Bowels.—Obstinate constipation is the rule. It is due to the fact that so much of the food taken is vomited. If the pyloric obstruction is complete the stools resemble meconium.

Wasting.—Progressive wasting is one of the striking symptoms, and a close observation of the weight one of our best guides to the progress of the case. If the loss amounts to two or three ounces a day the condition should be considered most critical. The rate of the loss depends naturally upon the completeness of the obstruction and it is proportionate to the amount of vomiting and the degree of constipation.

General Appearance.—At first nothing abnormal is seen, but soon

FIG. 31.—GASTRIC PERISTALSIS IN PYLORIC STENOSIS. (Thomson.)
Patient eight weeks old.

all the evidences of rapid malnutrition are present, without, however, the other usual symptoms of indigestion, such as might be expected with the vomiting. The tongue is usually clean; the appetite often voracious; there are no eructations of gas; the breath is sweet, and the color usually good.

Peristalsis.—On examination of the abdomen the epigastrium is usually full and the lower half of the abdomen may be sunken. If the skin is bared and the patient placed in a good light the characteristic peristaltic waves are seen which are the most diagnostic feature of the disease. One should not expect to see them if the stomach is empty; they are best seen immediately after taking food or water. When not appearing spontaneously they may often be excited by slight friction or tapping of the epigastrium. There is seen a slowly moving wave from left to right. First a ball-like tumor appears just below the ribs on the left side (see Fig. 31). It is usually about one and a half to two inches in diameter and slowly moves toward the right. It usually disappears just beyond

the median line. Sometimes one wave is quickly followed by another. Peristalsis of the intestine, in rare cases, may somewhat resemble these movements; but typical gastric contractions can hardly be mistaken for anything else. After marked peristalsis vomiting frequently occurs.

Tumor.—The hardened pylorus can with experience be felt in most cases. It may be obscured by distention of the stomach or the colon or by enlargement of the liver. The pylorus may be displaced. The position of the tumor is therefore of less importance in diagnosis than its character. It is usually felt about one and a half to two inches below the free border of the ribs, just inside of the right mammary line. It may be felt only during active peristalsis. It appears somewhat smaller than the little finger and about three-fourths of an inch long.

Gastric Retention.—The prolonged retention of food in the stomach is one of the characteristic features of pyloric stenosis. In healthy nursing infants the stomach is regularly found empty at the end of three hours, often at the end of two hours. But if stenosis is present, food in considerable amount is almost invariably found after three hours and, unless vomiting has occurred, usually after four hours. Sometimes this is also the case when there has been vomiting. This retention varies in amount, but when there has been no vomiting for several hours a larger amount may be removed than the last feeding taken, sometimes twice as much. Early in the morning after fasting eight or ten hours the stomach may contain three to four ounces of partly digested food. Abnormal gastric retention is one of the diagnostic features of pyloric stenosis. Gastric retention is best estimated by the removal of the stomach contents by means of suction. By this it can be determined how rapidly the food leaves the stomach quite as accurately as by the X-ray and in a much more convenient way. Not only is the X-ray unnecessary for diagnosis, but the administration of the large doses of bismuth for diagnostic purposes is objectionable and may even be dangerous in these very young patients. We have known of two instances in which its use was apparently the factor which determined the fatal result.

Course of the Disease.—Two types of cases are seen: (1) the severe form, in which the peristalsis and vomiting are but little influenced by medical treatment; the loss of weight is continuous and often amounts to two or three ounces a day; there is very little fecal matter in the stools; the constipation is very marked, and, unless relieved by operation, the condition generally proves fatal in from two to four weeks; (2) the mild form, in which the symptoms, though characteristic, are all much less marked, gastric peristalsis and tumor are present, but the vomiting is only occasional, fecal stools are passed, the loss of weight is not so marked and progress may alternate with periods of improvement in which there is gain in weight. Very many of these cases recover

without surgical aid, the chief danger being that the feeble infant is cut off by intercurrent disease. Whether there is a chronic form of infantile stenosis which persists into later childhood seems probable, but is not yet established.

Diagnosis.—The diagnosis of pyloric stenosis of infancy is usually easy after a few days of observation, but may be impossible at the first examination. The time of onset and nature of the vomiting are strongly suggestive but not quite conclusive. The abrupt development in a nursing infant from two to four weeks old of severe vomiting without assignable cause and its persistence in spite of all treatment, should always lead to the suspicion of pyloric stenosis. The diagnostic features of this condition are three: waves of gastric peristalsis, abnormal gastric retention and a tumor. The existence of a tumor may be a matter of some uncertainty in many cases, but its presence is of considerable positive value. The condition has been mistaken for cerebral disease on account of the projectile vomiting and chronic constipation; for renal disease, because of the vomiting and scanty urine. Usually, however, the only difficulty is to distinguish between the vomiting of gastric indigestion and that of pyloric stenosis. Gastric indigestion is an exceedingly common symptom in infancy; but it is not very common in nursing infants and rarely develops suddenly. The vomiting is apt to be in small quantities and many times repeated and generally occurs at a longer period after feeding. There are undoubtedly some cases of gastric indigestion in early infancy in which a temporary pylorospasm occurs, but this condition is quite different from the one we have under consideration. The existence of persistent spasm of the pylorus without hypertrophy has yet to be proven.

Impairment of motility is a symptom of gastric indigestion but in this condition the food seldom remains in the stomach for so long a time or in such amount as in stenosis. Besides it is unaccompanied by gastric peristalsis or a tumor.

Congenital obstruction of the duodenum or other part of the small intestine may lead to persistent, forcible vomiting and, if the obstruction is high up, even to visible gastric peristalsis. But in these cases, whether due to stenosis, atresia, twisting or pressure from bands, the symptoms appear soon after birth and the severe forms are fatal in a few days. The vomited matters consist of bilious material.

Prognosis.—Statements regarding prognosis will depend much upon what cases are included under the diagnosis. Limiting the term to the cases defined in the beginning of this article, the condition must be considered a serious one, often ending fatally unless properly treated. By older methods of treatment, fully 50 per cent of the children died. Of the last 50 operations done at the Babies' Hospital, the mortality was

28 per cent, but these figures include a considerable number of cases admitted very late in the disease in which the patients' condition was apparently hopeless when operated upon. With a fairly early diagnosis and resort to surgical treatment, the mortality in skilled hands should not be over 10 or 15 per cent. In any given case the unfavorable symptoms are, rapid and continuous loss in weight, i. e., one to three ounces a day, continued vomiting and meconium-like stools, both showing that little or nothing passes the pylorus. The lower the body weight has fallen before operation, the worse the prognosis, although we have seen a child recover whose weight at the time of operation was only four and a quarter pounds.

In cases not operated on complete recovery may result, though the tumor and active gastric peristalsis may persist for seven or eight months. Whether this condition may give trouble in later life cannot yet be definitely stated. The hypertrophy is certainly very slow in disappearing. A tumor has been found at autopsy in children dying of intercurrent disease as long as six months after recovery from all symptoms.

Of 122 cases which have come under our observation, chiefly in the Babies' Hospital, the general mortality was 43 per cent. But these figures include a very large proportion of severe cases, and many that were admitted very late and were in a hopeless condition.

Treatment.—The treatment adopted will depend upon the type of case with which we have to deal. With all cases, medical treatment should be given a faithful trial. If the patient is seen early this may safely be continued for a period of at least one or two weeks. With a large proportion of those previously classed as belonging to the mild type, medical treatment will be successful. The cases which are likely to recover usually show decided improvement in a few days,—less vomiting, fecal stools, diminished peristalsis and a stationary or slight gain in weight. If, however, when first seen, symptoms have already lasted three or four weeks without material improvement, or if there has been a steady though not rapid loss in weight, operation should be advised. Though some of these cases might recover without it, the risks of waiting are greater than the risks of the operation. Again, operation should be resorted to early in all cases classed as the severe type which show no improvement by medical treatment in a few days. If the child's condition is bad, no delay is admissible.

Medical Treatment.—This is carried out on the theory that the pyloric spasm to which symptoms are chiefly due will gradually subside if nutrition can be maintained. It consists in diet and stomach washing. If a child is nursing and the milk is normal, weaning is not advisable. Small meals, not too near together, are essential. The breast should

be given at four-hour intervals, and the nursing period varied from two to five minutes, according to the amount obtained. It is often advantageous to pump the breasts and give a measured amount of breast-milk. Usually for a child a month old not more than two ounces should be allowed at one feeding. On no account should an infant be weaned immediately because of the development of the symptoms of pyloric stenosis. For some infants who have been artificially fed nothing succeeds as well as a wet-nurse. The chief objection to the breast-milk is its high fat which sometimes increases the vomiting.

For infants who are artificially fed a few general principles are pretty well established. In all milk formulas the fat should be low, usually less than that in formulas from whole milk. The formulas from skimmed milk usually succeed best. Feeding should be regular and not oftener than every three hours, and in many cases a four-hour interval is better. The amount given at one time should be from one and a half to three ounces.

Stomach washing is useful to empty the organ of food and mucus and seems to have some effect in allaying spasm. Water used should have a temperature of 108° to 110° F. and be rendered alkaline by the addition of one per cent of bicarbonate of soda. The washing should be done about two and a half hours after feeding, and repeated twice in twenty-four hours. It should be continued for a considerable period. In cases which recover it has often been found necessary for six to eight weeks, twice daily, and for three or four months once daily. Hot applications over the epigastrium are of little value in relaxing spasm. The administration of drugs, especially preparations of opium, is not to be relied upon.

In the milder cases the effect of stomach washing and careful feeding is to bring about a gradual lessening in the vomiting and gastric peristalsis, though it is usually some weeks before any material gain in weight is seen. Relapses are not uncommon.

Surgical Treatment.—Several different operations have been proposed, but only two have been frequently performed: gastro-enterostomy and a form of pyloroplasty known as Rammstedt's operation. In the latter the circular muscular layer is divided externally without opening the stomach. After this is done the pylorus opens and food passes readily into the intestine. It is as yet too early to speak finally as to the relative value of these two operations; but our observations upon over fifty cases treated by the latter operation lead us to prefer it. It requires but fifteen or twenty minutes, while gastro-enterostomy requires fully twice as much time; it involves much less handling of the abdominal viscera; it is followed by much less shock; there is less subsequent vomiting; convalescence has been more rapid; fluids pass directly through the natural

channel, and diarrhea has been seen much less frequently than after the other operation. While few of our cases have as yet been followed longer than a year and a half, up to the present time all the advantages seem to be with this operation.

The after-treatment is exceedingly important and the outcome depends almost as much upon this as upon the operation itself. Feeding may be begun as soon as the child has recovered from the anesthetic. The food, if possible, should be breast milk. By all possible means should the mother's milk be conserved. Beginning with one or two teaspoonfuls, it may be given every two hours, alternating with the same amount of water, the amount being gradually increased and the interval lengthened so that the child at the end of forty-eight hours is usually taking one ounce of milk every three hours, and the same quantity of water between the feedings. At the end of a week or ten days the infant may in most cases be put back to the breast, but the amount taken at one time should be limited and the nursing closely watched. In the beginning not over one or two minutes' nursing should be permitted. Unless the bowels move freely, from one-half to one teaspoonful of castor oil should be given twenty-four hours after operation. The vomiting which not infrequently occurs occasionally for one or two days may sometimes be relieved by keeping the head of the child's crib considerably elevated, or supporting him in a semi-sitting posture and by putting him over the nurse's shoulder from time to time to enable him to get rid of the gas in the stomach, or by the occasional introduction of the stomach tube.

Hypodermoclysis is a procedure of much value, sometimes before and nearly always after operation, since these infants are usually suffering greatly from the lack of water. From 150 to 240 c. c. of a normal saline solution may be given, and in bad cases repeated daily for three or four days. It is often advantageous to add to the saline 3 per cent of glucose.

The shock of operation with most of these patients is surprisingly little. With careful feeding favorable cases begin to gain weight within a few days after operation and in a few weeks are apparently as well as ever. Cases we have followed for three or four years do not suffer subsequently from digestive disturbances more frequently than do other children.

Operation is to be looked upon not as a last resort in a condition well-nigh hopeless, but as offering in the hands of an experienced surgeon an excellent prospect of recovery. The statistics of operation by the three American surgeons, Downes, Scudder and Richter, who have published the largest number of cases, show a mortality of but 28 per cent in 122 operations. Of the last 62 consecutive cases operated on in the

Babies' Hospital, nearly all by Downes, there were 44 recoveries. The results of medical and surgical treatment cannot well be compared, for they usually refer to quite different groups of cases.

VOMITING

Vomiting is one of the most frequent symptoms of disease in infants and young children, and occurs from a wide variety of causes. The physician must have in mind both its common and its uncommon causes. Vomiting takes place with great facility in young infants even from slight causes, owing to the position and shape of the stomach.

1. *Vomiting from Overfilling of the Stomach.*—This is often seen in nursing infants, and there may be no other symptom of disease. It comes within a few minutes after nursing, is easy and without effort, and the food is but little changed. It may be excited by moving the child or making undue pressure upon the stomach, and requires no treatment except to diminish the quantity of food. Vomiting also comes from distention of the stomach with gas, most of it being air which has been swallowed with nursing or feeding. It is relieved by placing the child in an upright position or over the shoulder before he is put in his crib.

2. Vomiting is almost invariably present in cases of *acute gastric indigestion and acute gastritis.* With the former it does not usually come immediately after feeding, and it may be delayed for several hours; with the latter it is usually persistent. The vomited matter consists of the contents of the stomach, but often mucus, and, in severe cases, bile and traces of blood may be vomited for some time afterward.

3. In the *hypertrophic stenosis of the pylorus* of early infancy, uncontrollable vomiting without fever is the principal symptom. (See previous Chapter.)

4. In *acute intestinal obstruction* vomiting is rarely absent, and in most cases it is persistent. In the newly-born, persistent vomiting is almost invariably dependent upon congenital obstruction of the intestine, which is most frequently in the duodenum. In malformations of the colon and rectum it is less constant and appears later. In intussusception, vomiting is forcible, immediately excited by the taking of food, and is at first bilious, but later may become fecal.

5. Vomiting is a frequent and almost a constant symptom of acute peritonitis, whether localized or general, of which appendicitis is the usual cause. It is then associated with abdominal distention, tenderness, and fever.

6. In certain *nervous diseases,* especially tumor of the brain and

acute meningitis, whether cerebrospinal or tuberculous, vomiting is very common. Cerebral vomiting is usually forcible or projectile. It may have no relation to meals.

7. In infants, and less frequently in older children, vomiting is one of the most frequent symptoms to mark the *onset of acute febrile diseases* especially the beginning of scarlet fever, pneumonia, and malaria.

8. An accumulation in the blood of various *toxic* materials may provoke vomiting; the best known example is uremia. In cyclic vomiting it is quite probable that the cause is the accumulation of some toxic substance in the blood. The absorption of poisons taken in with milk or other food, or developing in the gastro-enteric tract, may excite vomiting. In some of these conditions it is possible that the vomiting may be eliminative. The cases dependent upon renal disease are discovered by examination of the urine. The other forms are often exceedingly obscure, and recognized only by the exclusion of all other causes of vomiting.

9. Vomiting may be *reflex* from irritation in the pharynx. This is frequent in young infants, who may induce vomiting by stuffing the fingers into the mouth. In certain cases the irritation from worms in the intestinal tract may cause vomiting, and it is possible that even dentition may produce it.

10. *Habit* is a frequent cause, in cases of chronic vomiting, especially in children of a neuropathic constitution. In young infants a habit may be acquired of regurgitating the food very much in the manner of the ruminating animals. Soon after feeding there is seen a movement of the mouth and fauces resembling swallowing, then the food appears in the mouth and is ejected without force. This may be repeated until a large part of the food taken is lost. The habit once formed may continue for months, the nutrition of the infant often suffering to a serious degree. To this condition the name *rumination* has been given. It is not difficult of recognition if the infant is closely observed after feeding.

The most successful treatment is the administration of a food so thick that it cannot be readily regurgitated by the infant in the manner described: e. g., four tablespoons of barley flour is cooked for thirty minutes in one pint of whole milk. From one to two ounces are given every three or four hours with a spoon, as it is too thick to go through a rubber nipple. Water should be given between feedings. Some children have the power of vomiting at will anything in the nature of food which they do not like, yet who retain other food without difficulty. One such child would tolerate large doses of quinin, to which he had no aversion, without the slightest disturbance. Habit is a potent cause in continuing vomiting when from any cause it has occurred frequently.

In children who have this habit the most trivial cause will provoke it. It may be present without any other sign of gastric disease, and appears simply to depend upon exaggerated reflex irritability of the organ. We have seen a number of children who up to the third or fourth year objected so strenuously to taking solid food that they would immediately vomit it, no matter of what variety or in how small a quantity, although fluids were taken and easily digested.

11. *Chronic vomiting* may depend upon habit, as just described, or upon chronic indigestion; or it may be associated with chronic pulmonary disease—vomiting here being excited by the attacks of coughing, at first only when the paroxysms are severe, and later even when they are slight.

The diagnosis of a case in which vomiting is the chief symptom may be difficult. The first important distinction to be made is between cases in which the vomiting is of gastric origin, and those in which it depends upon other causes. It is only by a careful consideration of the associated symptoms that an accurate diagnosis can be reached.

The treatment of vomiting is the treatment of the cause upon which it depends.

CYCLIC VOMITING

This is a frequent condition and up to recent times has attracted but little attention except in this country. Although the clinical picture is a very clear and definite one, its exact pathology is undetermined. It has also been described under the names *periodical vomiting* and *recurrent vomiting*. It is characterized by periodical attacks of vomiting, which recur at regular or irregular intervals of weeks or months, apparently without any adequate exciting cause. The usual duration of the attacks is two or three days, during which all attempts to control the vomiting are usually without avail, but at the end of this time it generally ceases spontaneously.

Etiology.—The first attacks are usually seen between the ages of two and four years, but they may date back to infancy. The two sexes seem to be almost equally liable. A few of the patients are strong children, but the great majority are rather delicate and of a highly nervous temperament. The cases are seen chiefly in private practice, often occurring among those who have the best surroundings. In most cases the antecedents of patients are of a neurotic type. The attacks are not usually traceable to distinct or flagrant errors in diet, and yet the habitual diet seems to bear some relation to the disease. The exciting cause is often a nervous one—great fatigue or unusual excitement, sometimes a railroad journey or a child's party; in many instances it

seems to be induced by some minor illness having no relation to the digestive tract, such as an attack of tonsillitis or bronchitis. In children subject to this condition serious diseases, such as scarlet fever or measles, may be ushered in by prolonged and repeated vomiting, which usually ceases before the end of the febrile period. General anesthesia, specially by ether, is very likely to precipitate an attack.

Symptoms.—The clinical picture presented by these cases is very characteristic, and is well illustrated by the history of the following case:

The patient was a well-nourished boy of six years when he first came under treatment. He belonged to a neurotic family, and the attacks dated back to infancy. From this time they had recurred usually at intervals of a few months; occasionally five or six months would pass without one. The symptoms in all the attacks were similar in kind, differing only in degree. They were preceded by a prodromal period lasting from twelve to twenty-four hours, marked by languor, dulness, dark rings under the eyes, loss of appetite, and a general sense of discomfort in the epigastrium. At this time the temperature was generally slightly elevated. The vomiting then began suddenly. It was attended with great retching and distress; it was often repeated every half-hour or hour for two days. On one occasion it occurred seventeen times in a single night. Vomiting was immediately excited by the taking of any food or drink, but it occurred also when nothing was taken. The vomited matters consisted of frothy mucus and watery material, frequently streaked with blood, apparently from the violence of the emesis, and often containing bile. The temperature usually fell to about 100° F. when the vomiting began, and continued at or below this point throughout the attack. By the end of the second day the exhaustion was very marked—so severe, in fact, as apparently to threaten life. The child lay in a semi-stupor, with eyes half open, lips and tongue dry, rousing at times to beg for water. The pulse was rapid and weak, and sometimes slightly irregular. There was no distention of the abdomen; it was usually flattened. By the third day the vomiting became less frequent and then ceased entirely. Convalescence was rapid, and by the end of the week the boy was almost as well as usual. The attacks continued to recur at gradually lengthening intervals until they finally ceased altogether at about the twelfth year.

A great number of these cases come under observation. The usual duration of the attacks is one to three days. In one child they lasted regularly for five days. Occasionally a severe attack will last a week. The average number of attacks is four or five a year.

Prodromal symptoms are present in most of them—headache, general languor, coated tongue, and anorexia are the most frequent; in some there is marked constipation, with a history of very white stools

for some time. But it is not uncommon for an attack to occur in the midst of apparently perfect health. The tongue is usually coated at the beginning of an attack, and at its height it is often dry and brown. The abdomen seems empty and its walls sunken; pain and tenderness are both rare. The bowels are usually constipated and move only with difficulty by artificial means. Very exceptionally there may be diarrhea with foul stools.

There is, as a rule, no desire for food, but the continual cry is for water to quench the constant, burning thirst. The pulse after the second day becomes rapid, soft, and often somewhat irregular. The respiration is shallow, and at times this also may be irregular. The temperature is usually under 100.5° F., rarely it may be 102° or 103° F. The low temperature is a point of much diagnostic value. The patients are dull, apathetic, and wish to be left alone. Headache is very common.

The disposition to vomit is sometimes so great that patients are afraid to move or even to talk lest it may be provoked. The vomited matter is often large in amount, considering that the patient is fasting. It is essentially gastric juice, containing free HCl, mucus, serum, many epithelial cells, and often traces of blood. Less frequently vomiting may occur only two or three times a day. The urine is concentrated, and frequently contains at the height of the attack a trace of albumin, a few hyaline casts, and some blood cells. An increase in the renal secretion may be the first sign of improvement. There is usually an excess of indican both during and between attacks. A condition practically constant, and first pointed out by Marfan, is the presence in the urine of acetone, diacetic and β oxybutyric acids. These substances often appear in the urine in large amounts so early in the attack that they can not be ascribed to starvation, and therefore may be of diagnostic value. However, it should be emphasized that acetonuria is not the same as acidosis; the latter is uncommon in cyclic vomiting, but occurs in a severe form.

The Nature of the Attacks.—These cases have little in common with the ordinary attacks of indigestion. With our present knowledge they are to be regarded as explosions due to faulty metabolism. They have much in common with attacks of migraine which in later life not infrequently replace the attacks of vomiting. The studies of Hilliger upon a child subject to attacks of cyclic vomiting showed an interesting reaction to the withdrawal of carbohydrates. When the carbohydrates in the diet were greatly restricted the blood sugar fell at once to one half the normal amount and vomiting was precipitated. Normal children were not influenced by the temporary withdrawal of carbohydrates.

Prognosis.—Although these patients very often seem to be most alarmingly ill, the danger to life is slight. We have seen but three fatal cases, and in one the diagnosis is open to question, as no autopsy could

be obtained. Griffith reports two fatal cases, the autopsy in one showing nothing definite. The probabilities are always in favor of a recurrence of the attacks. In most of the patients who have been observed they have extended over a series of several years, although by a careful regimen much may be done to reduce their frequency and diminish their severity. In a considerable proportion of cases they may be stopped altogether. Toward puberty there appears to be a strong tendency to spontaneous recovery.

Diagnosis.—Organic disease of the brain and kidneys must first be excluded. The first attacks witnessed may strongly suggest the onset of tuberculous meningitis; and only the course of the symptoms may show that this is not present. Usually a history of many previous attacks may be obtained. From acute indigestion, cyclic vomiting is differentiated by the fact that the attacks are not brought on by indigestible food, and also by the persistence of the vomiting, and the early presence in the urine of the acetone bodies. It is distinguished from gastritis by its severity, the shorter duration of its symptoms, and its self-limited course.

Appendicitis is excluded by the absence of pain, tenderness, and muscular rigidity; intussusception by the fact that the symptoms are less severe, by the absence of blood and mucus from the stools, and by the fact that intussusception is usually seen in infancy.

Treatment.—When the premonitory symptoms appear, starvation and free purgation offer the best prospect of aborting an attack. If the vomiting has once begun, nothing seems to have the slightest influence in controlling it. It is usually increased by the taking of food or drink or by any medication by the mouth, and all should be withheld. The patient should be kept absolutely quiet and a saline solution given, per rectum, at regular intervals, usually six to eight ounces, four or five times a day. To this twenty or thirty grains of sodium bicarbonate may be added. This keeps up the urinary secretion, allays thirst and often restlessness, and when it is retained usually adds much to the patient's comfort. When the vomiting has ceased for several hours it is not likely to recur if food is very judiciously administered, at first in small quantities. Broth, thin cereals, kumyss, or small quantities of iced milk and lime-water in equal proportions may then be given. In severe or prolonged cases, six to eight ounces of a 5-per-cent glucose solution may be given by rectum once or twice a day. If this is not retained, three to six ounces may be given subcutaneously in a saline solution.

When acidosis develops it should receive its special treatment. (See Acidosis.) Between the attacks, the alkaline treatment is to be recommended; it consists in giving bicarbonate of soda in doses of fifteen to thirty grains three times daily and continuing it for many months.

When the prodromal signs of an attack appear, larger doses, as much as twenty grains every hour, should be administered. The diet should consist principally of meat, vegetables, skimmed milk, cereals in moderate amount, and stale bread. In addition to careful regulation of the diet the general nutrition should be considered, and the patient's life so regulated that extreme fatigue and exhaustion, as well as nervous excitement, are prevented. In most cases close attention to these matters has resulted in a very great diminution in the frequency of the attacks.

ACUTE GASTRITIS

In comparison with the frequency of inflammatory diseases of the intestine, those of the stomach are rare, particularly so in infancy. Owing largely to the character of its secretion and its contents, the stomach is much more resistant to infection than are the intestines. Gastritis seldom exists alone, but is usually associated with enteritis or colitis.

Etiology.—The causes of gastritis are, in the main, those of acute gastric indigestion—improper food or feeding—to which possibly is added infection. Gastritis may also be caused by the introduction of irritants, which may either be swallowed accidentally or given as drugs.

Lesions.—The mucous membrane of the stomach may be the seat of acute catarrhal, ulcerative, or membranous inflammation, all forms except the catarrhal being rare. There is also seen a mixed form, which from its cause is usually termed "corrosive" gastritis.

Catarrhal Gastritis.—This is characterized by hyperemia of the mucous membrane, exudation of cells into the mucosa, a great increase in the secretion of the mucous glands, and changes in the epithelium. About the only change which can be recognized by the naked eye is congestion and swelling of the mucous membrane. These are usually more marked toward the pyloric end and along the greater curvature. There may be small extravasations of blood into the mucosa. The stomach contains undigested food and mucus, which may be thick and tenacious, adhering very closely to the mucous membrane. The mucus may be stained brown from the capillary hemorrhages. The stomach may be either distended or contracted. Under the microscope the changes are seen to be almost entirely in the mucosa. In some places there is loss of the superficial epithelium, in others only degenerative changes in it are seen. The mucosa is infiltrated with round cells, this process being rarely diffuse, but generally occurring in patches. The blood-vessels are distended and many small extravasations are seen. Sometimes there is a moderate infiltration of the submucosa. Acute catarrhal gastritis alone is rarely severe enough to cause death. It is usually seen in cases

which prove fatal from other causes, particularly diseases of the intestine.

Gastric softening (*gastromalacia*) is a condition dependent upon post-mortem changes—probably self-digestion of the stomach. It is found both where gastric symptoms were present and where they were absent. It is situated nearly always in the posterior wall, and usually covers a considerable area, about one-third or one-fourth of this wall. It is recognized by the gelatinous, translucent appearance of the walls of the stomach, which are so softened that the finger may be pushed through them without force, or that sometimes the stomach ruptures while it is being removed. This condition is rarely seen when the stomach is empty. It can scarcely be mistaken for a pathological condition if its occurrence is borne in mind.

Ulcerative Gastritis.—This was met with six times, not including tuberculous cases, in 390 consecutive autopsies upon infants in the Babies' Hospital. Three of the patients were less than four months old, and all were females. The ulcers varied from one twenty-fifth to one quarter of an inch in diameter, and usually from ten to fifty were present. They seldom extended to the muscular, and never to the peritoneal coat. The lesion was most marked in the posterior wall, toward the pyloric end and along the greater curvature. Evidences of catarrhal inflammation were present in most of the cases, and in four, of membranous inflammation. Lesions in some other part of the digestive tract were present in all but one case, in two there was thrush in the esophagus; in three there was ulceration somewhere in the intestines.

Membranous Gastritis.—This is even more rare than the varieties previously mentioned. We have met with it but four times in infants. One case was associated with a membranous colitis; a second case with a streptococcus inflammation of the fauces and larynx in an infant but six weeks old. The esophagus was not involved in this case; and indeed it often escapes. No Klebs-Loeffler bacilli could be found either in coverslip preparations or by culture.

To the naked eye the membrane appears of a grayish-green color; it is adherent, but can be detached in quite large patches. Only a portion of the stomach is usually affected. The microscopical appearances resemble those of membranous colitis. There is a pseudo-membrane composed of fibrin, granular matter, epithelial cells, and bacteria. The mucosa shows a moderately dense infiltration with round cells, and in places superficial ulceration. There is also infiltration of the submucosa, and in some places even the muscular coat is involved.

Membranous gastritis occurring in patients dying of diphtheria is not common. Councilman, Mallory, and Pearce noted its presence in only five of one hundred and twenty-seven autopsies.

Corrosive Gastritis (*toxic gastritis*).—This form of inflammation is

excited by various irritating and caustic substances, taken by accident. The most frequent are carbolic acid and caustic alkalis.

The lesions in the stomach depend upon the amount of the substance swallowed, the degree of concentration, and whether the stomach was full or empty at the time. Strong caustics, whether acids or alkalis, usually act more deeply and extensively in the pharynx and esophagus, for, owing to the spasmodic contraction of the muscles of these parts, often but a small amount of the substance reaches the stomach. Concentrated irritant poisons produce in the stomach, especially along the greater curvature, irregular ulcers, which may be so deep as to cause perforation, or they may affect the mucous membrane only. In severe cases death takes place early, often in a few hours. Dark, ragged ulcers are found in the stomach, the surrounding mucous membrane is the seat of intense congestion, and in places there are extravasations of blood. If death is delayed there are evidences of intense inflammation, sometimes with the production of a pseudo-membrane. If the amount of poison is not sufficient to cause death, and if the patient recovers from the resulting gastritis, a cicatricial condition of the stomach results, which later may lead to stenosis of the pylorus or other deformity of the organ.

Symptoms.—*Catarrhal gastritis* can not be distinguished at its beginning from an attack of acute indigestion. There is fever, pain, vomiting, thirst, loss of appetite, coated tongue, and prostration. The presence of inflammatory changes is indicated by the continuance of these symptoms, particularly the pain, vomiting, fever, and thirst. With the pain there may be epigastric tenderness. All food and liquids are immediately rejected, and even when nothing is taken the retching and vomiting may continue, nothing but frothy mucus or serum being brought up, sometimes streaked with blood. The vomited matters are usually very sour; they may be bilious. The temperature is rarely high except at the outset. After the first or second day it usually ranges between 100° and 101.5° F. Thirst is intense, and all liquids are taken with avidity, especially if cold, even though they are immediately vomited. The tongue is thickly coated with a white fur, and the breath may be foul. The constitutional symptoms are generally most severe at the outset. The usual duration of such attacks is from four to seven days, but with improper management, especially injudicious feeding, the disease may be much prolonged. One attack may follow another until a chronic condition is established. In most of the cases there is some disturbance of the intestines, usually a sharp attack of diarrhea. Sometimes the gastric symptoms subside after a few days and those of the intestines become the predominant ones. The symptoms above given are those in infancy. In older children there is less fever, prostration.

and diarrhea, but pain and vomiting are prominent. The attacks are usually shorter and altogether less severe.

The rare cases of *ulcerative gastritis* have nothing by which they can be distinguished from the form described, except a more prolonged course and a greater liability to hemorrhage.

Membranous gastritis also presents no peculiar symptoms. In fact, in the cases we have personally seen, the gastric symptoms were insignificant, and the condition not suspected during life.

In *corrosive gastritis* the effects of the caustic may be seen in the mouth and pharynx, the mucous membrane being usually of a gray or whitish color. Pain and a sense of constriction are felt in the esophagus and stomach, and thirst is great. Vomiting follows almost immediately, and the matters vomited are usually bloody. The subsequent course in most of the cases is the rapid development of collapse, and death in a few hours from shock. The younger the child the sooner does the case terminate. In irritant poisoning not severe enough to produce death, the symptoms of acute gastritis follow, usually accompanied by more or less enteritis owing to the passage of the irritant into the intestine. There is seen a continuance of the vomiting, pain and epigastric distention, and diarrhea, and from these symptoms death may result in two or three days. It is extremely rare in early childhood for the patient to survive both the stage of shock and that of acute inflammation, so that the deformities of the stomach and the chronic conditions mentioned are practically never met with except in older children.

Treatment.—Cases of acute catarrhal gastritis are to be managed very much like those of acute gastric indigestion. Thirst may be relieved by swallowing bits of ice. Where there is continuous vomiting of acid mucus, relief is sometimes afforded by repeating the stomach-washing once in twelve hours with a one-per-cent solution of bicarbonate of soda, at 110° F. In older children, beneficial results sometimes follow the use of bismuth subcarbonate (gr. x every two hours); but in infants we have seen but little effect from any form of medication, the reliance being upon rest, careful feeding, and stomach-washing.

Cases of corrosive gastritis require special treatment. The first indication is to administer the proper chemical antidote to the substance swallowed, and the next to use bland mucilaginous or oily fluids, such as milk, albumin water, oils in large quantities, etc. Especially should stomach-washing be avoided except immediately after the ingestion of the poison. Opium is always required, on account of pain, and should be given hypodermically. The general symptoms are to be treated according to the indications of the individual case.

CHRONIC GASTRIC INDIGESTION—CHRONIC GASTRITIS—GASTRIC CATARRH

Although from a pathological point of view these conditions may not be identical, from a clinical standpoint there is no advantage in attempting to separate them. Nothing distinguishes chronic indigestion from chronic gastritis except that in the latter, in addition to continued derangement of function, there is a greater increase in the production of gastric mucus. Chronic indigestion does not long exist without the production of a certain amount of catarrhal inflammation. This condition in the stomach seldom, if ever, exists without more or less involvement of the intestine, and in the majority of cases the intestinal condition is the more important. In some, however, the gastric symptoms predominate, and it is only those which are here considered. What is often called chronic gastric indigestion in infancy has already been discussed in the chapter devoted to Difficult Feeding. In this connection only the condition as it affects older children will be referred to.

Etiology.—Chronic gastric indigestion may follow acute attacks, or it may be chronic from the outset. Etiological factors of importance are overfeeding, too large meals, unsuitable food, especially solid food too early and in too large amounts for very young children. The condition generally accompanies dilatation of the stomach. As a consequence of imperfect digestion, fermentation in the residuum takes place, and the irritating products of this fermentation soon cause a catarrhal inflammation with a production of mucus. Chronic gastric indigestion also complicates many of the constitutional diseases of childhood, especially rickets, syphilis, tuberculosis, and malnutrition. It may follow any of the acute infectious diseases. In older children it is often due to the habit of rapid eating and insufficient mastication, the cause of which is very often carious teeth. It may complicate valvular disease of the heart.

Lesions.—The changes found in chronic gastritis are usually confined to the mucosa. In the mild form there are degenerative changes of the epithelium of the tubules, with an increased production of mucus; there may be a slight infiltration of the mucosa with round cells. The more severe form, with marked cell infiltration and the production of new connective tissue, is extremely rare. The submucous coat may be thickened and the muscular coat attenuated. The lesion can not be recognized by the naked eye. The stomach is apt to be more or less dilated, and its surface is coated with thick and very adherent mucus. This lesion rarely exists alone, practically never in infancy, but is associated with similar lesions in the intestines, the latter often being more severe.

Symptoms.—In all cases the most constant symptom is vomiting, which may occur regularly after meals, or only in the morning before breakfast. If the latter, the vomited matters consist chiefly of mucus. In addition to these regular attacks there may be the frequent regurgitation of small quantities of food. There are present gastric flatulence and pain, due to hyperacidity or to acid fermentation. The appetite is variable—sometimes inordinate, sometimes entirely lost; it may be capricious, there being usually a craving for highly seasoned food. The tongue is constantly furred, and the breath usually disagreeable. These symptoms are seen in all degrees of severity. Intestinal disturbances are not frequent. Constipation is more common than diarrhea. The general symptoms are those of malnutrition. There are anemia, wasting, constant fretfulness, disturbed sleep, and various other nervous disorders.

Prognosis.—The prognosis depends upon the age of the patient, the duration of the disease, the surroundings, and upon how well treatment can be carried out. There is little tendency to spontaneous recovery, but under favorable conditions, with constant care, much may be done for all these patients and many of them may be completely cured.

Treatment.—The general treatment is too apt to be ignored, but it is just as important as measures directed more specifically to the stomach. A large, roomy nursery, and plenty of fresh air by night and by day, are very important; equally necessary are quiet surroundings and freedom from disturbing conditions which sometimes obtain in the nursery. General friction of the body is useful in delicate children with poor circulation. Of the measures directed to the stomach, two are chiefly to be depended upon—stomach-washing and proper feeding.

Stomach-washing is useful, first, in removing the mucus which is abundant in most of these cases; secondly, in cleansing the organ thoroughly at least once a day, this of itself being most important; thirdly, as a stimulant to the gastric secretions, especially hydrochloric acid. Plain boiled water, or a weak alkaline solution—sodium bicarbonate, one dram to the pint—may be employed. In the early part of the treatment the washing should be done daily; later, every second or third day. The time selected is not very important, but it is better to make this about three hours after feeding.

With some children stomach-washing can not be easily employed, and other means must be used to clear the stomach of mucus. The best is undoubtedly the use of large draughts of water, as hot as can be borne, an hour before eating. From six to eight ounces should be taken, preferably slowly by sipping. To this may be advantageously added, in many cases, fifteen or twenty grains of bicarbonate of soda.

The diet should consist of diluted skimmed milk, whey, buttermilk, kumyss or zoolak, beef juice, rare meat, and a moderate amount of

starchy food, preferably dried bread or zwieback. All fruits should be avoided. All pastry, sweets, nuts, and candies should be absolutely prohibited. With improvement in the symptoms green vegetables may be added to the diet, and the amount of starchy food increased. The amount of water taken at meal-time should be carefully restricted. Beneficial results are often obtained in these cases by the use of nux vomica or simple bitters before meals, and the regular administration of hydrochloric acid (gtt. v to viij of the dilute acid) shortly after meals. The general treatment must not be neglected. The patient should lead an outdoor life as much as possible, and should take regular but very moderate exercise. Great caution is necessary against overfatigue. Iron may be given in most cases during convalescence; but cod-liver oil should be carefully avoided until the gastric symptoms have quite disappeared. Relapses are easily excited, and the most constant care regarding the food must be maintained for months, or even years.

DILATATION OF THE STOMACH

Moderate dilatation of the stomach is quite a frequent condition, although it is not so large a factor in the disorders of digestion in infancy and childhood as many who have written upon the subject would lead us to believe. A very marked degree of dilatation is rare, but in these cases its recognition is important and its treatment difficult.

Dilatation is almost invariably regular or cylindrical; it is usually most marked at the cardiac extremity. Cases of irregular or saccular dilatation, except when associated with cicatricial conditions, are of very rare occurrence.

Dilatation may also result from congenital stenosis of the pylorus. The most important predisposing cause, however, is the muscular atony which accompanies rickets. It is found to a slight degree in almost all severe cases of rickets. The principal exciting causes are continued distention from overfeeding and chronic indigestion.

In most cases the only symptoms are those of the chronic indigestion which almost invariably accompanies dilatation. The vomiting seen with dilatation is peculiar in that it is infrequent, possibly only once a day, but then the quantity vomited is larger than the last meal taken. In young infants the pressure symptoms resulting from acute dilatation may be very serious. This is particularly true of those with acute bronchitis or bronchopneumonia, or atelectasis. In such patients we have seen very grave symptoms accompany the rapid distention of a dilated stomach, and in one very delicate infant of three months this was apparently the cause of death. A positive diagnosis of dilatation is only

made by the physical signs. There is epigastric fulness and distention, and in some thin patients the outline of the stomach can be distinctly seen. Dilatation of the transverse colon, however, may be mistaken for dilatation of the stomach. In the latter, the lower outline is convex, while in the former it is usually slightly concave. The most satisfactory means of diagnosis is by percussion. The examination should be made three or four hours after feeding, at which time the whole abdomen is apt to be tympanitic. The stomach should then be filled with water; the lower limit of the area of flatness will be the lower border of the stomach. This is much more satisfactory than determining the outline after the generation of gas in the stomach. If the lower border comes below the umbilicus, it may be assumed that the stomach is dilated.

In moderate dilatation of the stomach the prognosis is good unless due to pyloric stenosis. If the infant has any acute or chronic pulmonary disease, dilatation of the stomach may add to the discomfort and even to the danger from that condition. The distention of a dilated stomach occurring in the course of any acute pulmonary disease should be relieved by the use of the stomach tube.

In the management of these cases the first point is to restrict the use of fluids, reduce the size of the meals, and regulate the diet in accordance with the general plan outlined in the chapter on Chronic Indigestion. If the dilatation is marked, the stomach should be washed once a day. The general condition of the patient usually requires tonics. Rickets, if present, should receive its appropriate constitutional treatment.

ULCER OF THE STOMACH

Ulceration of the stomach may be found in connection with several pathological processes which are quite distinct from one another:

1. *Ulcers in the Newly Born.*—These have already been referred to in the chapters on Hemorrhages of the Newly Born. The only characteristic symptom is hemorrhage.

2. *Ulcers Resulting from Acute Gastritis.*—These also are not frequent. As a rule they give no symptoms except those of gastritis, although in several cases we have known severe hemorrhage to result from them. This symptom will be considered later.

3. *Tuberculous Ulcers.*—These are quite rare. We met with gastric ulcers five times in one hundred and nineteen autopsies on tuberculous cases; however, the evidence was not conclusive in all of them that the ulcers were tuberculous; but in three the tubercle bacilli were found. Usually there were several small ulcers; in one case but two

were present, the larger one being nearly three-fourths of an inch in diameter, and situated on the posterior wall near the middle of the greater curvature. All but one of these cases were in infants, one child being only ten months old. The ulcers gave no symptoms during life, and death took place from general tuberculosis. This is the history of nearly all the few cases on record. In one, however, reported by Casin, a tuberculous ulcer perforated the stomach and caused death from peritonitis.

4. *Simple Perforating Ulcers.*—In young children these are of great rarity and uncertain pathology. Rotch has reported a case in an infant but seven weeks old, and Cade one in an infant of two months.

The symptoms of ulcer before perforation are gastric pain and tenderness, vomiting of blood, and often bloody stools. In most of these cases in children there were no symptoms until perforation; then followed collapse, sometimes high temperature, the rapid development of tympanites, and death from shock or from peritonitis.

The prognosis is bad in all forms of ulcer of the stomach, except the small follicular variety. In this, however, the diagnosis can not positively be made except by gastric hemorrhage, and it is only this which makes these cases serious.

Treatment.—The treatment is absolute rest, ice by mouth, small doses of opium, and rectal feeding; later, bismuth, arsenic, or nitrate of silver. If symptoms of perforation occur the abdomen should be opened without delay, as offering the only chance of recovery.

DUODENAL ULCER

Until recently these ulcers have been considered very rare in infancy and early childhood, but the increasing number of cases reported, especially since 1908, indicates that it has formerly been overlooked. From a study of ninety-five cases in infants under one year collected from the literature by one of us in 1913, the conclusions which follow have been drawn.

Duodenal ulcers are much more common than gastric ulcers; according to Entz they outnumber them ten to one. Not a single instance of peptic ulcer of the stomach in infancy has been observed at the Babies' Hospital in twenty-seven years. In but one case were duodenal and gastric ulcers found in the same patient, even including cases occurring in the newly born. Seventy per cent of the reported cases have been observed between the ages of six weeks and five months; about ten per cent occur in the newly born. The great majority of the cases have been seen in infants of the marasmus (atrophic) type. In most of them there

has been also a history of previous digestive disorders. In several cases duodenal ulcers have been associated with spasm of the pylorus.

The most frequent site of the ulcer is on the posterior wall of the duodenum and practically all are above the papilla. When but a single ulcer is present, as is true of about two-thirds of the cases, it is nearly always situated just below the pyloric ring. These ulcers are circular in shape; they have shelving, sharply defined edges, usually described as "punched-out" in appearance. At the base, blood vessels of considerable size are often seen. They may involve the mucous membrane only, in which case they may readily be overlooked, or they may go to the muscular coat, to the peritoneum or they may even perforate. Microscopical examination shows almost complete absence of round-cell infiltration and other evidence of inflammatory reaction. The rest of the duodenum usually shows a normal mucous membrane or one simply blood stained. Large clots or fresh blood may be found in the duodenum or in any part of the small or large intestine. The stomach also may contain old or fresh blood.

The generally accepted view of the pathogenesis of duodenal ulcers is that they are due to thrombosis followed by self-digestion of the mucous membrane over a circumscribed area. The situation of the ulcers, above the papilla, indicates that the lesion is due to the action of the gastric juice. Below the opening of the common duct the bile and pancreatic juice apparently protect the mucous membrane.

Symptoms.—In over one-third of the recorded cases no symptoms suggestive of the condition were present, the ulcer being found at autopsy in patients dying from other causes. In other cases death occurs suddenly in collapse, sometimes preceded by symptoms of an ordinary gastro-intestinal disturbance and sometimes by none at all. In such cases the autopsy frequently discloses severe concealed hemorrhage or perforation. If life is prolonged peritonitis may follow, but its recognition under these circumstances is exceedingly difficult since vomiting, fever and distention may all be wanting. Localized pain or tenderness in patients of this age is of no assistance to the diagnosis, though valuable symptoms in older children. The only definite symptom pointing to duodenal ulcer is hemorrhage. Blood may be vomited or passed in the stools. In sixty-four cases of ulcer reported with good histories, bloody stools were observed in twenty-eight, bloody vomiting in ten and both in six cases, four of these being in the newly born. Clear blood may be seen or blood changed by the action of the stomach or intestine. Once it occurs, hemorrhage is apt to continue until the death of the patient, usually in twenty-four to thirty-six hours. The appearance of blood in any considerable amount in the stools of a young infant should always suggest duodenal ulcer. Jaundice was a symptom in but one case in the series.

The diagnosis is made mainly by the presence of hemorrhage from the

stomach or intestine, usually associated with collapse. Perforative peritonitis may be due to appendicitis as well as ulcer and both intestinal hemorrhage and collapse may occur with intussusception. These should be borne in mind as two conditions which may be confounded with duodenal ulcer. Polypus and colitis must also be excluded. The prognosis of duodenal ulcer at present is very bad. The finding of healed ulcers at autopsy proves that recovery does sometimes take place, but it must be considered rare.

The treatment is purely symptomatic; on account of the present uncertainty of diagnosis, surgical measures are rarely justifiable.

TUMORS OF THE STOMACH

Although exceedingly rare, tumors of the stomach occur in childhood, and are seen even in infancy. A case of *sarcoma* of the stomach in a child of three and a half years has been reported by Finlayson. It was apparently primary. The microscopical examination showed it to be of the spindle-celled variety. This writer could find no other recorded case under the age of fifteen.

Lymphadenoma of the stomach in a rachitic infant of eighteen months has been recorded by Rolleston and Lathan. There were multiple tumors arising from the mucous membrane in the pyloric region. The case in many features resembled leukemia.

Six cases of *cancer* of the stomach in children under ten years are collected in an article by Osler and McCrae. Four of these were in young infants and probably congenital. One case, in a child of eight, presented the usual symptoms and lesions of the adult disease.

HEMORRHAGE FROM THE STOMACH (*Hematemesis*)

The most frequent variety of hemorrhage from the stomach, that is seen in the newly born, has already been considered.

Serious and even fatal cases of gastric hemorrhage though extremely rare may be seen in older infants. The source of the bleeding may be small capillary hemorrhages from the mucous membrane, it may be from single or multiple ulcers of the stomach or more frequently from duodenal ulcers.

Hemorrhages from the stomach may occur in purpura, hemophilia, scurvy, and rarely in malaria. In young girls about puberty it may be a form of vicarious menstruation. Occasionally blood may be vomited in cases of hemorrhagic measles. Two cases are reported in which fatal hemorrhage followed the swallowing of a foreign body. In both, vomit-

ing of blood occurred long after the original accident. In one case two and a half years had elapsed. The autopsy in this case showed impaction of the foreign body and ulceration into the arch of the aorta. Spurious hemorrhages may occur when blood has been swallowed and then vomited. The source of this is most frequently the nose or pharynx. It may happen in infants at the breast, when the blood is drawn from a fissure or ulcer in the nipple. The amount of blood vomited under these circumstances may be large enough to be quite alarming. It may be recognized by the child's general condition being normal, and by the presence of fissures or ulcers upon the nipple. It may sometimes be noticed that the vomiting of blood follows nursing from one breast and not from the other.

Symptoms.—There may be no symptoms except those of internal hemorrhage, but this is rare. Usually there is vomiting of blood, and blood appears in the stools. If the hemorrhage is rapid and vomiting speedily occurs, the blood may be of a bright-red color. If it has been long in the stomach it is of a dark-brown or black color resembling coffee-grounds. The stools containing blood from the stomach are black and tarry in appearance. The general symptoms will depend upon the amount of blood lost.

In a case where blood is vomited, the first point is to distinguish spurious from true gastric hemorrhage. The nose and pharynx, especially its posterior wall, should be carefully examined. If the child is at the breast, the nipples should be examined. In older children it is important to distinguish vomiting of blood from hemoptysis. This distinction is to be made in accordance with the rules laid down in text-books on general medicine. The prognosis is bad if the hemorrhage is due to ulcer, if it is very profuse, or if it occurs in young infants. When it occurs in connection with constitutional diseases the prognosis depends upon the original disease.

Treatment.—Altogether the most efficient remedy is the suprarenal extract. It may be given very freely, at least two grains every half hour to a child of one year. The patient should be kept quiet, preferably upon the back; if there are signs of collapse, stimulants may be given hypodermically or by the rectum. No food or water should be given by the stomach for at least twenty-four hours after the hemorrhage has ceased.

THE SWALLOWING OF FOREIGN BODIES

Between the ages of one and four years the habit of swallowing foreign substances is a very common one. The variety of objects swallowed includes all those articles which the young child can reach and put into

his mouth. The most common are detached parts of toys, marbles, pebbles, buttons, and coins. Not only are such smooth articles swallowed, but also with equal readiness, sharp ones, such as pins of every variety, bits of glass, fragments of bone, nails, and small toy knives and forks, etc. At the time of swallowing, choking attacks, severe pharyngeal pain, and sometimes slight hemorrhage may occur. Symptoms referable to the esophagus are very few. Nor in the stomach are symptoms often excited. While passing through the intestine there may be colicky pains, but in the majority of instances there are no symptoms whatever even with sharp or angular bodies. Impaction and perforation, while possible, are surprisingly rare. The usual time required for a foreign body to traverse the intestinal tract is from four to ten days, but it may be considerably longer. If the body swallowed is a smooth one, it passes the sphincter ani without difficulty. But with sharp bodies there may be severe pain and sometimes hemorrhage.

The diagnosis is often a matter of much difficulty, and without an X-ray examination a positive diagnosis is impossible. Very often when the physician is called because this condition is suspected by parents the alarm turns out to be a false one.

It is most surprising to see the size, variety, and dangerous character of the foreign bodies which pass through the intestinal tract without causing any symptoms whatever. The expectant treatment is therefore by all means to be recommended. No emetics or cathartics should be administered. The diet should be abundant and composed of articles of food which leave much residue, e. g., coarse cereals, bread, and vegetables. Most of all, operation should not be performed or even considered unless there are definite local symptoms.

Quite distinct from such accidental swallowing of foreign substances as has just been described, is the practice of pulling off and swallowing fur from rugs, wool from toys or blankets, shreds from clothing, and a great variety of other substances. This habit is usually seen in nervous children, and often in those where some gastric irritation seems to excite an abnormal craving. In infants the quantity of the substance is generally small and usually it provokes vomiting or the material is speedily passed by the bowel. In the Babies' Hospital a colored child of about eighteen months passed in one day a large mass of hair which she had pulled from her own head. Another child in the same ward pulled into shreds and swallowed a large portion of the foot of a cotton stocking, and passed the same by the bowel the following day. Such occurrences are not very common.

It occasionally happens that the substance swallowed does not pass the bowel but forms an intestinal tumor which may give rise to obscure and sometimes to severe symptoms of long duration. But more often the

tumor forms in the stomach. These gastric tumors are usually composed of hair from the patient's own head. They are more frequently seen in older children than in infants, and usually in girls on account of the long hair. Many of these patients are of the nervous type. The habit may continue until a tumor of considerable size may form, sometimes attaining two or three pounds in weight.

The symptoms of *hair ball in the stomach* are vague until the tumor is discovered. There are usually gastric disturbances of a rather indefinite character. Epigastric pain is common, but vomiting is not especially marked. The general health may suffer but little for a long time. The tumor may be mistaken for cancer, a displaced spleen or kidney, fecal impaction, or a tumor of the omentum. A correct diagnosis is seldom made until operation is done. In a few instances the tumor has disappeared after catharsis. If operation is done the outcome is almost always favorable.

CHAPTER VI

DISEASES OF THE INTESTINES

MALFORMATIONS AND MALPOSITIONS

Malformations are not very frequent, but are of great variety. With the exception of those situated at the lower end of the intestine they are not of much practical importance, for the condition is such ordinarily as to be incompatible with life. Malformations may be met with at any point in the canal, but most frequently in the rectum and anus. Aside from these, malformations of the large intestine are much less common than those of the small intestine.

Malformations of the Rectum.—In Fig. 32 are shown the usual varieties of malformation of the rectum. The most frequent is atresia of the anus (1). In this the cutaneous septum has not been absorbed, but the intestine is normal to its lower extremity. This form is readily curable by a surgical operation. In the next variety (2) the cutaneous orifice and the lower part of the rectum are normal, but a membrane separates this portion from the upper part of the gut; this is usually situated within two or three inches of the anus. The bulging of the lower part of the distended intestine can usually be felt by the finger in the rectum, and a simple division of the membrane by a guarded bistoury may relieve the condition. The third form (3) is more serious. Here the rectum terminates in a blind pouch at a variable distance from the anus, and is represented below by an impervious fibrous cord. The

diagnosis of this condition can not positively be made without opening the abdominal cavity. The bulging of the intestine appreciable by the finger in the rectum, is the only point which differentiates the preceding variety from this one. Instead of atresia of the rectum there may be stenosis of varying degrees, which may give rise to the usual symptoms of stricture. This is often curable by dilatation.

Malformations of the Small Intestine.—There may be stenosis or atresia at any point, often at many points. Obstruction is much more frequent in the upper than in the lower part of the small intestine, the most common seat being the duodenum. Atresia is more often seen than stenosis. There may be a single point of obstruction, or the lumen of the intestine may be obliterated for a considerable distance, the intestine being represented only by a fibrous cord which connects the two open portions, or there may be no connection between them. In all cases the intestine above is found very greatly distended, while that below is empty and usually atrophied. The causes of these multiple deformities are mainly two—fetal peritonitis and volvulus. In fetal peritonitis there are usually found bands of adhesions between the intestinal coils, and between the intestine and the solid viscera. Syphilis has been assigned as a cause in many cases. Volvulus, or a twisting of the intestine during its development, is a more satisfactory explanation of the majority of the cases, especially when there are multiple points of atresia. All these conditions are beyond the reach of surgical treatment. The symptoms appear soon after birth and are those of intestinal obstruction. The higher the point of obstruction the shorter the duration of life; it is rarely more than a week in any case of atresia; in stenosis it may be two or three months.

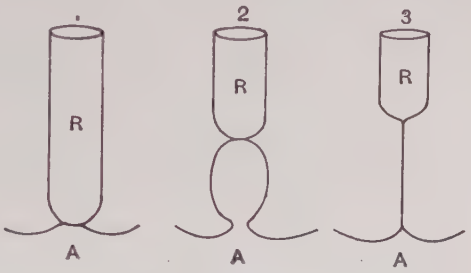


FIG. 32.—MALFORMATIONS OF THE RECTUM. A, anus; R, rectum.

Meckel's Diverticulum.—This is the remains of the omphalomesenteric duct, which in fetal life forms a communication between the intestine and the umbilical vesicle. It is given off from the ileum, usually about a foot above the ileo-cecal valve. Most frequently it exists as a blind pouch from one-half to two or three inches long, communicating with the intestine. At the extremity of this there may be a fibrous cord, which is free in the abdominal cavity or attached to the umbilicus. In other cases the duct may remain pervious quite to the umbilicus, so that there is a fecal fistula. Prolapse of the mucous membrane of the duct

may lead to an umbilical tumor, described elsewhere. Meckel's diverticulum, especially when present as a cord connecting the ileum with the umbilicus, may compress a coil of intestine, leading to obstruction or even strangulation. This may occur in infancy or later in life.

Malpositions.—The ascending colon may be found upon the left side. There may be a complete transposition of the abdominal viscera. In cases of congenital umbilical hernia a large part of the intestines may be found in the tumor, and in diaphragmatic hernia they may be in the thoracic cavity.

DIARRHEA

The term *diarrhea* is used to include all conditions attended by frequent loose evacuations of the bowels. These depend upon an increase in peristalsis and in the intestinal secretions.

The importance of diarrheal diseases in children can best be appreciated by reference to the following table, showing the mortality of diarrheal disease in children under two years, as compared with that from certain infectious diseases for all ages.

Deaths in New York City for Five Years

Measles, all ages	3,378
Scarlet fever, all ages	4,152
Pertussis, all ages	2,000
Typhoid, all ages	3,523
Diphtheria, all ages	10,277
Total deaths from five diseases	23,330
Diarrheal disease under two years	26,563

There are several important underlying factors upon which diarrheal diseases depend. Their greatest frequency belongs to the first year of life; and after the second year a notable diminution both in frequency and severity is seen, and a fatal outcome is relatively rare. The extreme susceptibility in infancy is due to several causes. The digestive organs are severely taxed to provide for the needs of the growing body. The mucous membrane of the gastro-enteric tract of all infants is very delicate in structure, and even in those with good health is exceedingly vulnerable. This vulnerability is enormously increased in the very young, and in those who are feeble, delicate, or suffering from any form of digestive disorder. The mucous membrane of the digestive tract is furthermore constantly exposed to injury, either mechanical or chemical, and to infection.

Everything which lowers the general vitality increases the liability to diarrheal diseases. Chronic disorders of digestion, marasmus, malnutrition, and rickets are especially important factors.

The most striking fact about diarrheal diseases is their prevalence during the summer season. This is graphically shown in Fig. 33, where are given by months the mortality records from this cause for New York City for ten years.

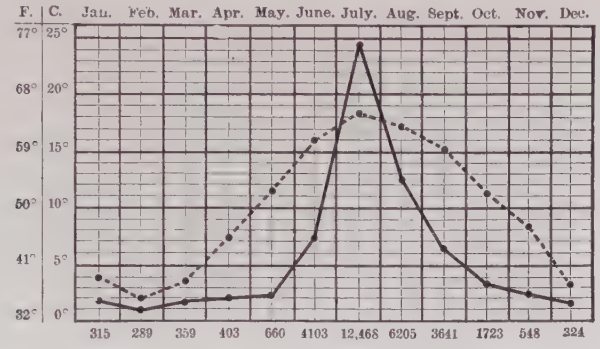


Fig. 33.—Mortality from Diarrheal Diseases in New York for Ten Years in Children Under Five; Compared with the Mean Temperature for the Same Period. ———, mortality; - - - - - -, mean temperature. (Seibert.)

While diarrheal diseases are met with in all seasons they regularly increase with the advent of hot weather. In this country the higher summer temperature of the inland cities, Philadelphia and Chicago, is associated with a higher mortality from diarrheal diseases than is seen in Boston and New York with a lower range of temperature. Thus in Philadelphia and Chicago 32 per cent of the deaths under one year have been due to diarrheal diseases; while in New York but 27 per cent, in Boston but 19 per cent, and in London but 13 per cent have been from this cause. The large cities of northern Europe—London, Paris and Berlin—witness nothing like the mortality from diarrheal diseases seen in the large cities of the United States.

How atmospheric heat acts in causing diarrheal diseases is not yet entirely settled. It was long the prevailing opinion that it was the effect of heat upon the infant's food, especially the bacterial contamination of cow's milk, that was the chief cause of diarrhea in summer. Without doubt thoroughness of milk inspection and the general use of sterilized milk in summer have materially reduced the mortality from this cause. But notwithstanding all the attention given to food there remains an enormous summer mortality from diarrhea. From the most recent study of this question the conclusion seems irresistible that heat itself has a direct, injurious effect upon the infant, and that it is not so much the outdoor temperature which counts as the stagnant heat of apartments in which the infant lives night and day. The effects of heat are intensified by want of ventilation and all unhygienic surroundings. Heat under

these conditions acts as a powerful depressant of the vital forces, disturbing metabolism, causing indigestion and diarrhea.

Diarrheal diseases are especially seen in cities, for there are combined the conditions of poverty, neglect, bad food and bad hygiene, all of which are important causes. That overcrowding and bad housing in our large cities are not the chief factors is shown by the fact that the death rate from diarrheal diseases is often higher in smaller places, especially factory towns, than large cities. Thus in New York State it has been higher in Troy, Cohoes and Newburgh than in New York City; and in Massachusetts, higher in Fall River and Lowell than in Boston.

Artificial feeding is an etiological factor of the first importance. Less than 5 per cent of the severe cases of diarrhea are among the breast-fed, and fatal cases among the exclusively breast-fed are really rare, no matter how bad the surroundings or how ignorant the mothers. Breast-feeding requires but little experience, and may be very successfully done even by those with a very low grade of intelligence and among the poor; but artificial feeding is not successful unless done with much intelligence and experience and also with good milk.

It is in factory towns, where the mothers work away from their homes and as a consequence breast-feeding is either not practiced at all or only for a short time, and where artificial feeding is usually badly done, that we see the highest mortality from diarrheal diseases. These conditions do not depend upon the size of the town and compared with them housing is of secondary importance.

Next to the kind of feeding as a cause of diarrhea must be placed gross or involuntary neglect or want of proper care. Ignorance and stupidity are large elements in the failure of artificial feeding among the poor. The simplest rules of hygiene are either unknown or ignored. The importance of cleanliness, fresh air, regularity and quiet is not appreciated. Under such conditions an infant, though often strong and healthy at birth, soon falls into a condition of malnutrition or marasmus with such feeble resistance that he readily succumbs to the depressing influences of the first hot weather, the intestinal tract being the most vulnerable point.

But all the other factors mentioned—artificial feeding, overcrowding, bad hygienic surroundings and neglect—exist the year round, yet diarrheal diseases are prevalent only in summer. We must therefore consider the direct or indirect effects of atmospheric heat as the primary exciting cause of paramount importance, the other conditions acting as secondary or predisposing causes.

The rôle of impure milk is so important as to demand further discussion; that it can cause diarrhea in infants is a fact that is established beyond question. We have seen every one of twenty-three healthy chil-

dren, all over two years old, occupying one dormitory cottage, attacked in a single day with diarrhea, which was traced to this cause.

When the enormous bacterial contamination of milk began to be appreciated, it was thought that in this was to be found the real cause of the prevalence and fatality of diarrheal diseases in summer. This belief carried with it the expectation that by furnishing to every artificially-fed infant a clean, fresh milk, or milk which had been pasteurized or sterilized, this great cause of infant mortality could largely be removed. It is true that a great reduction in infant mortality from

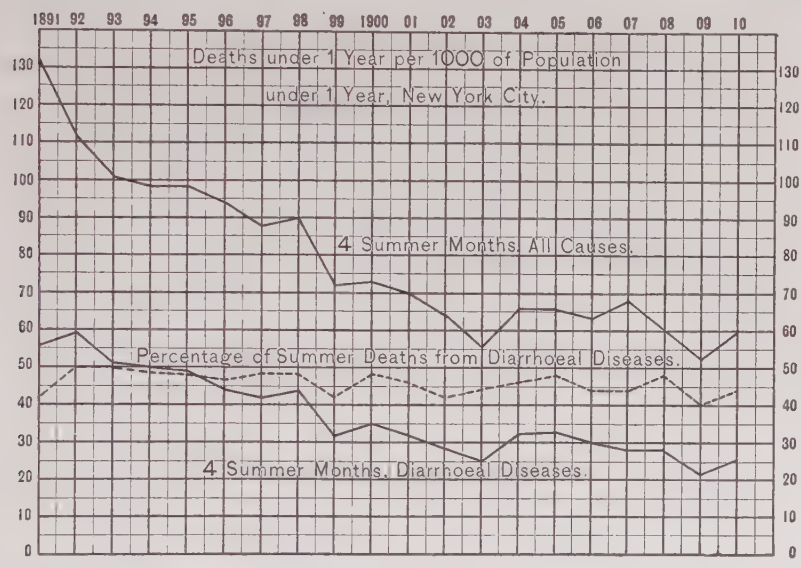


FIG. 34.—DEATHS UNDER ONE YEAR PER 1,000 OF POPULATION UNDER ONE YEAR, NEW YORK CITY. A comparison of summer deaths from all causes with summer deaths from diarrheal diseases for a period of twenty years.

summer diarrheal diseases has been effected during the last two decades; but it is also true that there has been quite as great a reduction in infant mortality in other seasons, and, in summer, from other causes than diarrheal diseases. (Fig. 34.) This leads us to question whether the bacterial contamination of milk is the great cause of diarrheal diseases, and whether the lowered mortality in summer has not been brought about quite as much by other conditions, such as better hygiene and care and a better understanding of infant-feeding, as by the exclusion of germs from milk or their destruction by heat.

In the years 1901 to 1903 an investigation [1] was undertaken by The

[1] The full report was published by Park and Holt in the *Medical News*, December 5, 1903.

Rockefeller Institute and the Health Department of New York to secure data regarding the following points: (1) The results in infant-feeding obtained with milk of different degrees of purity both in winter and in summer, as shown by the gain or loss in weight, the amount of gastro-intestinal disturbance, and the death rate; (2) the relation, if any, existing between the number of bacteria present in the milk and the frequency of diarrheal disease; (3) whether any organisms with pathogenic properties could be found in milk to which diarrheal disease could be ascribed as a cause; (4) whether the practice of heating milk—pasteurization or sterilization—affected the results obtained with any given milk; (5) to what degree older children as well as infants were affected by bacterial contamination of milk.

Observations were made upon 592 bottle-fed infants living in tenements of New York; 202 were observed in winter and 390 in summer. The infants were well when the observations were begun, and were watched for a period of about three months, being visited regularly by physicians. Samples of milk as fed were frequently examined as to the number and character of the bacteria present. Observations were possible upon infants taking (1) condensed milk, (2) the cheapest grade of store milk, (3) a better grade of milk delivered in bottles, (4) the best bottled milk sold in the city, all of the above being prepared at home, (5) milk modified at milk stations and dispensed in separate feeding-bottles.

During the winter, the mortality was but 2.5 per cent, and in but one case was death due to disease of the digestive tract. The health of the infants observed was not appreciably affected by the kind of milk nor by the number of bacteria which it contained. The different grades of milk varied much less in bacterial contamination in winter than in summer, the cheap store milk averaging only about 750,000 per c.c.

During the summer, the mortality was 10.5 per cent, four-fifths of the deaths being due to diarrheal disease. The worst results were seen in those whose food was either the cheap grade of store milk or condensed milk, and in those who received the poorest care.

The number of bacteria which milk may contain before it becomes noticeably harmful to the average infant in summer is not at all uniform. Of the usual varieties present, no strikingly deleterious results were seen until the number approached the one million mark. But much above this point injurious effects were usually manifest. Below it other factors seemed of greater importance in producing diarrhea. Thus in condensed milk the bacterial contamination was relatively small, yet the results were almost as bad as with the most highly contaminated milk.

No relationship could be discovered between any special forms of bacteria present and the health of children or the occurrence of diarrhea.

To test the effect of heating milk, observations were made during two summers upon 92 infants taking milk prepared at a milk station. It was from a good farm, and had been kept properly cooled. The infants were divided into two groups as nearly alike as possible. To one group the milk was given pasteurized (165° F. for thirty minutes), to the other group the same milk was given raw. All the infants were well at the beginning of the period of observation. The results are shown in the following table:

Food.	Total Number of Infants.	Remained Well Entire Summer.	Had Severe Diarrhea.	Average Days Diarrhea.	Deaths
Pasteurized milk containing 1,000 to 50,000 bacteria per c.c. at the time of use	41	31	10	4	1
Raw milk containing 1,200,000 to 20,000,000 bacteria per c.c. at the time of use	51	17	34	11½	2

Thirteen of the fifty-one infants on raw milk were changed before the end of the season to pasteurized milk because of serious diarrhea; but for this the results with raw milk would have been even more unfavorable. A similar experiment was made a third season with almost identical results.

Although the number of cases is not large, the results, which were practically uniform for three successive seasons, show unmistakably that in hot weather raw milk, although from a good source, but at the time of feeding highly contaminated with bacteria, causes illness in a much larger number of cases than when it has been previously heated.

After the first two years, children are much less affected by bacteria in milk. The observations seemed to show that milk from healthy cows, produced under cleanly conditions and kept at a temperature below 60° F., although containing large numbers of bacteria, sometimes amounting to many millions per c.c., might be taken in considerable quantities and for long periods by children over three years old, without any appreciably harmful effects. A single example is typical of a number of observations made. An orphan asylum, containing 650 children from three to fourteen years old, used during an entire summer milk in which the bacteria ranged from 2,000,000 to 20,000,000 per c.c.; yet during this period there occurred no case of diarrhea of sufficient severity to call a physician. The milk was kept cold, but was given without sterilization. Mere numbers of bacteria certainly appear to count for much less than was once supposed. But the fact should not be overlooked that milk abounding in bacteria because of careless handling is also always

liable to contain pathogenic organisms derived from human or animal sources.

These observations, continued for three seasons and giving each summer nearly identical results, indicate that we are to seek elsewhere than in a moderate bacterial contamination of milk the great cause of summer diarrheas. Though it is clear that excessive bacterial contamination is highly detrimental to infants, we must certainly look to the other factors for the explanation of a very large, possibly the largest, proportion of the cases. Of the other exciting causes, atmospheric heat, especially the stagnant heat of houses, is clearly first in importance. This may act by so interfering with normal digestion and metabolism as to lead to the formation within the body of injurious substances which excite diarrhea; or it may favor the excessive growth of bacteria ordinarily present in the digestive tract. In this group of cases the rôle of the bacteria seems to be secondary, though perhaps a very important one. According to this hypothesis, the exciting cause of the diarrheas under consideration is not something introduced from without, but something produced within the body itself.

From the foregoing discussion the measures to be employed in the prevention of diarrheal diseases are inferred. In the order of importance they are as follows:

1. Encouragement of maternal nursing and the adoption of measures to make this possible, particularly during the summer months.

2. Education of mothers in all matters relating to the care and hygiene of infants, best through the agency of the milk station and the visits of a trained district nurse.

3. Adequate supervision of the milk supply, the general use of pasteurized or sterilized milk during the summer, and furnishing good milk to those too poor to pay for it.

4. Instruction of mothers in regard to the care of milk in the home and in all matters of artificial feeding.

5. The constant supervision of artificially-fed infants either in the milk station or by visits to the home.

The adoption of these measures and their application on an extended scale by an efficient organization has resulted in a very great reduction in the deaths from diarrheal diseases wherever they have been tried. Nowhere have such results been achieved as in New York City, where the summer mortality in infants under one year has fallen in the Boroughs of Manhattan and Bronx from an average of 1069 for the three years 1908-10 to an average of 802 for the three years 1912-14. (See Fig. 5, Chapter on Infant Mortality.) These figures represent the actual number of deaths and take no account of the increase in population.

Another group of diarrheal diseases is seen which may be due to infection introduced from without, through water, milk, or other food; to these the term dysentery is more often applied. These cases have been found to be associated with definite bacteria or amebae. It is likely that intestinal disease of this type may supervene upon other forms.

ACUTE INTESTINAL INDIGESTION AND DIARRHEA

The term *intestinal indigestion* is not an accurately descriptive one but is as satisfactory as any that has been proposed until more exact knowledge as to the etiology and pathology of the condition is available.

The cases included in this chapter comprise many types which, however, are closely allied and shade into one another. Though the extremes of the series differ as widely as possible, yet intermediate types of almost every grade are met with. They are discussed under a single heading, since they have no essential anatomical differences, nor, so far as yet determined, do they differ etiologically. Some of the attacks are so mild in character that in children with normal resistance, and receiving prompt treatment, they may last but a few hours. On the other hand, they may be so rapid in development and so severe as to result in death in a few hours; or, beginning with less intensity, they may be the starting point of prolonged functional disorders or may prepare the way for the development of infectious processes.

Etiology.—The most important causes have been mentioned in the foregoing discussion on the General Etiology of Diarrheal Diseases. A predisposition to attacks is furnished by summer weather, a delicate constitution, and any previous derangement of digestion. The exciting cause of an attack may be the use of improper food, overfeeding or some sudden change in food as in weaning; but, the food remaining unchanged, it is often other influences affecting the child, such as summer heat. The most striking thing about these cases is their prevalence during hot weather. Year after year for generations have been repeated in New York the conditions which are graphically represented in Fig. 33, viz., an epidemic which, beginning in June, rapidly increases in severity, reaching its height in July or August, from which time it diminishes steadily, regularly coming to an end in September.

Despite the fact that since 1886 many series of bacteriological studies of the intestinal discharges have been made by Booker and by Park in this country, by Escherich, Baginsky, and others in Germany, our knowledge of this subject is still very incomplete. So far as is now known, no one form of bacteria can be assigned as the cause of this group of diarrheas. There seems to be evidence that the Shiga bacillus may

produce diarrheal disease which clinically does not differ from this type. But it is wanting in so large a proportion of cases, that it can not be regarded as the specific cause. With existing knowledge it seems probable that there are a number of organisms present in the intestine in disorders of digestion, which, under favorable conditions, may multiply to such a degree as to produce serious disturbances; but the rôle of the micro-organisms may be regarded as a secondary one.

There are certain cases in which symptoms of a severe type develop abruptly in children previously quite well. These only are to be regarded as examples of acute milk poisoning. Although the bacteria in the milk may have been previously destroyed by sterilization, the toxins produced by them may still be present. This is doubtless the explanation of the simultaneous development of several cases in families or institutions.

We can not believe that direct contagion is the usual way in which this disease is spread. When occurring in institutions or in families, it usually happens that a number of children are attacked simultaneously rather than successively, this indicating a common cause, usually to be found in the food, the surroundings, or the atmospheric conditions.

The irritating substances producing the diarrhea are largely the lower fatty acids. These are derived from the sugar and fat of the food probably as the result of bacterial action. It is not the presence of abnormal bacteria that brings about this result so much as the altered conditions under which they multiply and operate. These altered conditions may depend upon changes in the gastric, biliary, pancreatic and intestinal secretions or upon other factors that we do not yet understand.

Lesions.—In the milder cases which end in recovery, the anatomical changes are probably negligible. In those which prove fatal from the disease itself, or from some associated condition, the lesions may be a superficial catarrhal inflammation affecting the entire gastro-enteric tract, but varying much in severity in the different regions and in the different cases. Even after the most severe symptoms no lesions of consequence may be found.

The gross appearances may show but little that is abnormal. The walls of the stomach may be coated with mucus, and the mucous membrane may show congestion, generally in patches. The mucous membrane of the small intestine may be pale throughout; there are often irregular areas of congestion. With this there may be redness and swelling of Peyer's patches and the solitary follicles. In the colon the mucous membrane may be congested. The solitary follicles are usually swollen. The changes described are not at all uniform, and do not differ very greatly from the appearances often seen in the intestines when patients have died of other diseases.

In the cases classed clinically as cholera infantum, the pathological

changes are sometimes more characteristic. The greater part of the small intestine, and sometimes the entire colon, are distended with gas, and contain material of a grayish-white color about the consistency of a thin gruel. It has a mawkish odor, but usually not a very offensive one. The mucous membrane of the entire intestinal tract is in most cases pale. Sometimes this is only in the small intestine, while there are areas of congestion in the colon. If cholera infantum has been engrafted upon some other pathological process in the intestines, as is not infrequent, there is found post-mortem evidence of this in the form of severe catarrhal inflammation, sometimes old ulcerations.

Unless autopsies are made very soon after death—at most within four hours—it is not safe to draw conclusions from the conditions found, as post-mortem changes take place rapidly, and resemble those of the disease under consideration. This applies particularly to the microscopical examination of the epithelium. The cells may still be present, but with the cell protoplasm and nuclei so changed that they do not stain normally. In more severe and prolonged cases the superficial epithelium in places is entirely destroyed.

The changes in and about the blood-vessels are variable. The small vessels may be distended, and there may be hemorrhages or an exudation of leucocytes in their neighborhood. These appearances are seen either in the mucous or submucous layer. Peyer's patches and the lymph nodules may be enlarged from cell-proliferation.

The lesions in other organs are less frequent and less severe than in the more protracted cases of ileocolitis. Acute bronchitis and bronchopneumonia are frequent. Acute degeneration of the kidney is found to some degree in every case which is severe enough to cause death, and in a few there is acute nephritis. The liver may be much enlarged and very fatty or of normal size, but degeneration of the liver cells is frequent. There may even be small areas of necrosis. In rare cases a general septicemia, due most frequently to the streptococcus, is present.

Symptoms.—Clinically, these cases may be divided into four groups: (1) The mild form, with definite local symptoms, but few general ones; they may be of short duration or protracted; (2) the severe form in which there are not only local but marked constitutional symptoms, fever, etc.; (3) cholera infantum; (4) severe forms complicated by acidosis.

The Mild Form.—In infants, the symptoms are seldom limited either to the stomach or to the intestine, although in one case the disturbance of the stomach is slight and that of the intestine serious, and in another the reverse may be observed. In these little patients the intestinal symptoms are more frequent, and, as a rule, more severe than those referable

to the stomach. In older children it is not uncommon to see the intestinal symptoms alone. In infants, if the attack develops suddenly, gastric symptoms are usually present; if more gradually, they are usually absent. The local symptoms are colicky pain, tympanites, and later diarrhea. The constitutional symptoms, prostration and nervous disturbances, are slight or absent. Pain is indicated by the sharp, piercing cry, great restlessness, and drawing up of the legs. Tympanites is rarely very marked. The stools are always increased in number and are from four to twelve a day. If more frequent they are very small. The first stools are more or less fecal, but this character is soon lost. The color is at first yellow, then yellowish-green, and finally often grass-green. This color is due to biliverdin. If the child has been taking milk, masses of undigested milk, chiefly fat, are present. The reaction of the stools is almost invariably acid. The odor may be sour, or it may be foul. The stools are much thinner than normal, and often frothy from the presence of gases. Blood is not present, nor is much mucus seen, unless the symptoms have lasted several days. The microscope shows, in addition to food-remains, epithelial cells, usually of the cylindrical variety, which are numerous in proportion to the severity and duration of the attack. The bacteria are the ordinary forms found in the feces.

The course and termination of the disease depend upon the previous condition of the patient, the nature of the exciting cause, and the treatment employed. In a previously healthy child, if the cause is at once removed and proper treatment instituted, the severe symptoms rarely last more than a day or two, and in four or five days the patient may be quite well. In delicate infants, a severe attack of acute intestinal indigestion in the hot season is likely to prove the first stage of a pathological process which may continue until serious organic changes in the intestine have taken place. This result may not follow the first attack, but one is often succeeded by others until it occurs. If circumstances are such that proper dietetic treatment and general hygienic measures can not be carried out, this termination is very common.

In older children most of the cases seen are of the milder type. The onset is often with vomiting; pain is generally mild and precedes diarrhea by several hours. It is seldom localized but is more often referred to the navel. The stools are loose, frequent, and contain undigested food, and are of almost every conceivable color and variety. The temperature, if elevated at all, is so only for a short time. There is anorexia and a coated tongue. With proper treatment the attack is usually over in a few days. It is very seldom followed by the severer types of diarrhea, as is so commonly the case with infants.

The Severe Form.—This may follow after several days of an apparently mild attack, especially during hot weather or if improperly

treated. In the cases developing suddenly, the clinical picture is quite a definite one.

An infant is restless, cries much, sleeps but a few minutes at a time, and seems in distress. The skin is hot and dry, the temperature rises rapidly to 102° or 103° F., sometimes to 106° F., and all the symptoms indicate the onset of some serious illness. He may lie in a dull stupor, with eyes sunken, weak pulse, and general relaxation, or there may be restlessness, excitement, and even convulsions. There may be great thirst, so that everything offered is eagerly taken, or everything may be refused. Vomiting may be an early and important symptom. It is first of food, often that which was taken many hours before; retching continues even after the stomach has been emptied, so that mucus, serum, and sometimes bile may be ejected. Vomiting does not usually persist throughout the attack, and in many cases it is absent altogether. Diarrhea is sometimes delayed for several hours after the beginning of the grave constitutional symptoms. At first there are fecal stools, then great bursts of flatus, with the expulsion of a thin yellow material with an offensive odor. Four or five such discharges may occur in as many hours. At other times the stools are gray, green, or greenish-yellow, and sometimes brown. The characteristic features are the amount of gas expelled, the colicky pains preceding the discharges, and the foul odor. After the first day the stools may be almost entirely fluid, varying in number from six to twenty a day, and often large even then; but their offensive character frequently disappears. After two or three days mucus appears. The microscopical examination of the stools shows great numbers of separate epithelial cells, and sometimes groups of cells attached to a basement membrane. In addition there may be leucocytes and some red blood-corpuscles.

In many cases the free evacuation of the bowels is followed by a drop in the temperature and subsidence of the nervous symptoms, and the child may fall asleep. The prostration, though often great in the beginning, may not be of long duration. In the most favorable circumstances, after one or two days of severe symptoms, convalescence may take place. The stools continue frequent for five or six days, but gradually assume their normal character, and recovery follows. The chief factors contributing to such favorable results are a good constitution on the part of the child, prompt and intelligent treatment at the outset, and proper feeding afterward.

If the circumstances are not so favorable, if the patient is a very young or delicate infant, there may be no reaction from the first severe symptoms, and the attack may terminate fatally in from one to three days. In such cases the temperature remains high; the stomach may or may not be disturbed; but the diarrhea, prostration, and nervous

symptoms continue, and death occurs from exhaustion, in coma or convulsions. Instead of a rapidly fatal termination, the severity of the early acute symptoms may abate somewhat, and the attack assume the character of ileocolitis, with a lower but continuous temperature of 100° to 102° F., frequent mucous stools, wasting, etc. The urine is scanty and concentrated, and in most of the severe cases with very high temperature contains a small amount of albumin, and occasionally a few hyaline and granular casts. These are the result of degenerative changes in the renal epithelium. In rare cases there are evidences of acute nephritis. Bronchopneumonia is sometimes seen.

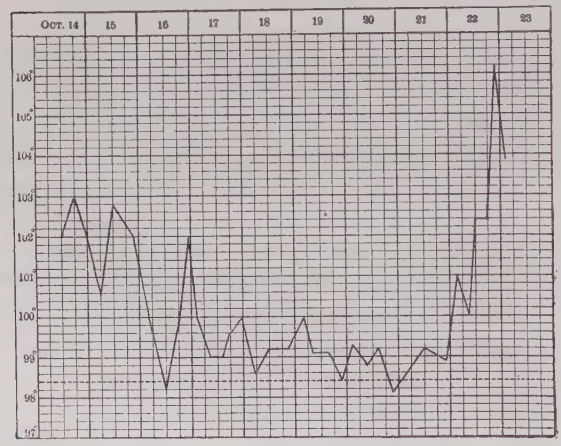


Fig. 35.—Severe Intestinal Indigestion with Fatal Relapse. Infant five months old; early symptoms, both intestinal and nervous, severe; rapid improvement followed stopping milk, free catharsis and irrigation. After stools had been nearly normal for three days relapse occurred, apparently from adding milk to the diet, although less than two ounces a day were given. Autopsy: Only mild intestinal lesions were present; other organs essentially normal.

It not infrequently happens, after the storm of the acute attack with its high temperature, intense prostration, and grave nervous symptoms is passed, and the stools are so much improved that the patient is regarded as out of danger, that all the former symptoms may develop with such rapidity and severity as sometimes to carry off the patient in from twelve to twenty-four hours. Such relapses are generally excited by some mistake in the diet, usually that of allowing milk too soon. The amount of milk given may be small, and yet the symptoms follow its administration so soon that there can be little doubt regarding the connection between them (Fig. 35). Besides such severe cases, many milder relapses are seen; the cause is usually some error in diet.

Attacks of acute intestinal indigestion with severe constitutional symptoms in which there is at first no diarrhea, but constipation instead, are most puzzling and frequently serious. Fortunately, they are not of common occurrence. It is somewhat difficult to explain such cases. There seems to exist for the time almost complete intestinal paralysis. The toxic materials are locked up in the small intestine, for the colon

is frequently quite empty. When one meets such a case he can appreciate the fact that diarrhea is a conservative process of the greatest possible value.

In children over two years old there are seen some features which differ from those of the cases above described as occurring in infants. The attacks are more often due to other causes than to milk. Vomiting does not occur so readily as in infants, pain is a more prominent symptom, and the temperature, as a rule, is lower. The nervous symptoms are much less prominent. Skin eruptions, however, are more frequently seen, particularly urticaria, which is a feature of many attacks, and in obscure cases has some diagnostic value. Although often beginning with severe symptoms, these cases usually make good recoveries; there is much less danger of repeated attacks or of the development of ileocolitis than in the case of infants.

Cholera Infantum.—This is only one type of the severe form of acute indigestion, yet clinically it differs from the others sufficiently to deserve separate consideration. It is not, however, a frequent form. What it is that determines the marked and characteristic symptoms in cholera infantum is entirely unknown.

Cholera infantum rarely occurs in an infant previously healthy. As a rule, there is some antecedent intestinal disorder. The development of the choleriform symptoms is usually very rapid, and a child, who perhaps has been regarded as scarcely ill enough to require a physician, may be brought, in the course of five or six hours, to death's door.

Usually there are general symptoms, such as prostration and a steadily rising temperature, for a few hours before the vomiting and purging, or these symptoms may be the first to excite alarm. Vomiting may precede diarrhea, or both may begin simultaneously. The vomiting is very frequent. First, whatever food is in the stomach is vomited, then serum and mucus, and sometimes there is regurgitation from the small intestine. If vomiting subsides for a time, it is almost sure to begin anew with the taking of food or drink. The stools are frequent, large, and fluid, and may occur once or twice an hour. They are of a pale green, yellow, or brownish color in the beginning, but as they become more frequent they often lose all color and are almost entirely serous. The sphincter is sometimes so relaxed that small evacuations occur every few minutes. The first stools are usually acid, later they are neutral, and when serous they are alkaline. In most cases they are odorless; in rare instances they are exceedingly offensive. Microscopically the stools show large numbers of epithelial cells, some leucocytes, and immense numbers of bacteria.

Loss of weight is more rapid than in any other pathological condition in childhood; it may be as much as a pound a day. The fontanel is

depressed, and in rare instances there may be overlapping of the cranial bones. The general prostration is great almost from the outset. The face, better, perhaps, than any single symptom, indicates what a profound impression has been made upon the system. The eyes are sunken, the features sharpened, the angles of the mouth drawn down, and a peculiar pallor with an expression of anxiety overspreads the whole countenance, which becomes almost Hippocratic. In the early stages the nervous symptoms are those of irritation. Later, these symptoms give place to dulness, stupor, relaxation, and coma or convulsions.

The temperature is invariably elevated, and usually in proportion to the severity of the attack. In cases recovering, it has generally been from 102° to 103° F., while in fatal cases it has risen almost at once to 104° or 105° F., and often shortly before death it has reached 106° or even 108° F. Such temperatures may occur with a clammy skin and cold extremities, and are discovered only with the aid of a thermometer. The pulse is always rapid, and very soon it becomes weak, often irregular, and finally almost imperceptible. The respiration is irregular and frequent, and may be stertorous. The tongue is generally coated, but soon becomes dry and red, and is often protruded. The abdomen is generally soft and sunken. There is almost insatiable thirst. Everything in the shape of fluids, especially water, is drunk with avidity, even though vomited as soon as it is swallowed. Very little urine is passed, sometimes none at all for twenty-four hours; this largely depends upon the great loss of fluid by the bowels.

In the fatal cases there is hyperpyrexia, a cold, clammy skin, absence of radial pulse, stupor, coma or convulsions, and death. The diarrhea and vomiting may continue until the end, or both may entirely cease for some hours before death occurs. The patients may pass into a condition resembling the algid stage of epidemic cholera, and die in collapse. In other cases, after the first day of very severe symptoms, the discharges diminish, but the nervous symptoms become specially prominent. There is restlessness and irritability or apathy and stupor. The fontanel is sunken; the eyes are half open and covered with a mucous film; respiration is irregular and superficial, sometimes even Cheyne-Stokes; the pulse is feeble, irregular, or intermittent; the muscles of the neck drawn back; the abdomen retracted. The temperature is not elevated, but normal or subnormal. From this condition recovery may take place or the symptoms may merge into those of ileocolitis; but much more frequent than either of the foregoing is the fatal termination.

The nervous symptoms have been ascribed to cerebral anemia, cerebral hyperemia (venous), edema of the meninges, thrombosis of the cerebral sinuses, and uremia. Although we have examined the brain in almost all our autopsies upon patients dying from diarrheal diseases, we have

never in such cases seen sinus thrombosis, and but rarely edema. Cerebral hyperemia is often met with in cases dying in convulsions, but not with any regularity otherwise. Nor have our observations upon the kidneys confirmed those of Kjellberg, whom most of the writers since his day have quoted, as to the great frequency of nephritis. A scanty, concentrated, and hence irritating urine is the rule, and a small amount of albumin and an occasional hyaline cast not uncommon; but either clinical or pathological evidence of a serious amount of nephritis has been, in our own experience, extremely rare.

An infrequent complication of cholera infantum is sclerema. This condition is found associated with muscular contractions, subnormal temperature and other signs of the most extreme depression. These cases are almost invariably fatal.

Of the children with true cholera infantum which have come under our notice, fully three-quarters have died.

Acidosis.—In the course of the severe form of diarrhea or of cholera infantum, symptoms referable to the nervous system and respiration may appear. There may be excitement and sleeplessness with a frequent, shrill, piercing cry. Later on there may be somnolence gradually increasing to stupor or even coma. The type of respiration is the most characteristic evidence of acidosis. This is altered so that there is an increased ventilation of the lungs, i. e., exaggerated inspiration and expiration. This is often difficult to recognize in its early stages, but frequently develops into a marked dyspnea of the "air hunger" type, without pause or cyanosis and without any evidence of obstruction. There is often a polymorphonuclear leucocytosis, generally between 20,000 and 30,000. There may be sugar in the urine which, if lactose is given in the food, is said to be galactose and lactose, or saccharose if this sugar is being taken. There are in addition the symptoms of severe general prostration.

When such symptoms are present, especially the nervous and respiratory ones, the condition is very grave. The majority of the children with manifest hyperpnea die, although life may be prolonged for several days. Though the hyperpnea may cease as the result of treatment, death usually occurs; for many abnormal processes at present not understood have undoubtedly been initiated and are sufficient to cause death.

It is to the train of symptoms just described that the name "food intoxication" (*alimentäre intoxication*) has been given by Finklestein. He claims that this condition is the result of the presence of products of intermediary metabolism, imperfectly elaborated, and that they are directly poisonous. Evidence of their presence is, however, lacking.

Recent studies have shown that in these cases there is an acidosis, that the disturbances of respiration are referable to this condition, and that the gravity of the symptoms is probably dependent directly upon

this acidosis. It has been shown that accompanying the hyperpnea there is a low carbon dioxid tension in the alveolar air; that the greater the hyperpnea, the lower the carbon dioxid tension; that in the most severe forms there is an increase in the hydrogen-ion concentration of the blood serum; that there is a great diminution of the alkali reserve of the blood and that a greatly increased quantity of alkali can be taken before the urine becomes alkaline. Soda bicarbonate, given by mouth, intravenously or subcutaneously, causes a cessation of the hyperpnea and a return of the alkalinity of the blood to normal. This furnishes a definite indication for treatment. But the relief of the acidosis does not necessarily cure the diarrhea. There is no doubt that there is an alteration in the normal relation between the acids and alkalies so that the former are in relative excess. What causes this alteration is not known at the present time. It is not due, as a rule, to an excess of the acetone bodies. These are but moderately increased in amount.

Diagnosis.—The acute gastric and intestinal symptoms which mark the beginning of many febrile diseases in infancy, particularly the exanthemata and pneumonia, are often difficult to distinguish from the more severe attacks of acute indigestion with constitutional symptoms. The question to decide is whether the digestive symptoms are the cause or the result of the fever. It is sometimes not until the case has been watched for some time that one can be certain. Usually when digestive symptoms are secondary they diminish after the first day or two, although the severity of the general symptoms may steadily increase. The characteristic features of the primary disease may also appear. When the nervous symptoms of the severe form of acute indigestion are prominent at the outset, it is sometimes difficult to exclude meningitis. We have seen many cases where great doubt existed for several days. One should always hesitate to make a diagnosis of meningitis when marked diarrhea is present.

Prognosis.—The milder forms of acute intestinal indigestion do not often prove fatal, except in young infants or those already suffering from malnutrition. In all cases the prognosis depends upon the previous health of the child, his surroundings, the season of the year, and whether or not the case receives prompt and proper treatment. Severe forms of the disease, especially those associated with nervous or respiratory symptoms, are very serious. A continuously high fever is a bad prognostic sign. The existence of rickets, pertussis, or any other disease, greatly increases the gravity of the attack. True cholera infantum is nearly always fatal.

Prophylaxis.—A better understanding of the etiology brings with it great possibilities in the prevention of this disease.

Prophylaxis must have regard, first, to the hygienic surroundings of

children, and to all sanitary conditions of cities. City children should be sent to the country, whenever it is possible, for the months of July and August. Where a long stay is impossible, day excursions do much good. The fresh-air funds and seaside homes have done much in New York to diminish the mortality from diarrheal diseases.

The second part of prophylaxis relates to food and feeding. Maternal nursing should be encouraged by every possible means. Nothing is better established than the close relation existing between artificial feeding and diarrheal diseases. Yet, as stated elsewhere, it is not artificial feeding *per se* but ignorant and improper feeding. Among infants in private practice who are properly fed these attacks are not common.

Overfeeding is particularly to be avoided during days of excessive heat. It is at such times an excellent rule with infants to diminish each feeding by at least one-half, making up the deficiency with water, and to give water very freely between the feedings. In summer all water given to infants or young children should be boiled. Children, like adults, require less food in very hot weather, but more water. Infants cry more from thirst and heat than from hunger, and even those at the breast are likely to be given too much food. Infants should never be fed more frequently, but always less frequently, during hot weather.

A very important work in practical philanthropy among the poor of our large cities in summer is to provide means for supplying pure milk to infants. This has been done on a large scale in many American cities, and it is one of the important agencies that have effected a decided reduction in the death-rate from diarrheal disease. It is not enough to furnish to the poor a pure, clean milk in bulk, or even in sealed quart bottles. The advantages of such milk may be entirely lost by the way in which it is cared for in the home or the way in which it is fed to infants. Since the milk must usually be kept at home without ice, sterilization is advisable. When milk is distributed from milk stations, a physician should be in charge who can keep a general supervision over the children, and advise as to the quantity of food, number of feedings, and the formula to be used. His work should be supplemented by visits of nurses to the homes of patients. An essential feature is to keep such close supervision over the infants as to recognize at once and promptly treat slight disturbances of digestion.

But even more important than pure milk is the education of the poor in all matters relating to infant feeding and hygiene. In no way can this educational work better be done than in connection with milk distribution.

Hygienic Treatment.—If the attack is a severe one and occurs in the excessive heat of midsummer, and does not readily yield to treatment, the child should, if possible, be sent to a cooler place. Convalescent cases

should also be sent away on account of the dangers of relapse. Children must not only be sent away, they must be kept away until quite recovered. In cases which have become somewhat chronic, more can sometimes be accomplished by a change of air than by all other means.

Fresh air is of the utmost importance for all diarrheal cases in summer. No matter how much fever or prostration there may be, these children do better if kept out of doors the greater part of the day. Children should be kept quiet, and especially should not be allowed to walk, even if they are old enough and strong enough to do so.

The clothing should be very light flannel; a single loose garment is preferable. Linen or cotton may be put next the skin if this is very sensitive and there is much perspiration. At the seashore and in the mountains, care should be taken that sufficient clothing at night is supplied. Bathing is useful to allay restlessness, as well as for the reduction of temperature. Scrupulous cleanliness should be secured in the child's person and clothing. Napkins, as soon as soiled, should be removed from the child and from the room and placed in a disinfectant solution. Excoriations of the buttocks and genitals are to be prevented by absolute cleanliness and the free use of some absorbent powder, such as starch and boric acid.

Dietetic Treatment.—It is of the first importance to remember that during the early stage of the acute cases, digestion is practically arrested. To give food at this time, manifestly can do only harm.

In nursing infants the severe forms of the disease are extremely rare; but the breast should be withheld so long as a disposition to vomit continues, and no food whatever given for at least twenty-four hours. Thirst may be allayed by giving frequently, but in small quantities, boiled water or thin barley or rice water or weak tea sweetened with saccharin. If these are refused or vomited, absolute rest to the stomach will do more than anything else to hasten recovery. After the stomach has been allowed to rest for twenty-four hours, it is generally safe to permit a nursing child to take the breast tentatively. The intervals of nursing should not be shorter than four hours, and the amount allowed at one feeding should not be more than one-fourth the usual quantity. This may be regulated by allowing an infant to nurse at first only two or three minutes. Between the nursings may be given boiled water or barley water. Nursing may be gradually increased, so that in three or four days the breast may be taken exclusively.

In infants who are being artificially fed, all food, and especially milk, should be stopped at once. Sweet milk should not only be withheld during the period of acute symptoms, but for several days thereafter. Besides the articles mentioned above as suitable for the period of most acute symptoms the following substitutes for milk will be found

useful: rice or barley water, the farinaceous foods, and broth or bouillon made of veal, chicken, mutton, or beef. Water may be allowed freely at all times unless there is much vomiting.

When milk is begun it should be remembered that the sugar is more likely to disturb digestion than any other element and that sugar and fat together are very badly borne. For this reason some form of fermented milk, buttermilk or protein milk is to be preferred. This latter may be given except in the most severe forms of the disease and except when vomiting is marked, almost from the beginning of symptoms. After twenty-four hours of preliminary starvation, if the symptoms are very acute and after cleansing of the intestinal tract has taken place either from the diarrhea itself or from cathartics or irrigations, its use may be begun. It has a marked effect in counteracting the diarrhea and is well borne by almost all infants except those under two or three months of age. At first the protein milk should be given in small amounts, one or two ounces every four hours, and to infants under six months of age diluted with an equal quantity of water. The increase in amount and in strength should be gradually made according to the improvement in symptoms. No sugar should be added until a day or two after the stools have become quite firm in consistency and not more numerous than three or four a day.

The sugar should be one of the dry preparations containing maltose such as dextrimaltose, Sohxlet's *nährzucker*, Liebig's *nährmaltose* or cane sugar. Lactose should not be used. The sugar should be added very gradually, beginning with one-quarter ounce a day and increased up to four or five per cent of the food. If loose stools result the sugar should be discontinued. A return to sweet milk should be made gradually and with caution. To this no sugar should be added until it has been demonstrated that the diluted milk can be tolerated. Wet-nurses are not to be employed during the acute symptoms, but during the period of prolonged malnutrition which follows an acute attack they may be of the greatest service.

The same general principles of feeding should be applied in older children. All food is to be withheld until the vomiting ceases, when broths and thin gruels may be given; later, buttermilk, kumyss and protein milk. Junket from which the whey has been carefully strained is very useful in checking diarrhea. Solid food should not be allowed for several days after the stools have become normal.

Medicinal and Mechanical Treatment.—It must be borne in mind that we are not treating an inflammation of the stomach or intestines, although such may be the ultimate result of the process. The essential condition, it should be remembered, is one of indigestion and intoxication arising from the intestinal contents—food-remains from arrested

digestion, altered secretions, acids, irritating and toxic substances produced by chemical and bacterial action—to which not only the constitutional but the local symptoms are chiefly due. We can hardly do better than to imitate and assist Nature in her treatment of this condition. Let us consider what this is. Lest too much food be swallowed, appetite is taken away; by vomiting, the stomach is emptied; to neutralize the acid poisons in the intestine, an alkaline serum is poured out from the intestinal walls; to remove irritant poisons, increased peristalsis is excited.

The first indication is, therefore, to evacuate the stomach and the entire intestinal tract at the earliest moment. Unless thorough evacuation of the bowels has taken place, treatment should not be begun with the use of measures to stop the discharges. To empty the stomach is not necessary in every case, since the initial vomiting may have done this effectively. If vomiting persists one may resort to stomach-washing. A single washing is generally sufficient, and if employed at the outset may shorten the attack. With high fever and great thirst, it is often advisable to leave a few ounces of water with ten to fifteen grains of bicarbonate of soda in the stomach. As a substitute for stomach-washing in children over two years old, or where it can not be employed, copious draughts of boiled water may be given. This is taken readily, and as it is usually vomited almost at once it may cleanse the stomach thoroughly. If there is distention with fever and foul stools, cathartics are indicated, but if the diarrhea has been profuse cathartics should not be employed. There is no greater mistake than to think that the character of the stools is likely to be improved by calomel or castor oil. The stools contain little if any fecal matter; what is passed by the bowel consists almost entirely of intestinal secretions.

To clear out the small intestine, only cathartics are available. For the colon, we may in addition employ irrigation. Calomel, castor oil, or the salines may be used as cathartics, and enough of any one of them must be given to clear out the intestinal tract thoroughly. Calomel has the advantage of ease of administration: one-fourth of a grain should be given every fifteen or twenty minutes up to four or six doses. When the stomach is not disturbed, castor oil is to be preferred as it is not so irritating, causes little griping and is very certain. Two drams should be given to a child six months old, and half an ounce to one of four years. Of the salines, the best are the sulphate of soda and Rochelle salts; from one to three drams may be given, well diluted, divided into four or five doses, at twenty-minute intervals.

Cathartics may be employed later in the disease if the stools become foul or there is distention, but care should be taken not to continue to irritate a hypersensitive intestine.

Early irrigation of the colon is advisable in all cases, as it hastens the effect of the cathartic and removes at once much irritating and offensive material. It should be done two or three times the first day, but afterward once daily is generally sufficient. A saline solution (one tablespoonful of salt to two quarts of water), at a temperature of about 100° F., is to be preferred; and a rectal tube well inserted should always be used. Thorough initial evacuation, no food, but plenty of water for twenty-four hours, and careful feeding after that time, are all the treatment that is necessary in most cases.

Other drugs are of secondary importance. Their value is certainly very much overestimated. It is very doubtful whether as yet any proper antiseptic treatment of the gastro-enteric tract is possible.

Of the drugs which are used to influence the intestinal process, bismuth is to be preferred. It has the advantage that it rarely causes vomiting, and that most of its preparations can be given in large doses. The subcarbonate is the safest. It may be given in doses of from ten to twenty grains every two hours, to a child of one year. Like the subnitrate it is insoluble and is best given suspended in the food or in water. It usually blackens the stools. It may be kept up throughout the attack. Our experience leads us to place little reliance upon astringents. They do little good, and often much harm.

While opium in some form is required in many cases, it is capable of doing much harm. The chief indications for opium are great frequency of movements and severe pain. It is contraindicated until the intestinal tract has been thoroughly emptied; also when the number of discharges is small, particularly if they are very offensive; it is especially to be avoided in the early stage of very acute cases, and never to be given when cerebral symptoms and high temperature coexist with scanty discharges. Opium is admissible after the tract has been thoroughly emptied. It is particularly indicated when there is a persistence of large, fluid movements attended by symptoms of collapse, and in all cases approaching the cholera-infantum type. Nothing requires nicer discrimination than the use of opium in diarrhea. It is wise to administer it always in a separate prescription, and never in composite diarrheal mixtures. The dose should be regulated according to its effect upon the number of stools. Enough is to be given to produce a distinct effect —the control of excessive peristalsis and the diminution of pain—but never enough to check the discharges entirely, or to cause stupor. The uncertainty of absorption must also be remembered; a second full dose should not be given until a sufficient time has elapsed for the effect of the first to pass away. For an average child of six months, ten minims of paregoric, one-half minim of the deodorized tincture, or one-half grain of Dover's powder, may be used as an initial dose, to be repeated every

one, two, or four hours, according to the effect produced. In severe cases it may be necessary to increase the dose considerably. When urgently required morphin should be given hypodermically, one-sixtieth of a grain to an infant of six months, to be repeated in two hours if no effect is seen.

Stimulants are often required in severe cases. The prostration is great and develops rapidly; frequently almost no food can be assimilated for twenty-four or thirty-six hours, while the drain from the discharges continues. The general condition of the patient is the best guide as to the time for stimulation and the amount required. Brandy is the best preparation for general use. An infant a year old may, as a maximum, take half an ounce of brandy, well diluted, in twenty-four hours. Caffein and camphor may also be given. While the use of stimulants is indicated in many cases their effects are disappointing. Taken by mouth they are frequently vomited. It is then necessary to give caffein and camphor hypodermically. In cases of extreme prostration and collapse the hot bath, mustard to the extremities and sometimes the mustard pack are beneficial.

When acidosis is present as indicated by dyspnea of the "air hunger type" or by stupor, alkalis are indicated, especially sodium bicarbonate. This may be given by mouth, intravenously or subcutaneously. Enough should be given to render the urine alkaline and to keep it so. As there is a greatly increased tolerance for alkalis the amount required may be large. With a normal infant the administration of fifteen grains of bicarbonate of soda is sufficient to render the urine alkaline. With acidosis six or eight times this amount may be required. It should be given in doses of fifteen to thirty grains every two hours. If vomited, it should be given subcutaneously or intravenously. The latter method is preferable if the injection can be made through the skin without exposing a vein. As much as 50 c.c. of a four per cent solution of sodium bicarbonate may be given at a time. If a vein can not be found, the solution may be injected subcutaneously. This method has the disadvantage of requiring a solution [1] which is somewhat difficult to prepare and even with all precautions sloughing may result from its use. The injection should be repeated with sufficient frequency to maintain the urine alkaline.

The early evidences of acidosis are difficult to recognize clinically; it is, therefore, safer to give soda in all severe cases of intestinal indiges-

[1] The solution is prepared by sterilizing a four per cent solution of carbonate of soda. This being irritating it is necessary to transform it to the bicarbonate by passing carbon dioxid from a Kipp generator or a cylinder through the cold solution until it is colorless to phenolphthalein. It may then be used. Solutions of bicarbonate cannot be sterilized without decomposing.

tion in quantity sufficient to maintain an alkaline reaction of the urine.

With the severe form of the disease, especially in the cholera infantum type, the great drains of water and salts from the blood may in itself be serious.

Vomiting is usually present which prevents the giving of water by mouth; enemata are not retained. It is therefore necessary in many cases to give water subcutaneously. This may be given by hypodermoclysis as described elsewhere in amounts varying from six to ten ounces daily. The bicarbonate of soda solution mentioned above may be employed or simple saline solution of a strength of eight-tenths of one per cent. These injections should be repeated until the cessation of vomiting allows sufficient water to be taken by mouth. Their beneficial effect is frequently striking. Glucose in three-per-cent strength may be added to the saline solution but in the majority of instances the sugar content of the blood is within normal limits or even abnormally high. Except in prolonged cases therefore the addition of glucose does not seem to be indicated.

CHAPTER VII

DISEASES OF THE INTESTINES.—(Continued)

ACUTE ILEOCOLITIS—DYSENTERY

(Enterocolitis; Enteritis; Inflammatory Diarrhea)

The term ileocolitis is a general one, embracing those forms of intestinal disease in which true inflammatory lesions are present. In the types of cases described in the previous chapter nothing more than superficial changes occur, while in ileocolitis the pathological process continues until there have been produced marked lesions, often involving all the walls of the intestine. Sometimes it is impossible, by symptoms, to draw a line between them. This is especially true of the cases terminating in follicular ulceration of the colon. In certain other forms of ileocolitis the evidences of a severe intestinal inflammation are often manifest from the very outset. This difference is probably due to a difference in the character of the infection. The extent of the lesions depends much upon the duration of the process.

Etiology.—The predisposing causes of ileocolitis are those common to diarrheal diseases in general, and have already been considered. Although seen with especial frequency in summer, and in children under two years old, it may affect those of any age, and occurs at all seasons.

Epidemics are not uncommon in the early fall months. While usually primary, ileocolitis often follows infectious diseases, especially measles, diphtheria, and bronchopneumonia. It frequently occurs, in institutions chiefly, as a terminal infection in infants suffering from extreme malnutrition or marasmus. All other forms of intestinal disease are predisposing causes. The question of contagion is unsettled; if at all communicable, it is feebly so. When it occurs epidemically a common origin seems more probable than that the disease spreads from one patient to another.

The only bacterium that up to the present time has been proven to be capable of producing this form of intestinal disease is the *B. dysenteriae* of Shiga. This organism, or, more properly speaking, this group of closely allied organisms, has now been found in all parts of the world in a sufficient number of cases to establish its etiological connection with ileocolitis. The *B. dysenteriae* was shown by Shiga, in 1898 and 1899, to be the cause of epidemic dysentery in Japan. In 1900, Flexner established its association with tropical dysentery in the Philippines, and in 1902, Duval and Bassett, pupils of Flexner, demonstrated its presence in a series of cases of diarrhea in children at Baltimore.

This organism is very frequently found in cases showing blood and mucus, or much mucus in the stools. Although usually the *B. dysenteriae* is greatly outnumbered by other organisms, it is not uncommon to find it in pure culture. A number of minor differences have been found in the bacilli from different cases; there are, however, two main groups, the division being made by reason of the difference in reaction with litmus mannite; one group is known as the "true Shiga," or "alkaline" type; the other, as the "acid" type,[1] which has been most frequently found in the diarrheal diseases of children in this country, although the true Shiga is occasionally present, and in rare cases they may be associated.

Whether the *B. dysenteriae* is present in normal stools of healthy children is still unsettled. Wollstein at the Babies' Hospital failed to discover its presence in the stools of 56 normal infants. The *B. dysenteriae* has never been found outside the body; we are therefore entirely ignorant both of its habitat and its mode of entry. There are grounds for believing that it appears at times among the saprophytic bacteria of the intestinal contents.

The rôle played by other bacteria, especially the streptococcus, in the production of the deeper lesions of the intestine may be an important one. This appears, however, to be rather in the nature of a secondary invasion; but the streptococcus is found at times in such overwhelming numbers that it is considered by some authorities to play the chief part

[1] The "acid" type includes the Flexner-Harris, the "Y" type of Hiss and Russell and the Strong (Manila) subvarieties.

in the production of the lesions. The gas bacillus of Welch, the bacillus pyocyaneus and the other organisms occasionally found in the stools are probably of accidental occurrence.

Lesions.—It is surprising that, so far as is known, a single organism can excite such a variety of lesions. The nature of the anatomical changes apparently depends upon other factors, such as the intensity of the infection, the local resistance, and still more upon the duration of the disease. The association of other organisms must also be considered.

The nature of the lesions in ileocolitis differs greatly, but their position is quite constant; they affect the lower ileum and the colon. In about half the cases only the colon is affected. The lesions of the ileum are usually limited to the lower two or three feet.

Acute Catarrhal Ileocolitis.—In the milder cases there is infiltration of the mucosa. In the severer cases the submucosa is involved, and the infiltration of the mucosa may be so great as to lead to necrosis and the formation of ulcers.

While the lower ileum and the colon are most seriously affected, it is not uncommon to find quite marked changes in a considerable portion of the small intestine, and even in the stomach. In the cases of short duration, the lesions are sometimes more marked in the small intestine than in the colon. The mucous membrane is often coated with tenacious mucus and may appear somewhat swollen. Congestion is a constant feature, and it may be simply upon the folds of the mucous membrane, or about the solitary follicles, or it may be intense and involve the whole intestine for some distance. Small hemorrhagic areas are often seen here and there, widely scattered. In the most severe cases there are marked thickening and uniform congestion. The solitary follicles throughout the colon are usually swollen, projecting above the mucous membrane and about the size of a pin's head. Peyer's patches may be normal, or they may be swollen and congested, or, more rarely, they may be involved when the rest of the mucosa appears healthy. The lymph nodes of the mesentery are usually swollen and acutely congested.

In interpreting the microscopical changes found in the mucosa, the same precautions must be observed as stated in the previous chapter. There is usually loss of the superficial epithelium and of that lining the tubular glands at their orifices. The lumen of the tubular glands is narrowed from pressure due to the swelling of the tissue which separates them, which is partly from edema, and partly from cell infiltration. A thick layer of mucus and round cells, adhering closely to the surface, may resemble a pseudo-membrane (Fig. 36). The superficial portion of the mucosa may be infiltrated with round cells and crowded with bacteria of many kinds; the depth to which this infiltration extends depends upon

the severity and duration of the process. In very severe cases there is found a dense infiltration of the mucosa and of the submucosa also, which in places extends quite to the muscular coat. The lymph nodules of the colon are swollen to a greater or less degree, chiefly from an increase in the number of lymphoid cells. This swelling may be the most prominent feature of the lesion. If the process is sufficiently prolonged, the lymph nodules may break down and ulcerate. The changes in the lymph nodules of the small intestine and in Peyer's patches are similar to those seen in the colon, but are less marked, and are frequently absent altogether. Ulceration in Peyer's patches is extremely rare. The small veins

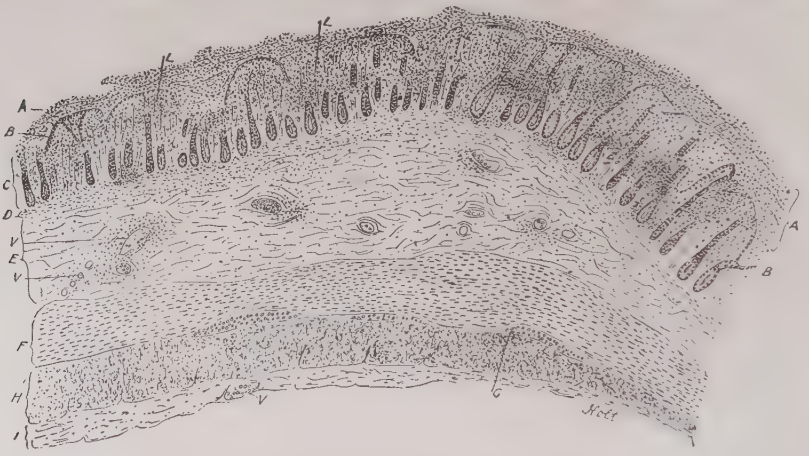
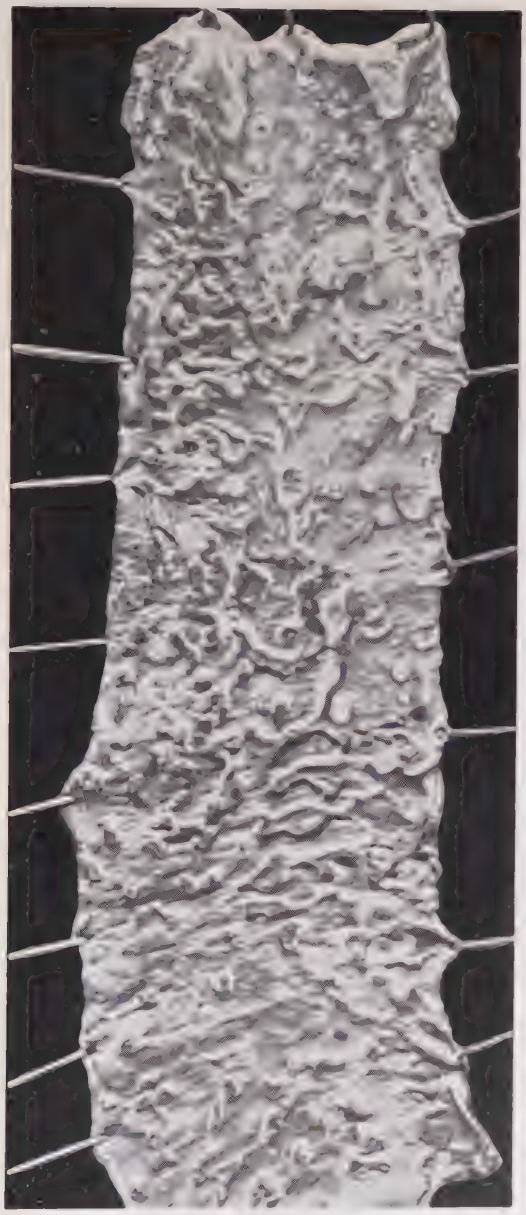


FIG. 36.—ACUTE CATARRHAL INFLAMMATION OF THE ILEUM; SEVERE FORM. The mucosa, *C*, is everywhere densely infiltrated with round cells, compressing the tubular follicles, and in places, *L*, *L*, almost effacing them. Upon the surface of the mucosa is a thick layer of cells and mucus. Beneath this the epithelial arches, *B*, *B*, covering the villi can be seen. The lesions are almost entirely of the mucosa. The only changes in the submucosa, *E*, are groups of cells about the small blood-vessels, *V*, *V*. *History.*—Infant six months old; moderate diarrhea twelve days; severe symptoms with high temperature for six days. There was intense inflammation of the entire colon and lower three feet of the ileum. Intestine greatly congested and thickened. Specimen is from the ileum.

and capillaries of the mucosa and submucosa are usually distended with blood; small extravasations are very common, and occasionally larger ones are seen.

Catarrhal inflammation, except in its very severe form, which is not frequent, causes no lesions that can not readily be repaired. The most persistent change is usually the swelling of the lymph nodules, which may last a long time. There is often pigmentation which may occur as striae in the mucous membrane but which is more frequently limited to Peyer's patches and the solitary lymph nodes. Under the microscope there may be found more or less cell infiltration of the mucosa, but rarely any destructive changes or new connective tissue.

PLATE V



EXTENSIVE SUPERFICIAL ULCERATION OF THE COLON

Female child nine months old; symptoms of acute ileocolitis of fifteen days' duration; temperature, 101° to 104.5° F., and from six to eight stools daily—thin, green, and yellow, but no blood.

Extensive ulceration throughout the colon, most marked in descending portion, from which specimen is taken.

A A are small circular ulcers; B B, larger ones from coalescence of several of these; C C, large areas of ulceration, the mucous membrane being almost entirely destroyed.

Catarrhal Inflammation with Superficial Ulceration.—In the most severe form of catarrhal inflammation which does not prove fatal in the earlier stages, extensive ulceration occasionally takes place; usually these ulcers are seen throughout the entire colon, and occasionally a few are found in the lower ileum. They generally begin in the mucosa overlying the lymph nodules, and while they have a wide superficial area, they do not extend deeper than the mucosa. The small ulcers are circular and usually show at the center a small granular body—the lymph nodule. The larger ulcers result from the coalescence of several small ones, and are irregular in shape. They may be two or three inches in diameter. Sometimes for a considerable distance a large part of the mucosa may be destroyed. Often the entire surface presents a worm-eaten appearance. (Plate V). On microscopical examination there is seen, in the greater part of the ulcer, complete destruction of the mucosa, the submucosa being densely packed with round cells quite to the muscular coat.

Inflammation of the Lymph Nodules—Follicular Ulceration.—Follicular ulcers are found at autopsy in about one-third of the cases dying from diarrheal diseases. They are rarely seen in those which have lasted less than a week, and not often before the middle of the second week. The average duration of the disease in these cases is about three weeks.

In thirty-six cases in which follicular ulcers were found at autopsy, they were present in the small intestine alone in but three cases; in the small intestine and in the colon in six cases; in the remaining twenty-seven they were present only in the colon. When in the small intestine they were seen only in the lower ileum. Ulceration was seen a few times in one or two of the nodules of a Peyer's patch. Ulceration of the large intestine involved the whole colon in about half the cases; while in the remainder the process was limited to its lower portion. The deepest and also the largest ulcers were usually in the descending colon and sigmoid flexure.

In the early stage these ulcers appear as tiny excavations at the summit of the prominent lymph nodules. Later, the whole nodule may be destroyed, and a small round ulcer is formed from one-twelfth to one-fourth of an inch in diameter (Plate VI). These are quite deep and have overhanging edges; when closely set they give the intestine a sieve-like appearance. By the coalescence of several of them, larger ulcers may form which are an inch or more in diameter. At the bottom of these larger ones the transverse striae of the circular muscular coat are often plainly seen. Perforation is extremely rare.

Microscopically the lymph nodules appear swollen, principally from the accumulation within them of round cells. This is followed by softening, which usually begins at the summit of the nodule and extends downward; the reticulum breaks down, and the cellular contents escape

into the intestine (Fig. 37). Softening may begin at the center of the nodule, which ruptures like an abscess. The destruction of the whole nodule leaves a cavity, which is the follicular ulcer. At first the ulcer corresponds in size to the nodule, but infiltration of the adjacent tissue soon takes place, which may become necrotic. In this way the ulcer extends chiefly in the submucous coat. The lesion is never limited to the lymph nodules; but the extent of the other changes found depends

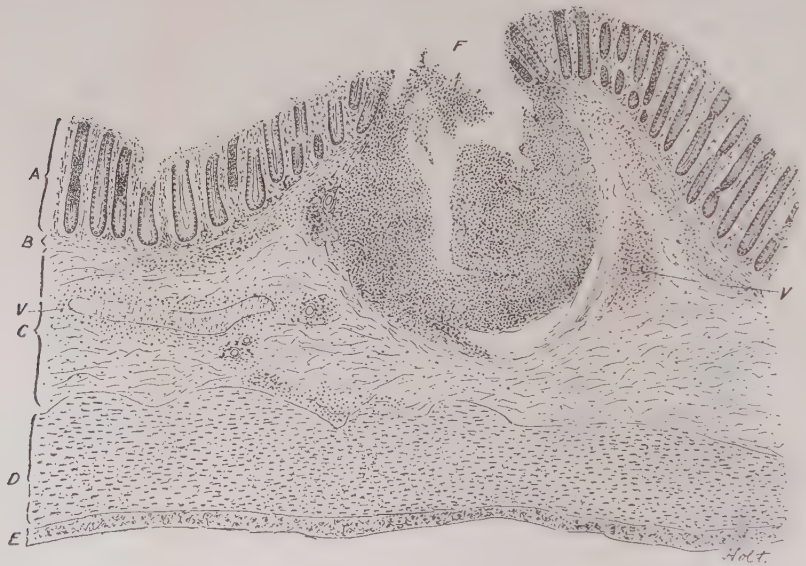


FIG. 37.—LYMPH NODULE OF THE COLON IN THE EARLY STAGE OF ULCERATION.—FOLLICULAR ULCER. The nodule, *F*, is much enlarged, and is breaking down and discharging into the intestine. The other changes are not marked. The superficial epithelium is gone; the mucosa, *A*, shows a slight increase of cells, and in the submucosa, *C*, are nests of cells about the small vessels, *V*, *V*. *History.*—Delicate child, thirteen months old; slight diarrhea four weeks; severe symptoms five days. The colon was filled with ulcers one-twelfth of an inch in diameter, one of which is shown in the illustration.

upon the severity and the duration of the process. In cases fatal after an illness of a week or ten days, we usually find only moderate changes in the mucosa, and in the submucosa.

Follicular ulceration of the intestine in infancy usually terminates fatally if the process is an extensive one. In less severe cases recovery may take place, the ulcers healing by granulation and cicatrization in the course of from four to twelve weeks. It is very doubtful whether stricture ever results from these ulcers in children. Among the very rare lesions are cysts of the colon that are produced by dilatation of some of the tubular glands whose orifices have been obliterated.

ACUTE MEMBRANOUS ILEOCOLITIS.—This is the most severe form of intestinal inflammation seen among children. The most frequent type

PLATE VI

Deep Follicular Ulcers of the Colon

A delicate child, fourteen months old, sick twelve days; stools green, yellow, brown, and watery; no blood; temperature, 100° to 101° F.

The small intestine was normal; ulcers throughout colon. The specimen is from descending colon; the ulcers are deep, and most of them extend to the muscular coat.

of membranous colitis is that with severe acute symptoms, both constitutional and local, with a duration of from six to fourteen days. In young infants its symptoms and course are very irregular, and it may be found at autopsy when no serious intestinal lesion has been suspected.

Gross Appearances.—There is visible to the naked eye usually very little pseudo-membrane and no deep sloughing. The lesion affects the last two or three feet of the ileum and the entire colon, sometimes only the colon. It is exceedingly rare to meet with any marked lesions higher in the small intestine. The most marked changes are near the ileocecal

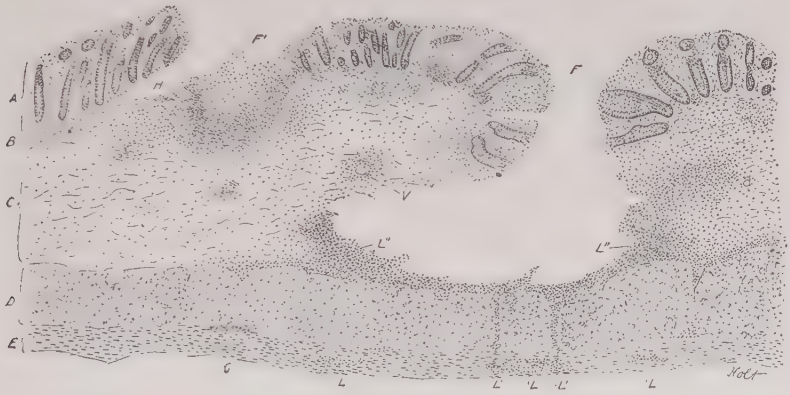


Fig. 38.—Deep Follicular Ulcer of the Colon. A deep ulcer is shown at *F*, a smaller one at *F'*. The separation of the mucosa at *H* is accidental. There is no trace of the lymph nodule from which the large ulcer had its origin. The destructive process has extended laterally in the submucosa, *C*, and the mucosa, *A*, is falling in to fill up the space. In the vicinity of the ulcers, the submucosa is densely infiltrated with round cells *L''*, *L''*, which also are seen in the lymph spaces between the bundles of circular muscular fibers, *L'*, *L'*, and some are seen in the longitudinal muscular coat, *L*, *L*. *History.*—Thirteen months old, delicate; continuous diarrheal symptoms for three weeks. Ulcers found throughout the colon, the largest, one-half an inch in diameter. The illustration shows one of the small ones like those in Plate VI.

valve or in the sigmoid flexure and the rectum. In the ileum they may be quite as severe as in the colon (Plate VII). The intestinal wall is firm and stiff, and is two or three times its normal thickness. It is not thrown into deep folds, as is the healthy intestine when empty. It is very rare to find false membrane that can be stripped off in patches of any considerable size. When membrane exists, the color is a yellowish or grayish green, and the surface is often fissured, giving a lobulated appearance. In the parts where no pseudo-membrane can be seen, the surface is usually of an intense red color and is rough and granular, in striking contrast to the normal glistening appearance. Here and there small extravasations of blood may be seen. In the regions most affected, the normal structures of the mucous membrane—the villi, Peyer's patches, and solitary follicles—can not be distinguished. Except in the

lower ileum the small intestine shows no constant changes, and none are usually found in the stomach.

Microscopical Changes.—These (Fig. 39) are much more uniform than the gross appearances. The most characteristic feature is the exudation of fibrin, which forms a distinct pseudo-membrane upon the surface of the intestine; it may infiltrate the mucosa, and even the submucosa. Fibrin is seen under the microscope in parts of the specimen, which to the naked eye show no distinct pseudo-membrane, but only a

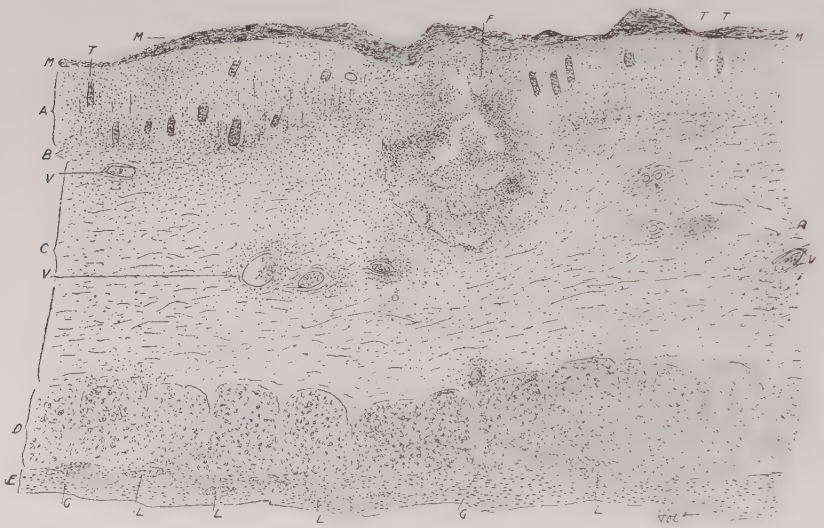


Fig. 39.—Membranous Inflammation of the Colon. The intestine is covered with a pseudo-membrane, *M*, which is composed chiefly of granular fibrin; the mucosa, *A*, is densely packed with round cells, and the tubular follicles have almost disappeared, traces only being left, at *T*, *T*. The submucosa, *C*, is greatly thickened, partly from cells, but chiefly from fibrin, which with a high power is seen to be everywhere in this coat, as well as the mucosa. Nests of cells are seen in the muscular coats at *L*, *L*. At *F* is a lymph nodule covered by pseudo-membrane, but breaking down at its center. *V*, *V*, are small blood-vessels with nests of cells about them. *History.*—Fourteen months old; ill nine days; temperature 101° to 105° F.; all stools containing blood. Lesions found throughout colon and in lower ileum. Intestine greatly thickened. Specimen is from ascending colon, where lesion was especially severe.

granular appearance. In rare cases a fibrinous exudation may be found upon the peritoneal covering of the intestine. The pseudo-membrane is made up of a fibrinous network containing small round cells, some red blood-cells, and numerous bacteria. The mucosa, and usually the submucosa, are densely infiltrated with small round cells, which in places may be so numerous as to efface the normal elements of the intestine. The tubular follicles are in some places quite destroyed, not a vestige of them remaining. In other places they are compressed and distorted by the accumulation of cells. The great thickening of the intestine is due

partly to the cell infiltration, partly to the fibrinous exudation, and partly to edema. All the blood-vessels, both in the mucosa and submucosa, are gorged with blood, and many small extravasations are seen. A necrotic process with the formation of deep ulcers we have never seen associated with membranous colitis.

Associated Lesions of Ileocolitis.—The most important one is bronchopneumonia. It is found in quite a large proportion of the protracted cases, and not infrequently it is the cause of death. There is no evidence that it is due to an infection from the intestine, although such a thing is possible in septicemic cases. Pulmonary tuberculosis is not infrequently met with in hospital cases, having no relation to the intestinal disease. Peritonitis is infrequent. We have met with it but once or twice, and then it was localized and of the plastic variety. Inflammations of the other serous membranes—pleurisy, pericarditis, and meningitis—are all very rare.

The renal lesions of ileocolitis have been the subject of considerable discussion, some observers holding that nephritis is a frequent complication of the severer forms of diarrhea, while others have held it to be rare. The lesions that we have usually found coincide with those described by others, and consist in marked degeneration of the epithelium of the tubes with but few glomerular or interstitial changes. Acute diffuse nephritis is a very infrequent though sometimes a most serious complication. The lesions mentioned as usually present are properly classed as acute degeneration rather than as inflammation of the kidney. Degenerative changes may be found also in the heart muscle, the liver, spleen, and even in the central nervous system.

Considerable attention has been given to a study of the blood in intestinal inflammations, to determine how frequently and in what circumstances a general blood infection (septicemia) from the intestines occurs. In the great majority of the cases studied under proper precautions the blood is sterile.

Symptoms.—(1) *Catarrhal Cases of Moderate Severity.*—The onset is usually sudden, often with vomiting, and for twelve, sometimes twenty-four hours the symptoms may be those of acute indigestion: vomiting, pain, fever, and frequent, thin, green or yellow stools, which are partly fecal and contain undigested food. Later the discharges contain blood and mucus, are often preceded by pain and accompanied by tenesmus. The stools are very frequent, often every half hour, and proportionately small, sometimes less than a tablespoonful being found upon the napkin after severe straining efforts. The mucus may be clear and jelly-like, or it may be mixed with fecal matter. Blood is seen in some cases in almost every stool, but rarely in clots, usually streaking the mucus. These stools are almost odorless. After a few days the blood usually

disappears, or is seen only as traces in an occasional stool; but mucus is still present in large quantities. The color of the discharges now becomes dark brown or brownish-green. Prolapsus ani is frequent, and may occur with nearly every stool. Abdominal pain is present, and is often quite intense just before the stool; frequently there is tenderness along the colon. For the first twenty-four hours the temperature is usually high, from 102° to 104° F. During the greater part of the attack it ranges from 99° to 102° F. There is considerable prostration; the loss in weight is usually marked and continuous; appetite is lost; the tongue is coated and the general appearance of the children indicates serious illness, although no really grave symptoms are present. Convalescence is always slow, and it may be months before the lost weight is regained.

In the milder cases the symptoms point to inflammation of the lower part of the colon only. The constitutional symptoms are not at all marked. The temperature may not be above 101° F.; the tongue may remain clean and the appetite good; the child may be bright and active, and hardly seem at all ill, and yet have from six to eight mucous and bloody stools a day.

The duration of the acute symptoms is usually two weeks, and yet in such cases, even though the child was previously in good condition and properly treated, recovery is slow. The first symptom of improvement is generally the disappearance of blood from the stools, which at the same time become less frequent, and the pain and tenesmus cease. Gradually the stools assume more of a fecal character, but mucus is likely to persist for two or three weeks; it may be seen in all stools, or only occasionally. In some cases both the mucus and blood disappear and the stools become thin, brown, or green, like those of an ordinary diarrhea. Relapses are readily excited, but cases such as have been described are rarely fatal except in delicate infants. This is the most common form of ileocolitis which terminates in recovery.

(2) *The Severe Catarrhal Form.*—This form of ileocolitis, like that just described, is usually primary. The symptoms closely resemble those of the membranous variety, and a diagnosis from it is in most of the cases quite impossible. The most rapidly fatal case we have seen lasted only three days, but the usual duration is from one to two weeks. The temperature is steadily high; the stools continue very frequent and generally contain blood; there is great prostration, dry tongue, sordes on the lips and teeth, and prominent nervous symptoms. Death usually occurs from exhaustion and profound sepsis while the acute symptoms are at their height. If the patient survives this stage, the case may drag on for four or five weeks with a temperature curve much like that of typhoid fever, and then terminate in recovery or in death from slow

asthenia, bronchopneumonia, or from an acute exacerbation of the intestinal symptoms. The autopsy in such cases usually reveals the presence of superficial ulcers. If recovery is to be the outcome, after the symptoms have been nearly stationary for a long time, there is seen a gradual improvement first in the general and then in the local conditions. Convalescence is very slow, often interrupted by relapses, and it may be months before the patient is quite well.

(3) *Follicular Ulceration—Ulcerative Inflammation of the Nodules.*—Follicular ulceration is often preceded by other forms of intestinal disease. It is much more frequently met with in infants over six months of age. The great majority of those affected are institutional children or those who are in poor condition at the time of the attack.

To understand the symptoms of these cases, it must be remembered

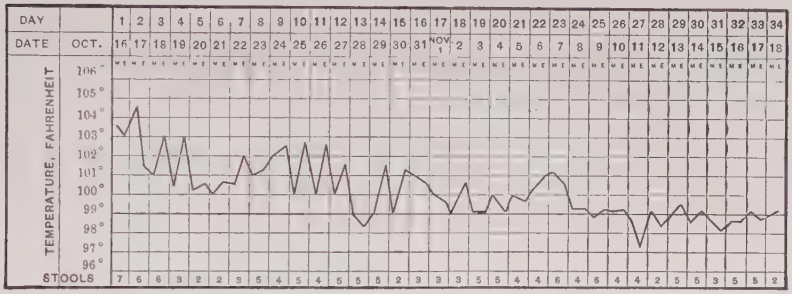


FIG. 40.—TEMPERATURE CHART OF ILEOCOLITIS, FATAL ON THIRTY-FOURTH DAY. Autopsy showed follicular ulcers throughout the colon.

that follicular ulceration is often a terminal process following other forms of diarrhea. It may be preceded by one or more acute attacks, or by a protracted subacute attack. On account of the feeble resistance of the child or the continuance of the exciting cause, the pathological process gradually extends to the lymph nodules of the intestine, chiefly the colon, which, as already described, pass successively through the stages of swelling, softening, and ulceration. The onset of the illness may therefore be abrupt, with vomiting and high fever; or gradual, without vomiting and with very little fever.

Vomiting is not a feature of these cases; but it is often present at the onset. Throughout the attack it is easily excited by injudicious feeding or medication. The temperature is seldom high, except at first; its usual range is from 99° to 101° F.; toward the close, even of fatal cases, it may be scarcely above the normal. The accompanying chart (Fig. 40) is a very good illustration of the course of the temperature in cases beginning abruptly and ending fatally.

The stools are seldom very frequent, the number being from four to eight a day. The most constant feature is the presence of mucus,

which is mixed with the stools and usually abundant. Blood is not generally present, and a large amount of blood is extremely rare. Large hemorrhages from ulcers we have never seen. The color of the stools is most frequently dark green or brown. Fluid stools are seen only during exacerbations. The odor is usually offensive, particularly in protracted cases. The microscope shows epithelial cells in great numbers, and very often an abundance of small round cells, which may be looked upon as the most constant sign of ulceration.

The failure in nutrition and steady loss in weight are very constant in these cases. As emaciation goes on, the skin hangs in loose folds on the thighs; it becomes dry and scaly and loses its elasticity, and occasionally small petechial spots are seen upon the abdomen. The skin over the buttocks becomes excoriated, and bed-sores form over the heels, the sacrum, or the occiput. The abdomen may be moderately distended, or it may be relaxed and soft. Tenderness is not usually present. The appetite is lost, and in most cases great difficulty is experienced in inducing children to take a proper amount of nourishment. Occasionally, when there is fever, fluids are taken eagerly. A returning appetite is always an encouraging sign. The mouth is often dry, the tongue coated, sometimes dry and brown; there may be sordes upon the lips and teeth. Superficial ulcers form upon the mucous membrane of the mouth, and often thrush is seen. The urine is usually diminished, high-colored, and loaded with urates. Albumin and casts are occasionally present. Rarely is nephritis severe enough to be a factor in the result. Tenesmus and prolapsus ani are uncommon.

The usual duration of the fatal cases is three or four weeks, but may be very much longer; their course is often marked by exacerbations and remissions. If recovery takes place, convalescence is always very slow and relapses are easily excited.

Very few of these cases recover completely. Even those who survive the primary illness are likely to suffer from intestinal symptoms for many months. Fatal relapses are often brought on by injudicious feeding when the children are apparently almost well. The general health is usually so undermined that the patients continue to suffer from all the symptoms of malnutrition, and ultimately succumb to an attack of some intercurrent acute disease.

The diagnosis of ulceration is to be made from the case as a whole rather than from any special symptoms. If a delicate infant, who has previously been prone to diarrheal attacks, has green mucous stools with low fever, and these symptoms continue with unabated severity for two or three weeks, ulceration is probable. If such symptoms continue for three or four weeks with steadily failing strength and loss of weight, the diagnosis is almost certain. If, on the contrary, after three or four days

of acute symptoms there is improvement in the stools and occasionally some which are quite fecal in character, even though it may be a week or more before the mucus disappears, we may be quite certain that no ulcers have formed.

(4) *The Membranous Form.*—This is the gravest form of inflammation of the intestines seen in children, and its symptoms are more often obscure than are those of any other variety. This is particularly true when it affects young infants. There may be at the onset and throughout the course of the disease severe local and constitutional symptoms; or with well-marked constitutional symptoms, the local symptoms may be slight or of very doubtful character, so that it is often mistaken for some other disease.

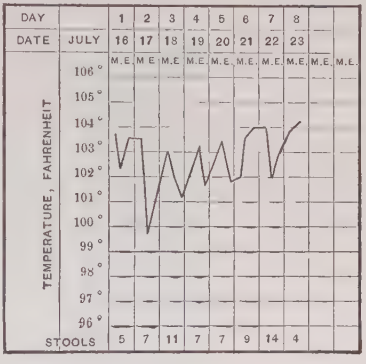


FIG. 41.—TEMPERATURE CHART OF MEMBRANOUS COLITIS; FATAL.

In the first form it closely resembles the most severe cases of catarrhal inflammation. The disease begins abruptly with vomiting, high temperature, and several large, fluid stools. The vomiting does not often continue after the first twenty-four hours. The temperature is at first from 102° to 105° F., and its course may be steadily high (Fig. 41), or remittent. The abdomen is often tender and sometimes swollen. There is severe pain, and at times tenesmus, with prolapse of the rectum. This is seen to be intensely congested, and sometimes shows patches of pseudomembrane upon its surface, thus establishing the diagnosis.

The stools often resemble those of the catarrhal variety, except that blood is more constantly present and usually more abundant, but the only positive point of difference is the presence of shreds or flakes of pseudomembrane. If the stools are thoroughly washed with water these may be seen as small gray opaque masses, which are then easily distinguished from the transparent mucus. Large shreds of membrane are seldom seen in children. Both blood and mucus sometimes disappear from the stools, which may consist only of dirty water. Under the microscope there may be seen epithelial cells, red blood-cells, and round cells in great numbers.

The presence of cerebral symptoms in these cases of membranous ileocolitis may lead to great obscurity in the diagnosis. This is most frequently true at the onset. There may be high temperature, great prostration, vomiting, stupor, delirium, and even convulsions; and such symptoms may for two or three days completely mask the intestinal con-

dition. As the case progresses, however, the intestinal symptoms come more and more into prominence, and the cerebral symptoms usually subside. But sometimes this is not the case.

Membranous colitis is also obscure when it affects young infants. The prominent symptoms are, rather high, continuous temperature, usually ranging between 101° and 104° F., but following no distinct curve (Fig. 42); wasting, which is not rapid but progressive; frequent stools, which have no constant or striking characteristics. They are usually thin, yellow or greenish in color, often containing no mucus or blood. Occasionally for a day the stools may be almost normal in appearance. In number they average five or six a day, but often for days only two or three. Outside of a hospital where autopsies are regularly

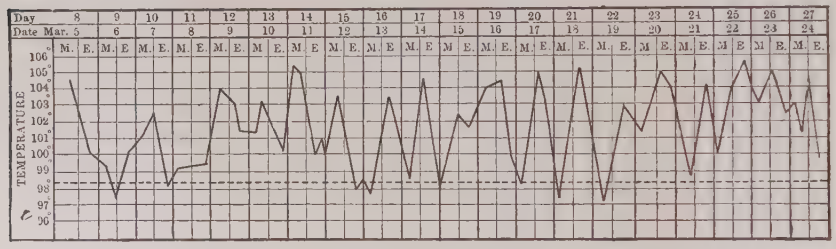


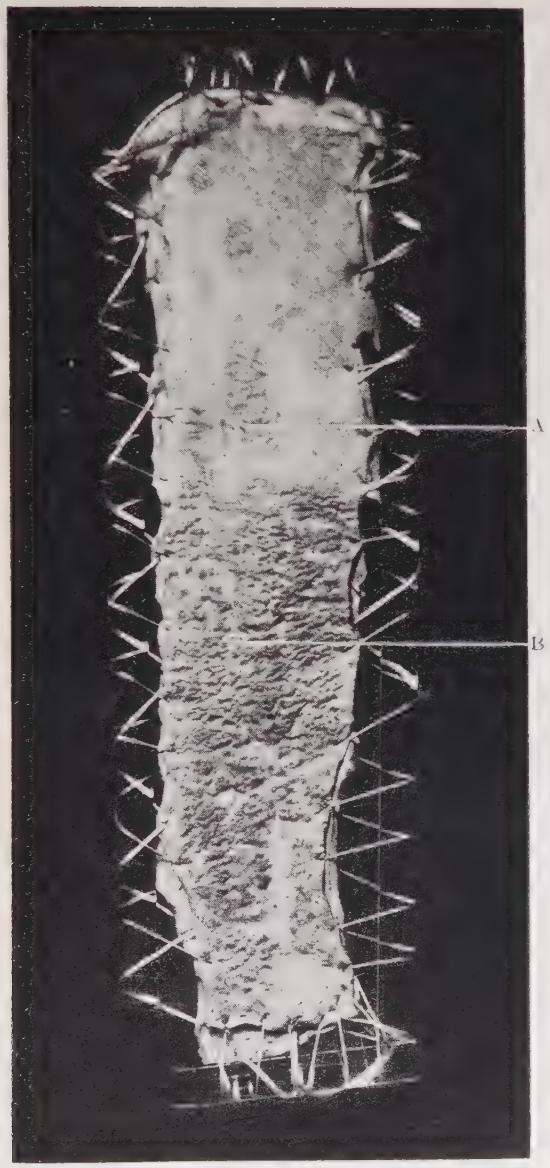
FIG. 42.—TEMPERATURE CHART OF MEMBRANOUS COLITIS. Infant fourteen months old, Babies' Hospital. Symptoms for the first two weeks obscure. Intestinal symptoms for the last two weeks only, never very severe; stools four to six daily, generally green, thin, with much mucus at times, and once or twice traces of blood. Autopsy: No lesion of importance except membranous colitis involving entire colon; a slight catarrhal enteritis.

made these cases are usually overlooked and considered as obscure pneumonia, tuberculosis, septicemia, typhoid, etc.

The duration of membranous ileocolitis is usually from one to three weeks. Death takes place from sepsis, exhaustion, or from complications. It is probable that almost every case of the severity described terminates fatally when it occurs in an infant. In older children the prognosis is much better as to life, but in them the acute attack may be followed by the chronic form of the disease.

Diagnosis.—Ileocolitis is to be distinguished chiefly from typhoid fever, intussusception, and meningitis. Typhoid is distinguished by the slower invasion, more constant temperature, enlargement of the spleen, tympanites, and most of all by the Widal reaction and the eruption. Acute colitis should not be confounded with intussusception; yet the records of intussusception show that a very large proportion of the cases were regarded in the beginning as cases of dysentery. In intussusception, although there is a sudden onset with acute pain, tenesmus, vomiting, and marked prostration, there is rarely fever. The later symptoms

PLATE VII



MEMBRANOUS INFLAMMATION OF THE ILEUM

A delicate child, eleven months old; mild diarrhea for two weeks without fever; acute severe symptoms for twelve days; temperature, 100° to 102.5° F.; green and mucous stools; no blood.

The lesions involved the last foot of ileum and entire colon. Specimen is from lower ileum, and shows the abrupt termination of the lesion; the upper part shows normal small intestine; A is a Peyer's patch; B is the inflamed part of the intestine; it has a rough granular appearance and is much thickened.

—absolute constipation, tumor, stercoraceous vomiting, and collapse—have nothing in common with colitis. The membranous form may be confounded with meningitis, and in some cases a differential diagnosis is impossible except by lumbar puncture. Marked diarrhea, even though the stools are not characteristic, should always make one doubt meningitis.

A diagnosis between the different varieties of ileocolitis is not always possible. Follicular ulceration is distinguished by its lower temperature, rather subacute course, infrequency of blood in the stools, and by the fact that it is usually preceded by diarrheal attacks which are often prolonged.

In the catarrhal form, the symptoms of an acute inflammation of the colon are usually manifest from the outset—bloody stools, pain, tenderness, tenesmus, and fever. In the membranous variety such symptoms are sometimes seen; but, as a rule, the local symptoms are less pronounced, while the constitutional symptoms, especially those relating to the nervous system, are usually marked. The course is usually shorter and more intense than in the other forms.

An agglutination reaction of the *B. dysenteriae* with the serum of affected children is usually present. But for general use in diagnosis this is not of great assistance. It is subject to considerable variation. Moreover, it is seldom present until the end of the first week of the disease, by which time the nature of the attack is evident by clinical symptoms. Agglutination in the higher dilutions is seen only with the particular type of organism with which the infant is infected.

Prognosis.—The younger the patient the worse the outlook. The prognosis is rendered unfavorable by extreme summer heat and by prolonged previous attacks of intestinal disturbance. The outlook is worse in secondary than in primary cases. In a given case bad prognostic symptoms are: continuous high temperature, the persistence of much blood in the stools, and severe nervous symptoms. The prognosis is always worse in institutions than in private practice.

Prophylaxis.—What has been said in a previous chapter regarding the general prophylaxis of diarrheal disease, applies equally well to cases of ileocolitis.

Special emphasis should be placed upon the necessity of energetic early treatment of all the milder forms of diarrhea, and particularly the cases of acute intestinal indigestion, in order that the process may be arrested before serious anatomical changes have taken place. Equal stress should be laid upon the importance of prompt and intelligent treatment at the very beginning of the cases with a sudden onset.

Hygienic Treatment.—The general plan recommended in the previous chapter should be followed here. A change of air is desirable for

most cases as soon as the acute inflammatory symptoms have subsided. In the protracted cases which drag on a subacute course, this change will often do more than anything else. Plenty of fresh air is necessary in all cases. The indications for bathing are the same as in other cases of acute diarrhea. It is undesirable to crowd these patients in institutions, as they always do better when separated.

The diet during the acute stage should be the same as in other forms of acute diarrhea. In the protracted cases the diet presents great difficulties, as the children have little or no appetite, and soon come to refuse everything in the shape of food that is offered. In infancy, for the first day or two only, barley or rice water or weak tea should be given. As soon as the vomiting ceases protein milk may be given in the manner described in the previous chapter. Buttermilk may be used as a substitute if protein milk cannot be obtained, but is not so efficacious. Especially to be avoided, not only in the acute stage but during convalescence, are cream, all top-milk mixtures, and also the malted foods. Infants, when very ill, are much more likely to take too little than too much food. A careful record should be kept of the amount actually taken in each twenty-four hours. In no case should food be given oftener than every four hours, water and stimulants being allowed between the feedings. In older children the diet during the acute stage should be much the same as in infants, but to them junket from which the whey has been carefully strained may also be given with a spoon. At a later period, rare scraped beef, kumyss, buttermilk, skimmed milk, and zoolak will be found useful, and during convalescence, eggs, boiled milk, or milk gruel made with rice or barley. Special care should be given to the diet for a long time. For months after an acute attack the intestines are very easily deranged. Relapses are excited by changes in the temperature, by great fatigue or exhaustion, but most of all by improper feeding. Especially in older children should such articles as cream, corn, tomatoes, green vegetables, and all fruits be withheld for a long time.

Medicinal and Mechanical Treatment.—Cases, the early stage of which is marked by vomiting and thin diarrheal stools, are to be managed at the outset according to the plan outlined in the previous chapter, viz., purgation, irrigation of the colon, and stopping all food. Castor oil should be administered at the outset—one dram at six months, two drams at one year, and half an ounce at four years. The salines may be used as described in the previous chapter. If the stomach is at all irritable, calomel, one-fourth grain every half-hour for four doses, may be substituted. Opium is usually required on account of the pain, tenesmus, and great frequency of stools. The dose should be regulated by the severity of these symptoms. The deodorized tincture and paregoric

are, we think, preferable to other preparations. Repeated small doses are better than a single large dose. It is very important that opium should be withheld for at least twelve hours after the initial purgative.

As the pathological process is principally in the colon, and most severe in the lower half of the colon, it can often be much more effectively treated by injections than by drugs given by the mouth. Irrigation of the colon is one of our most valuable means of treatment in these cases. For general purposes a saline solution at 100° to 104° F. should be employed. One or two quarts should be used for each irrigation. The solution should be injected high into the colon through a rectal tube, and early in the disease repeated at least twice a day. When the tenesmus is very great and blood abundant, small injections of either hot water (106° to 110° F.) or ice water may be used, and later astringent injections.

The most useful astringent is tannic acid of which one dram may be added to a pint of hot water. Whether injections are to be used regularly or not will depend much upon the patient. If they are well borne, they may be given once or twice a day during the attack; but if at every attempt to give them the child struggles, screams, and resists, they may do more harm than good. Complete rest is a very important part of the treatment.

For cases not influenced by the measures mentioned, or those not seen at the outset, bismuth should be tried, but it is of no use whatever unless large doses are administered. From two to four drams of the subcarbonate should be given in twenty-four hours to a child two years old, and proportionate doses to older children. This may be suspended in mucilage. Tenesmus and pain are sometimes relieved by the injection of three or four ounces of a starch solution to which from five to ten drops of laudanum are added. Severe tenesmus, when not controlled thus, and when associated with prolapsus ani, is sometimes immediately relieved by a suppository containing cocain. Not more than one-fourth grain should be used for a child of three years.

Although a serum has been produced which protects animals against inoculation with the *B. dysenteriae* its use in the treatment of the various forms of ileocolitis in children has not been followed by any very striking benefit.

Alcoholic stimulants are needed in many cases. They are indicated by a weak pulse, cold extremities, and great general prostration, no matter at what stage in the disease these symptoms are seen. Brandy is usually to be preferred. Generally not more than fifteen or twenty drops every three hours should be given to an infant of one year. Brandy should always be well diluted.

In cases where symptoms have lasted two or three weeks, and the

active ones have subsided, when the temperature is scarcely above 100° F., and the stools reduced to four or five a day, it is wise to stop all medication and attend only to the feeding, with irrigation of the colon every two or three days. One is often surprised at this stage to find that patients do better without drugs than with them. The prevailing tendency is to overdose cases of this type. No greater mistake is made than to give these children week after week the various diarrhea mixtures, with the expectation that ultimately the formula will be found which exactly meets the requirements of the particular case. The essential and important part of the treatment consists in injections, careful feeding and change of air. Astringent enemata, however, are of some value; they should not be given continuously but from time to time should be omitted for several days. Cases are not infrequently seen where the constant use of such injections is an important factor in keeping up the production of mucus. The colon should first be washed with a large amount of a tepid salt solution and then four or five ounces of the astringent solution injected and held in place by compressing the buttocks for half an hour. The patient should be placed in the best possible surroundings; in no disease is a change of air more to be desired than in this. They should be in the open air as much as possible but should be kept warm for their temperatures quickly fall to subnormal. The dangers of relapses and acute exacerbations continue long after the primary attack has subsided.

AMEBIC COLITIS

Amebic colitis is rare in children in this country; it is particularly so in infants, probably owing to the fact that nearly all the water taken at this age is boiled. Most of the cases in children thus far reported have been observed in warm climates, although Amberg has recorded five which occurred in Baltimore, the youngest child being two years and eight months old.

The symptoms in the few cases that have been reported in children have differed in no important particular from the disease as seen in adults. In exceptional cases the onset may be abrupt and the attack may run an acute course, terminating fatally in two to three weeks. Such cases are characterized by much abdominal pain and tenderness, frequent mucous and bloody stools containing amebae, and some fever, which, however, seldom reaches 102° F.

More frequently this acute onset is followed by a subacute or chronic form of the disease, or the disease may be subacute from the beginning. The protracted cases are those most frequently seen. They are very

obstinate to treatment. Periods of constipation and apparent recovery often alternate with exacerbations in which the bloody and mucous stools return, with pain, tenesmus, and slight fever. The duration may be from a few months to one or two years. Death may finally occur from exhaustion with extreme wasting, or from some complication, such as hemorrhage, abscesses of the liver being very rare in children. The diagnosis from other forms of colitis is made only by the discovery of pathogenic amebae in a freshly voided stool.

The general treatment is the same as for other forms of acute or subacute colitis. The special treatment for the purpose of destroying the amebae locally is the use of injections of quinin which may be employed in solutions varying in strength from 1 to 5,000 to 1 to 250. Recently subcutaneous injections of emetin hydrochlorid have been used for amebic colitis with very favorable results. Emetin should be given in doses of gr. 1/12 to gr. 1/4 depending upon the age of the child. The dose should be repeated two or three times at intervals of a day or more. The drug is a very powerful one and is to be used with caution.

AMYLOID DEGENERATION OF THE INTESTINES

This is rarely met with in infants. It is not so infrequent in older children, where it is associated with amyloid changes in the liver, spleen, and kidneys, usually as a result of prolonged suppuration in connection with bone tuberculosis. It is sometimes met with in syphilis. The ileum is the part of the intestine most affected. The process begins in the walls of the arterioles and capillaries, particularly of the villi, and later involves the vessels of the submucosa; subsequently the epithelium may be affected. The mucous membrane in these cases is pale, somewhat translucent. The condition is recognized by the application of the iodin test; the affected villi become of a brownish-red or mahogany color.

Amyloid degeneration produces no definite symptoms. Diarrhea is frequent but by no means constant. The anemia and waxy cachexia which are present are probably dependent much more upon the associated lesions of the liver and kidneys than upon the changes in the intestines.

TUBERCULOSIS OF THE INTESTINES AND MESENTERIC LYMPH NODES (MESENTERIC GLANDS)

These two conditions are usually, but not invariably, associated, and may be conveniently considered together.

Frequency.—In a series of 386 autopsies upon tuberculous cases from

our hospital records, the intestines were involved in 40 per cent. The great majority of the patients were under three years of age. In 131 autopsies upon tuberculous cases published in the Pendlebury Hospital Reports, the intestines were involved in 50 per cent. These patients were mainly between four and fourteen years old. In 209 autopsies upon tuberculous children, chiefly infants, reported by Müller, the intestines were involved in 28 per cent. In 1,346 autopsies collected by Biedert there were intestinal lesions in 31.6 per cent. Intestinal tuberculosis is most common from the third to the eighth year. The mesenteric lymph nodes are more frequently involved than are the intestines, though the two are usually associated. They were tuberculous in 59 per cent of the Pendlebury cases; and in 178 recent autopsies at the Babies' Hospital upon tuberculous patients, published by Bartlett and Wollstein, these nodes were involved in 63 per cent; in 10 per cent they were apparently the oldest tuberculous lesions.

Etiology.—While it is no doubt possible for infection of the mesenteric nodes to occur through the general circulation, this is exceptional. In the great majority of cases infection takes place from the intestines; i. e., these are examples of tuberculosis by ingestion rather than by inhalation. The bacilli in the intestinal tract may be derived from food or from sputum which has been coughed up and swallowed. Of 96 cases of abdominal tuberculosis of all varieties in children under sixteen years, studied by Park and Krumwiede, the infection was of the bovine type in 52, and the human type in 44 cases. Of these children, 71 were under five years and 25 between five and sixteen years. The proportion of bovine infections was slightly larger in the younger group. Primary intestinal tuberculosis in this country is relatively infrequent. When it does occur, however, it is more often due to a bacillus of the bovine than of the human type. The inference is probably justified that in cases of bovine infection, tuberculous milk was the source of the infection. The intestinal lesions most often found in infants and young children are mild in character and are usually associated with and secondary to an advanced pulmonary lesion. They are doubtless due to swallowing tuberculous sputum. In such cases the human type of bacillus is found.

Lesions.—*Intestines.*—The usual seat is the small intestine, chiefly the jejunum and lower ileum. With extensive disease the large intestine may also be involved, most frequently the cecum, and exceptionally it alone may be affected. Tuberculous ulcers may be found in the appendix.

The early deposits appear as tiny yellow nodules, not numerous but widely scattered and generally affecting Peyer's patches. Usually, however, ulcers are present, and often only ulcers are seen. Their size and number vary greatly; there may be only five or six tiny ulcers, or there

may be forty or fifty, the largest being two or three inches in diameter. They very frequently involve Peyer's patches. The typical tuberculous ulcer is of irregular shape, with rounded borders and with its longest diameter at right angles to the intestinal axis. When large, it may nearly encircle the gut. The ulcers are excavated; they have overhanging, infiltrated edges of a deep-red color. The surface is covered with granulations. In those which have partially healed a distinct puckering of the intestine occurs, which is especially noticeable upon the peritoneal surface. The small ulcers involve the mucosa only; the larger and older ones the submucosa and the muscular coats, and not infrequently also the serous coat. Perforation may occur, but rarely into the general peritoneal cavity, as a localized plastic inflammation precedes it. There may be adhesions of adjacent intestinal coils, and fistulae may form, owing to ulceration at the point of contact. With these severe cases there is always associated more or less extensive tuberculous peritonitis, frequently of the ulcerative variety. Like other tuberculous processes, the infiltration and ulceration may cease at any stage, and cicatrization follow. If the ulcers have been large ones, there is always some narrowing of the lumen of the intestine. Stricture is rarely seen because most of the children die from the general disease before it has had time to occur. Monti has reported a case of obstruction at the ileocecal valve, due to an old tuberculous cicatrix, in an infant of twenty-one months. One has come under our observation in a child of nine years, in which the obstruction was in the colon, just beyond the ileocecal valve.

Mesenteric Lymph Nodes.—Usually these tuberculous lymph nodes are from half an inch to an inch in diameter; occasionally they may reach the size of a hen's egg. From a fusion of several of them, tumors of considerable size may be formed. We have seen one such mass as large as the head of a child at birth.

The process is the same as that which occurs in other lymph nodes of the body. There is a tuberculous inflammation, followed by caseation, softening and abscess, or by calcification. Localized peritonitis is found in all the marked cases; this is usually plastic, but may be suppurative when due to the rupture of an abscess. Pressure upon the vena cava may lead to dropsy in the lower extremities. Ollivier has reported a case in which thrombosis of the vena cava occurred. Pressure upon the portal vein may lead to ascites and dilatation of the superficial abdominal veins. There may be pressure upon the thoracic duct.

Symptoms.—The symptoms of intestinal tuberculosis are exceedingly irregular. Ulcers are very frequently found at autopsy when there have been no marked intestinal symptoms; this is especially true of the small ulcers usually seen in infants. On the other hand, diarrhea is not uncommon in cases of advanced general tuberculosis where no ulcers are

present. It is the most frequent symptom of ulceration, and may be exceedingly obstinate. The stools do not differ essentially from those in protracted cases of ileocolitis except in the occurrence of hemorrhages and in the presence of tubercle bacilli. Hemorrhages are not very frequent, but they may be so large as to be the cause of death. This occurred in one of our cases, an infant nine months old, the blood coming from a single ulcer in the ileum. Hemorrhage is more common in older children. In some cases localized abdominal pain or tenderness is present. In advanced cases the symptoms of intestinal ulceration are usually mingled with those of peritonitis, and there are also present the enlarged mesenteric lymph nodes, which may aid in the diagnosis. In the majority of cases, these nodes are recognized only by deep palpation. A rectal examination may give additional information. The tumors are generally felt as irregular nodular masses, lying close against the spine, not movable, and sometimes tender on pressure. Other tumors from deposits in the peritoneum may be present anywhere in the abdomen; they may be superficial or deep. The other symptoms are due to the complications already mentioned and to tuberculosis elsewhere.

Diagnosis.—The only positive evidence of intestinal tuberculosis is the discovery of the bacilli in the stools. They are here to be carefully differentiated from smegma and other forms of acid-fast bacilli. In the absence of such evidence, the disease is differentiated from simple ileocolitis, first, by the signs of tuberculosis elsewhere in the body, especially in the lungs, these being almost invariably involved; secondly, by the slow onset and gradual development of the symptoms, while in ileocolitis an acute attack has almost invariably preceded. Large hemorrhages should suggest tuberculosis. A positive reaction to the tuberculin skin test is of much assistance in diagnosis, as is also the presence of palpable mesenteric glands.

Prognosis.—This depends altogether upon the extent of the tuberculous disease elsewhere, as it is extremely rare for the intestinal lesion to be the cause of death. Once formed, the ulcers probably remain, cicatrization being very rare, and then only partial.

Treatment.—The only symptom which ordinarily demands treatment is the diarrhea. When severe, this is to be managed much as in cases of ileocolitis, except that irrigation of the colon is, of course, not called for. The chief reliance must be upon diet. Bismuth and opium may diminish the peristalsis somewhat. No drugs can affect the process.

CHAPTER VIII

DISEASES OF THE INTESTINES—(Continued)

CHRONIC INTESTINAL INDIGESTION

The diagnosis of chronic intestinal indigestion is frequently made when it is not the digestion of the child but the character of the food which is at fault. The term should be reserved for those cases in which, with proper feeding, there are marked and persistent evidences of disturbance in intestinal digestion, usually with great retardation in physical development.

Chronic intestinal indigestion is especially common in children from the first to the fifth year.[1] It is seldom seen after that time. In a small proportion of cases it is apparently the result of a constitutional weakness. Nursing infants or infants who have been artificially fed during the first few months in a manner that cannot be criticized and who have thrived fairly well may, when the change to solid food is made, be quite unable to digest this or may even gradually manifest an inability to digest and thrive upon cow's milk however modified.

Some cases are clearly the result of improper feeding. With bottle-fed infants this is usually the giving of too great proportions of fat. With children taking solid food the trouble usually arises from giving this too early or in too large quantities, especially when the food has been improperly cooked, such as cereals, vegetables, and especially potato. But the most frequent cause of the condition is a previous severe or prolonged attack of diarrhea or dysentery from which the child seems never to have entirely recovered. Those who have previously been delicate or who have had prolonged digestive disturbance before the acute attack are particularly liable to be affected. The condition is seen in all grades of society but more commonly in the middle or upper classes, for among the very poor indiscretions in diet are likely to precipitate attacks of acute indigestion which may be fatal.

There are no characteristic pathological changes other than a dilatation of the small and large intestine, chiefly the latter. In some cases this may be extreme. Children who are the subjects of chronic intestinal indigestion seldom die from the condition itself, but usually from some acute process engrafted upon it, chiefly of the lungs or gastro-intestinal tract. There are then found only the lesions of the terminal infection or condition.

[1] Prolonged disturbances in intestinal digestion during the first year have been considered under Difficult Feeding Cases.

Symptoms.—The clinical picture which these cases present is a very common one, and the symptoms are quite uniform. The patients are generally very thin, with small extremities, a small amount of subcutaneous fat, and a large protuberant abdomen (Fig. 43). The size of the abdomen is perhaps the most striking feature of the condition. This is partly due to dilatation of the small intestine, but chiefly to dilatation of the colon which is regularly present in this condition. It occurs partly as the result of an excessive fermentation of food and partly from the relaxed condition of the muscular coats of the bowel. There is no hypertrophy and no ulceration. Dilatation of the intestine is further favored by a similar condition of the muscular walls of the abdomen which in marked cases become extremely attenuated, almost transparent. This relaxation is to be attributed partly to the poor nutrition and partly to the constant pressure from within.

FIG. 43.—CHRONIC INTESTINAL INDIGESTION.—Patient four years old; symptoms of three years' duration, following attack of acute ileocolitis. Height, 34 inches; circumference of abdomen, 22¾ inches; weight, 24 pounds.

The colon is often dilated to a diameter of three or four inches, as shown by X-ray examination, and sometimes even more than this. An erroneous diagnosis of Hirschsprung's disease is often made in such cases. The circumference of the abdomen may be several inches greater than that of the chest. Tympanites is constantly present although much gas may be passed per rectum. There is a marked tendency for the tympanites to increase during the day time and to diminish at night so that the variation in the circumference of the abdomen is usually two or three inches and sometimes as much as four or five inches in twenty-four hours. This variation is of assistance in differentiating the condition from tuberculous peritonitis with which it is frequently confounded. Such children are pale, anemic, sallow in complexion and haggard looking; they have dark rings under the eyes; they are fatigued on slight exertion; they are very cross, irritable, and emotional to an unnatural degree. They are hard to amuse, hard to control, and altogether exceedingly difficult patients to deal with. Their growth is retarded if the symptoms have lasted long. They are much below the average in height and weight, but mentally often quite precocious. One of our patients at three years weighed twelve and a half pounds and was twenty-nine

inches tall and another patient at five years weighed twenty-two pounds and was thirty-three inches tall. The sleep is always unnatural and disturbed; and at night the children toss about their cribs, waking frequently, crying out and often grinding their teeth. They perspire very readily, and suffer from cold extremities.

The bowels alternate between constipation and diarrhea, the former being more frequently present. At such times the stools are generally of a light gray color or nearly white. The odor of the stools is usually extremely foul. With diarrhea the stools are often not very frequent, not exceeding four or five a day, but they are large, gray, green, or brown in color, acid in reaction, often frothy, offensive, and always contain undigested food. A stool in many cases is immediately excited by the taking of food. From time to time, in many patients, large quantities of mucus are passed; in some cases this comes to be a constant feature of the disease. A striking feature is the large size of the stools in proportion to the amount of food taken. The chemical examination of these stools when cow's milk is taken, shows that the chief solid constituent is fat which frequently forms as much as 60 to 70 per cent of the dried matter of the stool, as compared with the normal of 20 to 40 per cent. The carbohydrates which are taken are largely broken down by the excessive fermentation which takes place in the intestinal tract. Large quantities of gas are expelled. Pain is not a very common symptom, but discomfort from the great tympanites is frequent. The appetite is capricious and usually poor, though some patients have a voracious appetite and will eat everything offered. The tongue is usually clean and the breath is not offensive unless the stomach is also affected, when the tongue may be coated.

The nervous symptoms which these patients present are exceedingly varied, and often of the most puzzling character. In some cases there are from time to time attacks in which they are so severe and so persistent as to lead to the diagnosis of organic disease of the brain. In addition to the condition of general nervous irritability, there may be tetany, fainting attacks resembling somewhat the seizures of *petit mal,* exaggerated reflexes, attacks of dulness or sometimes stupor, with irregular pulse and respiration and other symptoms strongly suggestive of tuberculous meningitis. Convulsions are not uncommon. They are usually accompanied by fever, and may be repeated at intervals of a few minutes. There is almost no end to the combinations of nervous symptoms which these patients may present. The skin shows frequently eruptions of erythema or of urticaria.

Most of these cases are without fever; but in some a slight fever is present for weeks at a time, the temperature usually varying between 99° and 101.5° F. Occasionally it may rise to 102° or 103° F. during

an acute exacerbation in the symptoms. The urine of most of these patients contains a great excess of indican and the amount present often fluctuates regularly with the nervous symptoms. The weight may remain stationary or there may be a gradual loss for some time. When improvement takes place the gain is apt to be rapid but very irregular. Great fluctuations in weight are characteristic of this condition and are to be explained by retention and loss of water. Attacks of general edema with rapid gain in weight are occasionally seen. Intercurrent attacks of acute indigestion, with diarrhea and sometimes also vomiting, are frequent and easily excited. Occasionally there are seen attacks of intercurrent intestinal infection with the dysentery bacillus, or other organisms.

The course and duration of these symptoms are indefinite. The milder cases if recognized early and promptly treated often recover in a few months, though careful feeding must be continued for a long time to prevent relapses. The severe cases under the most favorable circumstances last many months and usually several years. In those which progress favorably, improvement is usually first seen in the digestive symptoms, next in the nervous symptoms and last of all in the weight. In the most severe forms, if untreated, the patients gradually waste until they die from exhaustion, or fall easy victims to any acute disease which they may happen to contract. There is but little tendency to spontaneous recovery.

Herter has called attention to a type of this disease associated with marked arrest in growth to which he gave the name Intestinal Infantilism. In several such cases studied he found a failure of retention of calcium and magnesium salts over a prolonged period of time. To this he ascribed the arrested development of the skeleton. Associated with this, there were present evidences of excessive intestinal putrefaction. The bacteriology of the condition he believed to be characteristic, viz., a preponderance of the *B. bifidus,* with great diminution or entire absence of the *B. coli.*

Prognosis.—This depends upon the duration of the symptoms, the general condition of the patient at the time treatment is begun, and upon how thoroughly it can be carried out. The symptoms, in the great majority of cases, have existed for several months at the time the case comes under observation. Generally, the greater the mistakes in feeding have been, and the greater the violation of hygienic and dietetic rules, the better the prognosis. A child who has developed chronic intestinal indigestion of a severe type, in spite of the fact that the hygienic surroundings were good, and when the dietetic errors were not flagrant, is not nearly so hopeful a subject for treatment as one whose hygienic surroundings have been poor and whose diet has been especially bad.

In cases like the latter, a removal of the causes and the institution of proper methods of treatment almost invariably result in immediate and striking improvement, unless the general vitality of the patient has been reduced to a very low point. In the other cases where the mistakes have been less marked and the condition is due more to constitutional than to local causes, the improvement is slower and less striking. Thus, as a rule, hospital patients improve more rapidly than those seen in private practice.

Treatment.—In no class of cases that the physician is called upon to treat are results more satisfactory than in many of those of chronic intestinal indigestion, when intelligent coöperation can be secured. But the reverse is also true and no cases are more unsatisfactory than these when intelligent coöperation cannot be secured. Treatment is very difficult at best; recovery is a very slow process and the periods of exacerbation of symptoms that occur with almost every case are exceedingly trying to anxious parents and relatives. If the parents themselves are lax in discipline, and are unable to control the child, an efficient trained nurse should be secured, into whose hands the exclusive management of the child should be placed. In any case it should be understood that the duration of the symptoms is likely to be from one to two years and may be much longer. The adoption of a consistent plan of treatment continuously carried out for a long period is indispensable to success.

The essential part of the treatment is diet and general management. It should be remembered that the condition is in most cases primarily one of fat indigestion and intolerance. To this there is soon added intolerance of carbohydrates and often the latter becomes the prominent feature. When there is intolerance of both carbohydrates and fats, it is apparent that there can be no gain in weight. The best that can be done with these patients is to keep them for a long time upon a diet made up almost entirely of protein food. On this one should be content if the weight remains stationary or if there is but a slight loss. As the digestive condition improves, fats or carbohydrates, according to the tolerance, can gradually be added to the diet—at first only in very small amounts. In most cases the conditions must be met empirically and many mistakes and consequent relapses are likely to occur.

At the outset the most important thing is to stop all starchy food for a considerable time, and put the patient upon a diet consisting only of rare beef, beef juice, junket without whey, buttermilk or protein milk. Skimmed milk is well borne by only a limited number. After some improvement has occurred carbohydrates may be added, but very gradually beginning with small quantities (not more than one tablespoonful

a day) of well-cooked cereal. The number of feedings should not be more than four a day during the second year, and three or four a day for children during the third and fourth years. These should always be at regular intervals, and nothing whatever given between meals. The meat should be rare scraped beefsteak or lamb chop; from one to three tablespoonfuls may be allowed once a day. The white of egg may be given early, and after a time, the whole of a hard-boiled egg very finely grated.

After improvement has been going on for two or three months, bread may be added, at first in small quantities and once a day. This should preferably be stale, cut thin and dried in the oven until it is crisp, and given without butter. Mutton, chicken, or beef broth, without vegetables, may be given occasionally in the place of one of the milk feedings. After this diet has been kept up for three or four months, if improvement continues, one of the green vegetables thoroughly cooked and strained may be added once a day. A striking feature of these cases is their marked intolerance for sweet cow's milk. This must be withheld for a long period. This restricted diet should be continued for at least a year or until all the symptoms have disappeared. Potato should be forbidden for a long time. A few of the patients can take olive oil when they cannot tolerate any other form of fat. This may be tried very carefully, beginning with one teaspoonful a day.

Intestinal irrigation is occasionally useful for brief periods in some cases in which there is much mucus passed; no astringents, but only a warm saline solution should be used. But it should not be forgotten that continued irrigation often keeps up the production of mucus, and also that the introduction of large amounts of water may increase the intestinal distention.

The constipation can sometimes be controlled by the diet alone; but in most cases drugs are needed also. As laxatives in this condition preparations of rhubarb, or cascara and the compound licorice powder are serviceable. On account of the great tendency to abdominal distention due to excessive fermentation and atony of the intestinal walls the bowels must be kept well emptied. Most patients do better when two stools a day are secured, the second if necessary by an enema, but the frequent use of large intestinal injections is to be avoided. Abdominal massage is of much benefit in most cases.

Drugs directed against the process of putrefaction are extremely unsatisfactory even in older children and are not to be recommended. Of little value also is the administration of the various digestive ferments. General tonics are sometimes useful during convalescence and apparently assist in the improvement of the general condition, but during acute exacerbations their use should be interdicted. Nux vomica

is the best combined with some mild preparation of iron. Cod-liver oil, particularly in the early stage, is badly borne.

Relapses are easily excited by indiscretions in diet, and parents should be impressed at the very beginning with the necessity of adhering rigidly to the diet prescribed for a long period. It very often happens that the improvement which is seen after one or two months of careful treatment is so marked as to lead the parents to the belief that a cure has been accomplished, so that they relax their vigilance and allow improper articles of food which are almost certain to induce a relapse. If the case is an aggravated one, and the symptoms of long standing, it is wise to tell parents at the outset that a year's treatment is the minimum in which anything permanent can be accomplished.

The general treatment of the patient must not be overlooked. Proper clothing, regular exercise in the open air, cool sleeping rooms, massage and, when the condition is such as to permit it, sponging every morning with cool water are all of very great importance. An elastic abdominal bandage giving moderate support not only adds to the comfort of these patients but to some degree prevents the excessive distention likely to occur on account of the loss of muscular tone in the abdominal walls. The improvement in the nervous symptoms of the patient is often one of the first things noticed. From an irritable, fretful, peevish child the patient is sometimes totally changed in disposition in a few weeks, so as to become quiet, affectionate, docile, and playful.

INTESTINAL COLIC

The term colic is applied to any severe paroxysmal pain occurring in the intestines. It may be due to many causes. The colic of lead and arsenic poisoning are both very rare in children; but colicky pains are present in appendicitis, intussusception, ileocolitis, and, in fact, in all the severe forms of intestinal inflammation. Colic may be due to swallowing certain substances, especially foreign bodies and the seeds of fruits; and in rare cases it may be excited by the presence of roundworms when they are numerous. In all the conditions mentioned, colic is only one of the symptoms, although it may be a very prominent one.

The peculiar colic of infancy is clearly caused by spasm of the muscular wall of the intestine. It is a heightened reflex from irritation of which we have many other illustrations at this period of life. The cause of the irritation is usually the presence of some undigested food in the intestine. Colic is therefore essentially a symptom of indigestion. Flatulence and colic are very often, but not always, associated. Colic is always increased by the coexistence of constipation, which in many

cases is its sole cause. Almost any of the elements of the food may give rise to colic.

Sugars and starches produce it by causing excessive fermentation and flatulence. Fats are less frequently at fault; but the presence of large unabsorbed masses in the intestine may be a sufficient cause of irritation. The actual pain in colic is partly from distention, but chiefly from muscular spasm. In some of the most severe cases of colic it is possible that the spasm may be accompanied by a slight transient intussusception. Colic may follow chilling the surface of the body. In these cases, also, muscular spasm appears to be the principal factor in causing the pain. The colicky period of infancy is chiefly the first three months; after this time the peculiar susceptibility gradually passes off.

Symptoms.—These are in most cases so typical as to be easily recognized. They are always more severe in delicate and highly nervous children. In the severe attacks there is contraction of the features, a loud paroxysmal cry, subsiding for a few moments and then beginning with renewed intensity, drawing up the lower extremities, and in male infants contraction of the scrotum. With these symptoms the abdomen is usually found tense and hard. With the expulsion of gas, the symptoms usually subside at once, and the child falls asleep. In the most severe attacks there may be considerable prostration, cold extremities, and perspiration. When the symptoms are less severe there is only continual fretfulness, and the child can not sleep. When colic is habitual there are very few hours in the twenty-four when the child seems to be entirely comfortable. In nursing infants there may at times be difficulty in distinguishing the cry of colic from that of hunger, as infants suffering from colic will usually take food eagerly, and this is often followed by temporary relief. In colic, however, the pain soon returns, and often is more severe than before. The cry of colic is usually violent and paroxysmal; that of hunger is apt to be prolonged and continuous, and is not accompanied by the other symptoms mentioned as indicating abdominal pain. In older children the less frequent causes of colic mentioned at the beginning of this article, especially appendicitis, should be borne in mind.

Treatment.—When colic is due to flatulence of the intestine, nothing given by the mouth has much effect in relieving the symptoms. Certainly food should not be given. The purpose of treatment during the attack is to assist the child to get rid of the gas; as this is usually in the colon, the most efficient means is by massage or enemata. At first an injection of four or five ounces of lukewarm water should be used. If this is not successful, two ounces of colder water with half a teaspoonful of glycerin may be tried. This rarely fails to start peristalsis and

expel the gas. In conjunction with these measures, dry heat should be applied to the abdomen by means of hot flannels or a hot-water bag, and the feet should be well warmed. The treatment between the attacks and the treatment of habitual colic should be directed toward the constipation and the indigestion, upon which they depend.

CHRONIC CONSTIPATION

Constipation may be said to exist whenever the stools are less frequent and firmer than normal. During the early months infants usually have two movements a day. Many, however, have only one; but if this is normal in character the child is not constipated. In other cases, although there are two and even three stools a day, they may all be small, dry, and hard, having all the characters of constipated stools, and the case should be treated accordingly.

Etiology.—The causes of chronic constipation are many and far-reaching. It may be due to a diminution in the secretion of the intestinal glands or of the liver. The movements are then hard, dry, very light-colored, and are associated with much flatulence and other signs of intestinal indigestion. Very often the principal factor in constipation is insufficient muscular contraction in the intestine. The fecal masses are then propelled so slowly and remain so long in the intestine that the fluid portion is absorbed, the residue becoming, in consequence, so dry and hard that it is difficult to expel. In other cases constipation is due to the fact that there is insufficient volume to the stools, as may be the case when the food leaves very little residue. Constipation may depend also upon local causes, as, for example, where an evacuation of the bowels is resisted on account of pain from fissure of the anus or from hemorrhoids. Although not the primary cause, this condition may be sufficient to keep up the constipation indefinitely. It may in rare cases be due to a congenital condition, such as narrowing or twisting of the large intestine at some point. Another rare cause seen especially in infancy is tonic spasm of the anal sphincter. The most important causes of constipation may be grouped under two heads: diet, and conditions giving rise to muscular atony.

Diet.—In breast-fed infants the trouble is usually low total solids in the milk. In those who are artificially fed it is most often because the sugar is too low, and sometimes because all the solids are too low, the stool lacking volume. In other cases the cause of constipation is indigestion, especially of fats, in still others the use of sterilized milk. During the second and third years the cause may be too much cow's milk, particularly that which has been boiled, or the use of an excessive amount

of starchy food. In older children the cause may be an excess of milk and starchy food and a lack of green vegetables, coarse cereals, meat, fruit, and water.

Muscular Atony.—The most common cause of muscular atony is habit; in a large number of cases lack of proper training is the principal etiological factor. If the inclination to have a stool is regularly disregarded it soon ceases to be felt. The ordinary irritation from fecal masses produces no response whatever. The longer such a condition continues the more obstinate does it become. This is an important factor in all cases. Another cause of muscular atony is rickets. In this disease the muscular walls of the intestine suffer like the muscles of the extremities, and become incapable of doing their work. Again, any form of malnutrition in which there is feeble muscular tone may cause or aggravate constipation. It is often seen as a sequel to acute attacks of diarrheal diseases, particularly when these have been prolonged. Want of sufficient muscular exercise is a frequent cause. There are many children who rarely suffer from constipation in summer when they have plenty of outdoor exercise, who very often do so in winter when such exercise is wanting. A loss of muscular tone is not an infrequent result of the prolonged and indiscriminate use of purgative drugs or enemata.

Symptoms.—In most children no symptoms are present except the local ones, the general health being excellent and the nutrition in no way disturbed. In some, however, there are symptoms of greater or less severity, depending somewhat upon the cause of the constipation. There may be simply flatulence and colicky pains, or the irritation of the hardened fecal masses may produce a slight catarrhal inflammation of the sigmoid flexure and the rectum, so that mucus and sometimes traces of blood may be passed with the stool. Hemorrhoids may develop even in infancy, and frequently the constant straining leads to the production of hernia. In many cases there are from time to time nervous symptoms resulting apparently from the absorption of various toxic materials from the intestine. There may be headache, dulness, fretfulness, disturbed sleep, and associated signs of intestinal indigestion. The urine often contains indican in excess, and there may be slight fever.

Diagnosis.—This includes the discovery of the cause and the principal seat of the constipation. To arrive at the former the most careful and thorough investigation should be made of the child's diet and habits. It is desirable to determine whether the seat of trouble is the rectum, the colon, or the small intestine. If a suppository is almost immediately followed by a normal stool, one may be sure that the rectum only is at fault, and that it needs but a little extra stimulus to make it do its work. This is common in infants who are too young to make any

voluntary efforts. In such cases there are no other symptoms present. In others, the white or gray stools, marked flatulence, offensive breath, and general irritability, leave no doubt of the fact that the trouble is due to indigestion.

Treatment.—The successful treatment of chronic constipation in children is accomplished only by a careful study and regulation of the child's routine. In treatment, training, habits, diet and exercise play the most important, and specific remedies the least important part. Cure of the constipated habit is always difficult, and in most cases treatment must be continued for a long time. The coöperation of an intelligent mother or nurse is absolutely indispensable. To establish the habit of regular stools should be the first step, for without it nothing can be done. This training should be begun in infancy. Even in young infants regular habits are formed without difficulty if the child is put upon the chamber or chair invariably at the same hour. When a local stimulus is required in addition, an oiled glass rod or a gluten suppository may for a time be inserted. An older child must be taught to heed the first impulse to evacuate the bowel. Regular habits can hardly be formed unless the same time each day is chosen for the movement. That to be preferred is soon after the morning meal, as taking food into the stomach starts a peristaltic wave which is continued throughout the intestine. This has been demonstrated by the X-ray to occur even in the colon. With older children breakfast should be early enough to allow ample time for this duty before the other engagements of the day; and nurses should be impressed with the importance of the early formation of proper habits on the part of their charges. It is a part of nursery discipline which should invariably be insisted upon. Stretching the sphincter under an anesthetic is sometimes of great benefit in infants, especially when tonic spasm is present.

Food.—With nursing infants who get good breast-milk constipation is not common. When the milk is low in solids, constipation is frequent. In feeding cow's milk, constipation is overcome by giving the proportions of sugar, protein and fat which are best suited to the infant. It is rather more apt to occur with infants when, on account of digestive symptoms, modifications of whole milk or skimmed milk are given instead of those from top-milk. But constipation is also seen at times when the fat is too high. The laxative effects of all sugars, but especially maltose, should be remembered (see Infant Feeding). With infants during the first year, chronic constipation may be largely prevented by proper milk modification.

During the second year children who suffer from constipation are usually benefited by reducing the amount of milk and giving more solid food. Especially valuable are the coarser cereals thoroughly cooked and

purées of green vegetables,—peas, string beans, spinach or asparagus tips. Meat broths and beef juice are somewhat laxative on account of their extractives and salts. Fruits are valuable in all these cases; but only the juices should be given until a child is about fifteen months old. That of cooked fruit or almost any fresh fruit may be employed. After fifteen to eighteen months pulpy fruits may be given, but only after thorough cooking and straining,—apples, prunes, peaches, plums and pears, in moderate quantities; but berries should be avoided. Raw fruits should seldom be given to children under three years old, and after that age in moderate quantities only.

For older children who are on a mixed diet the amount of starchy food should be moderate. Coarse cereals only should be given. Milk should be given rather sparingly; it is sometimes advisable to stop it altogether. All bread should be made from whole wheat or unbolted flour. Bran biscuits are also useful. Meat and broth may be allowed freely, also green vegetables and vegetable salads. All fruits allowed infants may be used, but in larger quantities, and in addition scraped raw apple. Of the dried fruits, dates, prunes and figs are permissible, but only after cooking. Fresh fruit is preferably given in the morning, oranges being especially useful when taken on rising. A caution is necessary in the use of fruits and coarse foods for constipated children. It often happens that constipation is only one of the symptoms of a chronic intestinal indigestion, and such foods as those mentioned, while they may cause the bowels to move, frequently aggravate the primary condition. They produce abdominal pain, flatulence, and the discharge of mucus in the stools. The administration of some mild laxative even over a considerable period is often much less objectionable.

The laxative effect of sugars may be utilized with older children, but they must be given with caution not to disturb digestion. Two or three teaspoonsfuls of honey may be given with the breakfast or supper. Molasses may be used upon bread or may be added to cooked foods.

Either hot or cold water, when taken an hour before breakfast, may be of considerable benefit to older children. The necessity of supplying sufficient fluids is apt to be overlooked, especially when milk is excluded from the diet. While a liberal amount of water is indispensable, there is no advantage in excessive water drinking. The sparkling waters, like Vichy or Apollinaris, are sometimes better than plain water.

Massage, when properly employed, is useful in conjunction with other measures, but rarely succeeds alone. It should be given for five or ten minutes after retiring and just before rising. A proper amount of general muscular exercise is necessary and should be made a part of the treatment in every case. Special exercises for the development of the abdominal muscles when faithfully carried out are of particular benefit.

Posture during the stool is of some importance; in certain cases a cure is effected simply by substituting a low seat on a nursery chair or closet for the high one previously used.

Suppositories.—In many cases, particularly in young infants who are not old enough to initiate the muscular effort, a slight stimulus to the rectum is all that is required. The cone of oiled paper has a great reputation in domestic practice and is not objectionable. It may be of assistance in establishing a proper habit. Soap suppositories produce a more marked irritation; although their immediate effect is quite satisfactory, they should not be continuously used. Glycerin suppositories are even more objectionable. For occasional use they are convenient, but their frequent use, especially in infants, is likely to cause too much irritation. Gluten suppositories produce less irritation and are consequently slower in their effect, but they have not the same disadvantages. Suppositories are useful only when the trouble is in the rectum.

Enemata.—Water enemata should not be used regularly for the relief of chronic constipation. For immediate relief they are often necessary. The injection of one or two drams of glycerin in a few ounces of water is one of the most efficient means of moving the bowels at our command. Cases of fecal impaction are rarely met with in children. They are to be managed as in adults, by repeated injections of soap and warm water or of ox-gall, and sometimes by mechanical removal. An injection of an ounce or two of sweet oil may facilitate the passage of very hard and dry stools, and a regular nightly repetition of this, or a somewhat larger amount, for several weeks will sometimes break up a constipated habit.

Medicinal Treatment.—This is the least important part of the management of chronic constipation. The most valuable laxatives are preparations of cascara, nux vomica, belladonna, hyoscyamus and phenolphthalein. Though in most obstinate cases they are necessary, they should be used as little as possible and the dose gradually diminished. With most drugs the prolonged use of small doses is better than the occasional use of large ones. Cascara may be used either in the form of the elixir (dose from one-half to one dram), or the fluid extract, from one to five drops. Rhubarb, either in the form of the syrup or the mixture of rhubarb and soda, may be given occasionally, but it is not adapted to continuous use. Of salines, magnesia and phosphate of soda are best for continuous use in infants. All the preparations of malt possess slight laxative properties, and are useful in conjunction with dietetic and other medicinal means; any of the extracts of malt may be employed. Olive oil is often of assistance in the treatment of the constipation both of infants and older children. To the former the usual dose is one teaspoonful three times a day; to the latter, two or three times this amount should

be given. Mineral oil (petrolatum liquidum) is a valuable remedy, but is applicable only to older children, to whom from half an ounce to one and a half ounces daily must be given. It should be administered on an empty stomach, or it is likely to disturb digestion. As it is not absorbed, its action is purely local. The latest investigations indicate that the Russian oil has no advantages over American products, provided the latter have been suitably refined. Agar-agar has a beneficial action by rendering the fecal mass softer and more easily expelled. It should usually be combined with some other laxative such as phenolphthalein, cascara or rhubarb. It should be broken up into fine fragments and may be mixed with the cereal, with thick soup or simply with water. The dose is two or four teaspoonfuls daily.

HYPERTROPHY AND DILATATION OF THE COLON

(*Hirschsprung's Disease*)

Hirschsprung's disease is characterized by a great increase in the diameter of the colon and in the thickness of its wall. It was originally believed to be an idiopathic condition for which no sufficient anatomical cause could be found. Hence it has been known as congenital or "idiopathic" dilatation of the colon. Within recent years, however, it has become increasingly clear that in the majority of cases there is an obstruction to the passage of the intestinal contents through the large intestine, although when the intestines are removed and laid open, no evidence of obstruction can be found. The dilatation and hypertrophy are greatest in the sigmoid, and in about one third of the cases, this alone is affected. In the majority of instances, however, all of the colon is involved; very rarely only the colon above the beginning of the sigmoid is affected. The degree which the dilatation and hypertrophy may reach is enormous. The colon may fill the greater part of the much dilated abdominal cavity. There may be pressure upon, with a certain amount of atrophy of, the rest of the abdominal contents and the capacity of the thorax may even be encroached upon, the diaphragm being displaced upward to a marked extent. The inspissated contents of the colon may be many pounds in weight. The hypertrophy is chiefly due to an increase in the circular muscular fibers of the affected portion of the large intestine. The mucous membrane may be normal or there may be large and oftentimes deep ulcers which usually do not extend beyond the muscular coat but may involve this and even lead to perforation of the intestines with the consequent lesions of peritonitis.

At operation and at autopsy, when attention is especially directed

to the obstruction, it is found that this is usually the result of an abnormally long sigmoid and mesosigmoid which allows the lower portion of the sigmoid flexure to fall forward and downward, thus producing an angulation at its junction with the rectum. With the formation of this angle, the tendency is for the obstruction to increase and as the result of the effort of the portion of the large intestine proximal to it to overcome this obstruction, hypertrophy and dilatation take place. This is the factor which, in a majority of the more recently studied cases, has evidently been the determining one. In a small number of instances, hypertrophy of the transverse striations of the rectum have been found sufficiently marked to cause some obstruction. Other causes, such as spasm of the intestine, deficient innervation and congenital dilatation and hypertrophy, have been used to explain the condition when no anatomical basis for it has been found but they lack any convincing proof.

The symptoms may appear soon after birth or may be delayed months or even years, depending upon the severity of the mechanical disturbance. The most striking symptom is the increase in the size of the abdomen; this may develop rapidly or slowly. The distention may reach an extraordinary extent, the abdomen being almost spherical. The greatest circumference is usually just above the navel. The distention is chiefly due to gas, although there may be a sufficient accumulation of fecal material to cause circumscribed dulness and marked resistance over the colon.

The constipation is more marked than is seen in any other condition. Days, even weeks may pass by without an evacuation from the bowels. The feces are then usually dry, dark brown or greenish and very foul. Occasionally mucus and blood are passed and in the late stages of the disease there may even be diarrhea, the result of ulceration. Marked peristaltic waves are almost always seen; they are usually in the lower part of the abdomen and on the right as well as on the left side. Pressure upon the abdomen is seldom painful and only to a slight extent unless some complication such as peritonitis is present. By rectal examination an obstruction to the finger is frequently encountered. This may be at times overcome and not infrequently a rectal tube may be passed beyond it. It is then frequently found that water may be injected, which is only expelled after a considerable length of time. The urine is usually normal except for the presence of indican in large amount.

Attacks of vomiting from time to time are not unusual, but in general the digestion is good. The condition may last for many years and may not be incompatible with normal growth. Very occasionally spontaneous recovery apparently occurs. Usually the condition becomes gradu-

ally worse, the nutrition fails, there may be attacks of diarrhea with fever, or death may be due to some intercurrent infection, frequently of the lungs. Perforative peritonitis is an occasional fatal complication.

The two conditions most likely to be confounded with Hirschsprung's disease are tuberculous peritonitis and chronic intestinal indigestion. Chronic intestinal indigestion is a relatively common condition. It occurs frequently as the result of some frank intestinal disease, usually between the first and second year. There are frequent attacks of diarrhea alternating with constipation which is never so marked as in Hirschsprung's disease. The distention is of the small and large intestine as well and is seldom accompanied by peristaltic waves which are never very marked. Chronic intestinal indigestion is seldom seen at the early age at which Hirschsprung's disease is often found and the general condition of the child is always bad, while with Hirschsprung's disease the general health may be excellent for a long time.

Tuberculous peritonitis is characterized by a more rapid onset, by the presence, oftentimes, of fluid in the abdominal cavity and of abdominal tumors, by evidence of tuberculosis elsewhere and by the presence of the von Pirquet reaction. Compared with the frequency of these two diseases, Hirschsprung's disease is a very rare condition.

The treatment of Hirschsprung's disease is palliative so long as the general health remains good and without evidence of increase in the distention. It consists in careful feeding, occasional enemata and by the attempt, which is sometimes successful, of overcoming the angulation of the intestine by preventing fecal retention. In case the symptoms become more severe and the general health undermined, it is evident that obstruction is becoming more marked and operative procedure should be considered. Many different operations have been suggested; the only one which can be successful is one that involves the entire removal of the obstruction wherever this may be. In the past the results have not been very satisfactory, but with increasing knowledge and experience, operative treatment has become more successful.

INTUSSUSCEPTION

Intussusception consists in the invagination of one portion of the intestine into another. It occurs most frequently in infancy, being at this age the most common cause of acute intestinal obstruction. The accident is not a common one, but the life of the patient generally depends upon its prompt recognition.

Varieties.—Usually the upper part of the intestine is invaginated into the lower, although the reverse is occasionally seen. Intussusceptions

may occur at any point in the intestinal tract. Those of the small intestine are called *enteric;* those of the colon, *colic;* and those occurring at the ileocecal valve, *ileocecal* (Fig. 44). Of 90 cases under ten years of age, in which the variety was determined by autopsy or operation, 75 were ileocecal, 9 colic, and 6 enteric. In the ileocecal form a few inches of the ileum pass through the ileocecal valve, and then invagination of the colon occurs. Cases in which the ileum passes through the

FIG. 44.—ILEOCECAL INTUSSUSCEPTION. A specimen removed from a child in the New York Infant Asylum.

valve, but without invagination of the colon, are sometimes classed separately as an *ileocolic variety.*

Intussusceptions of the dying, as they have been called, are met with in about eight per cent of all autopsies made upon infants; they are not often found in children over two years of age. They are descending, enteric, easily reducible, and multiple—usually from eight to twelve invaginations being present. They are more frequently in the jejunum than in the ileum. They usually involve but two or three inches of the intestine, but may include ten or twelve inches. They are found in autopsies upon patients dying of all varieties of disease, and are

probably produced in the death agony. Such intussusceptions are without symptoms, and are of no clinical importance.

Etiology.—Of 358 collected cases under ten years, the following are the ages reported: under four months, 28 cases; from four to six months, 113; seven to nine months, 71; ten to twelve months, 18; one to two years, 32; two to ten years, 96. Three-fourths of the cases which occur in childhood are, therefore, in the first two years, and one-half of them between the fourth and ninth months. The greater frequency in infancy is attributed to the thinness of the intestinal walls, the greater mobility of the cecum and ascending colon, and the presence of other intestinal derangements at this age.

Males are more often affected than females. Of 268 cases in which the sex was mentioned, there were 174 males and 94 females. For this fact there is no explanation. The exciting causes of an attack are extremely obscure. The great majority of cases occur in children who are apparently in perfect health. Some previous intestinal disorder was present in about three per cent of the cases we have collected—diarrhea, dysentery, colic, chronic indigestion, and constipation, all being mentioned. In four cases the intussusception was ascribed to injury of the abdomen.

Lesions.—Nothnagel's animal experiments have shown conclusively that intussusceptions are formed by the irregular action of the muscular walls of the intestine. They can be produced or released at will by varying the application of the electrical current. In the artificial intussusception there is first a contraction of a certain part of the intestine, and if this ceases abruptly the normal gut below this point turns upward and folds over upon the contracted portion, thus forming a minute intussusception (Fig. 45, A). When once begun, the intussusception increases solely at the expense of the external layer (Fig. 45, B). Thus,

Fig. 45, A.

Fig. 45, B.—Mechanism of Intussusception. (Treves.)

while the apex of the tumor D remains unchanged, the part of the sheath at A passes to B and then to C, so that the lower part of the intestine is drawn over the upper, rather than the upper crowded into the lower. The mechanism of the invagination was apparently the same when a part of the intestine was first paralyzed by crushing, as in the case in which a spasm of the intestine was first produced.

There is little doubt that pathological intussusceptions are produced

in the same way as in these experiments. As the invagination takes place, the mesentery is drawn in with the bowel, and always lies between the sheath and the inner layer. To allow intussusception to occur, the mesentery must be unduly long, stretched, or lacerated. Its attachment to the spine causes the intussusception to describe an arc of a circle, the concavity of which is always toward the spine. It also causes a puckering of the tumor. Invagination does not necessarily produce either obstruction or strangulation, but usually both are present, and are the chief causes of the symptoms. Traction upon the mesentery leads to obstruction in its vessels, causing congestion, edema, hemorrhages, and even gangrene. Obstruction is chiefly due to swelling. It may be due to dragging of the mesentery, which brings the apex of the tumor against the side of the gut, or to bending of the intussusception. Intussusception is usually of all the coats of the intestine. We have, however, seen one, the exact nature of which was determined by operation, in which only the mucosa and submucosa were involved. The invagination was at the ileocecal valve. The symptoms were characteristic except for the absence of tumor.

The great cause of irreducibility in the first two or three days is swelling. We have several times seen at autopsy or operation an intussusception easily reduced, except the last two or three inches of the cecum or ileum, which was swollen to the thickness of from a fourth to half an inch. Adhesions may prevent reduction, but rarely before the fourth day; they are often absent as late as the sixth or seventh day. They are usually between the internal and middle layers of the intussusceptum, and are due to local peritonitis. In chronic cases, however, they form the principal obstacle to reduction. Other causes of irreducibility are twisting of the tumor and pinching of the prolapsed intestine, especially the ileum by the ileocecal valve.

Gangrene and sloughing of the gangrenous portion of the intestine occur much more often in acute than in chronic cases. Portions of intestine were passed *per anum* in 24 of 362 cases under ten years, or about six per cent; but only two of these were in infants. Toward the end of the second week is the time when the separation of the sloughs is to be looked for. The amount of intestine discharged varies from a few inches to several feet. Two cases are on record in which the entire colon was passed, the patients recovering, but dying several months later from other causes. At the autopsies the ileum was found attached to the lower part of the rectum just above the anus. In acute cases gangrene occurs about the upper end of the tumor, and the intestine usually comes away in one large mass. In chronic cases shreds of intestine may be discharged for several weeks.

Symptoms.—The clinical picture of a case of intussusception is a

striking one, and when acute the symptoms are so uniform that, once seen, it can scarcely be overlooked a second time. The patient, usually between six and twelve months of age, is taken suddenly ill with severe pain and vomiting; the pain recurs paroxysmally every few minutes, and the vomiting is first of the contents of the stomach, and afterward bilious. There may be one or two loose fecal stools, then only blood or blood and mucus are passed without any admixture of feces. The general symptoms are those of great prostration, or even collapse—pallor, feeble pulse, apathy, and normal or subnormal temperature. The abdomen is relaxed. A tumor is usually present in the epigastrium or the left iliac fossa, or it may be felt *per rectum*. Later there is tympanites: the vomiting and pain continue; there is a steady increase in the prostration, and toward the end a rapidly rising temperature which may reach 105° or 106° F. before death occurs from collapse. If the symptoms continue longer the signs of peritonitis are added. In subacute cases the onset is less abrupt, and pain, vomiting, and constipation less constant and less severe; but the same symptoms are present. In chronic cases the onset is with vague, indefinite intestinal symptoms; pain, vomiting and bloody discharges are usually wanting; there is progressive wasting and more or less diarrhea, but only the presence of the tumor leads to the recognition of the condition.

Onset.—Of 193 cases under ten years in which data upon this point could be obtained, the onset was sudden in 181 and gradual in 12 cases. By far the most frequent symptoms of onset are pain and vomiting. In a smaller number of cases the initial symptom is diarrhea or a discharge of blood and mucus.

Pain.—This is rarely continuous, but is intermittent, recurring in paroxysms like those of ordinary colic, but of great severity. Few pains in infancy are to be compared with it. The child sometimes shrieks so as to be heard all over the house. Pain is a prominent symptom in over three-fourths of the cases, and is very rarely absent. It is generally more marked for the first two days, but may continue throughout the attack. In a few cases the pain is localized, being usually referred to the region of the umbilicus.

Vomiting is more marked at the onset, but may continue throughout the attack. Like the pain, it is more frequent in the acute cases. It is due to intestinal obstruction. Vomiting is present in fully four-fifths of all cases. Usually it is persistent and often projectile. If food is given, vomiting often occurs as soon as it reaches the stomach. Stercoraceous vomiting occurs in about fifteen per cent of the cases in children under ten years, but is not common in infancy. It is rarely present before the third or fourth day. Although a bad sign, it is not by any means a fatal one, as nearly one-half the cases in which it has been noted

have recovered; it is to be regarded as indicating complete intestinal obstruction rather than strangulation.

Tumor.—This is one of the most important symptoms for diagnosis because of its frequency and its peculiar character. It is present early in the disease, often in a few hours after the initial symptoms. The following table shows the frequency with which a tumor was present in the different varieties, and the position which it occupied in each. The anatomical variety was determined either by autopsy or operation.

The Relation between the Tumor and the Different Varieties of Intussusception in 188 Cases under Ten Years.

Seat of Tumor.	Seat of Intussusception.					
	Ileocecal.	Ileocolic.	Colic.	Enteric.	Not Stated.	Total.
Region of cecum	..	3	..	1	7	11
" " ascending colon	1	..	..	..	12	13
" " transverse colon	3	..	..	..	13	16
" " descending colon	3	..	..	..	18	21
" " sigmoid flexure	4	..	1	..	8	13
Rectum	25	1	7	..	28	61
Protruding from anus	9	..	1	..	12	22
Umbilical region	..	..	..	1	..	1
Movable	..	..	..	1	2	3
Site unknown	1	..	..	..	..	1
Total	46	4	9	3	100	162
No tumor felt	10	2	..	1	13	26

Tumor was thus made out during life in eighty-six per cent of the cases; and in the great majority of these it was discovered at the first careful examination.

It will be noted that in nearly half of the cases the tumor was either felt in the rectum or protruded from the anus, and that in over two-thirds it had advanced as far as the descending colon or beyond. The tumor may reach the rectum in a surprisingly short time, even when the invagination begins at the ileocecal valve. In one of our cases it was felt in the rectum in less than twelve hours from the onset. The usual description, "sausage-shaped," is accurate when the invagination is large, the tumor then being from four to six inches long and about an inch and a half in diameter. It is often curved.

During manipulation, or during an attack of pain, the tumor may become more prominent and may be distinctly erectile. To the touch the rectal tumor closely resembles the os uteri, the central opening being the apex of the intussusception. When protruding from the body, the tumor is rarely more than two inches long. It is usually of a deep-

purplish color, and may be gangrenous. It has been mistaken for prolapsus ani, polypus, and even hemorrhoids.

Condition of the Bowels.—Bloody stools are a very constant symptom. Of 186 cases under ten years in which the condition of the bowels was noted, blood in the stools was present in seventy-six per cent. There are very often two or three thin, diarrheal movements, and then only blood and mucus are passed with no trace of feces and with no fecal odor. The amount of blood varies from a quantity sufficient to stain the mucus, to an ounce of semi-fluid blood. It rarely occurs without some mucus. Such discharges frequently follow attacks of severe colicky pain, and may occur several times in an hour. They may continue, or after a day or two they may be succeeded by absolute stoppage. Diarrhea throughout the attack is rare in children, particularly so in infants. It belongs generally to chronic cases. Constipation is complete in most of the acute cases, neither gas nor feces being passed—a fact which the discharge of blood and mucus may lead one to overlook.

Tenesmus is very common if the tumor is rectal. Relaxation of the sphincter is met with in a considerable proportion of the cases when the tumor is in the sigmoid flexure, or rectum.

During the first twenty-four or forty-eight hours the abdominal walls are soft and relaxed, and may even be retracted. Usually there is then little resistance to abdominal palpation. After the second or third day there is usually tympanites; but this does not necessarily mean that peritonitis exists. Localized tenderness is a symptom of some importance when a tumor is absent. Scanty urine has been noted in a few cases, but is of no special value in showing the seat of obstruction.

In the acute cases the general symptoms are very striking. They are the ordinary ones of severe shock—marked prostration, pallor with an anxious expression of the face, general muscular relaxation, cold extremities, cold perspiration, and often a subnormal temperature. Early there is marked restlessness, and even convulsions may occur. Later there is apathy, dulness, even semi-stupor. The temperature during the first twenty-four hours is usually not elevated, and is frequently subnormal. Toward the close of the disease it rises rapidly to 103°, 104° F., or even higher, quite independently of peritonitis. A rapidly rising temperature is always a bad symptom, and usually betokens death within twenty-four hours. Wasting is seen in the chronic cases, and may be quite rapid.

Course, Duration, and Termination.—Of 198 cases under ten years, 155 were classed as acute, lasting less than seven days; 33 as subacute, lasting from one to four weeks; 10 were chronic, lasting over four weeks. Nearly all the cases occurring in infancy are acute.

Spontaneous reduction is, without doubt, possible in intussusception.

Treves and others are of the opinion that this happens much more frequently than is generally supposed, and that many cases of severe colic are really cases of slight intussusception. There are seen in both conditions the tendency to vomit, the paroxysmal pain, the constitutional depression, and often the sudden cessation of the symptoms, especially under the influence of opium; but to make a positive diagnosis of invagination in such cases is impossible. Intussusception may be cured spontaneously by sloughing of the invaginated part, the continuity of the intestine being preserved by adhesions. Such a result is rare at all ages, and is almost never seen in infancy.

The most frequent cause of death in acute cases is shock. Peritonitis is not found at autopsy or operation so often as might be expected. In fifty-eight autopsies, it was seen but twenty times, and in seven of these it was limited to the intussusception. In but seven cases was there perforation.

Diagnosis.—This usually presents no difficulty in acute cases provided the physician has the condition in mind. The great majority of such cases present nearly all the classical symptoms, viz., sudden onset, recurring colicky pains, frequent vomiting, bloody and mucous stools without fecal matter, general prostration or collapse, and low temperature. The records show that the most common error is to regard the case for the first few days as one of gastro-enteritis or ileocolitis, the physician's attention being engrossed by the vomiting and bloody stools. Given the other usual symptoms, the presence of the characteristic tumor is conclusive evidence of intussusception. Unless the patient is very much relaxed, a satisfactory examination is possible only under full anesthesia. In any case of acute intestinal obstruction in infants, intussusception should first be considered. We once saw in a young infant with strangulated hernia nearly every symptom of intussusception except the abdominal tumor; in another infant with an inflamed Meckel's diverticulum there was vomiting, bloody and mucous stools and an elongated tumor in the hypogastric region. Cases of chronic intussusception present no diagnostic symptoms except the tumor. In both acute and chronic cases the rectal examination is most important for diagnosis, and often settles the question at once.

Prognosis.—The prognosis of intussusception depends upon the age of the patient, upon the variety of the disease—whether acute, subacute, or chronic—and upon the time when proper treatment is begun.

There were collected by Pilz in 1870, 94 cases under one year, the mortality being 84 per cent. Of 135 cases of the same age reported between 1870 and 1891 the mortality was 59 per cent. Results in older children were somewhat more favorable. Formerly recovery was rare, except in cases with sloughing; but with earlier diagnosis and a better

understanding of the proper methods of treatment, the mortality has been very much reduced. Combining the figures of Pilz with our own, there are 362 cases with 231 deaths, or 63.5 per cent.

Gibson (New York) has collected reports of 187 operations for intussusception, with a general mortality of 51 per cent; in 126 cases, in which the tumor was reducible, it was but 36 per cent; in 61, in which it was irreducible or gangrenous, it was 80 per cent. The table following gives the mortality in relation to time of operation:

Time of Operation.	Mortality, Per cent.
First day	37
Second "	39
Third "	61
Fourth "	67
Fifth "	73
Sixth "	75

After the second day the chances of success are greatly reduced.

Treatment.—The diagnosis of acute intussusception once made, laparotomy should immediately be performed without an hour's unnecessary delay. The results following inflation of the intestine with air and injection with water are too uncertain to be depended upon.

Operation should be looked upon as a measure which, if employed reasonably early, offers a good prospect of success. All statistics show that the result depends more upon the time when the operation is done than upon any other single factor. With earlier diagnosis and more prompt resort to operation, the mortality from acute intussusception has, during the past fifteen years, been steadily failing. In chronic cases, also, laparotomy offers altogether the best chance of success.

CHAPTER IX

DISEASES OF THE INTESTINES.—(Continued)

APPENDICITIS

Appendicitis is met with at all ages, and is not especially a disease of children. When it attacks those over ten or twelve years of age it does not differ greatly from the types observed in adults. All that will be attempted in this chapter will be a consideration of the peculiarities

of the disease as it is seen in children, particularly young children. For a fuller discussion of the disease as a whole the reader is referred to works on general medicine and surgery.

Etiology.—Of 1,000 cases of appendicitis personally observed by McCosh, 85 occurred in children between the ages of ten and fifteen years; 51 between the ages of five and ten years, and only 17 under five years; of these but 4 were under two years. Churchman's figures from the Johns Hopkins' Hospital, in a total of 1,223 cases, give only 9 cases under five years, and 50 between five and ten years. In infancy and early childhood appendicitis is, therefore, a relatively rare disease. The youngest case that has come under our observation was in an infant of ten weeks. Operation was done and recovery followed. Appendicitis in young infants has been reported by Goyen (six weeks), Shaw (seven weeks), Demme (seven weeks) and Savage (nine weeks). The predominance of the male sex holds true even in childhood. Of 101 cases under fifteen years, 72 were males and 29 were females.

Regarding the exciting cause of an attack but little is yet definitely known. In only a very small proportion of the cases is a foreign body discovered in the appendix. In one of ours a pin was found, and a number of similar cases are on record. There is, however, often a fecal concretion which is moulded into the shape of a foreign body, and formerly was often regarded as such. This probably has some relation to the attack by causing disturbances of circulation and increasing the chances of infection. Still and others have called attention to the frequent occurrence of pin worms in the appendices of young children. There is abundant reason for believing that these may at times be the exciting cause of an attack. The bacteria most frequently found in abscesses from appendicitis are streptococci, usually associated with colon bacilli.

Lesions.—All the common varieties of acute appendicitis,—the catarrhal, suppurative, and gangrenous,—are met with in children; and, much less frequently, the chronic form. The lesions present few peculiarities in early life except that, owing, possibly, to the relation of the appendix to the omentum, perforative inflammations are less likely to be circumscribed by inflammatory products and much more likely to result in a general peritonitis than in adults. Whether or not this be the correct explanation, it is certainly true that general peritonitis is a much more common sequel than in adults. Another point of some importance is the fact that in early life the appendix is rather more frequently found out of the usual position. The inflammation excited by pin worms is usually a superficial one; perforation and abscess formation are almost unknown when they are the cause.

Symptoms.—In many of the cases the familiar symptoms of appen-

dicitis—vomiting, localized pain and tenderness, muscular rigidity, abdominal distention, and fever—are all present, and the diagnosis is easy. But in perhaps the larger number the disease is irregular in its onset, insidious in its course, and presents at times great difficulties in diagnosis. This is particularly true of appendicitis in children under five years. Vomiting is probably the most constant symptom; it is seldom absent, and usually persistent. If accompanied by pain and constipation, appendicitis should at once be thought of. Pain, though usually present, is often indefinite; it is generally hard to localize and difficult to interpret. It may be referred now to one and now to another part of the abdomen. Often the only evidence of pain is restlessness, irritability, and, in infants, frequent crying. Tenderness is even more difficult to elicit than pain. Young children, especially if nervous and sensitive, shrink from any touch, and the results of abdominal palpation may be most unreliable. In others of a different temperament positive information may be obtained. In any child under three years, it is almost impossible to make out localized tenderness. The same is true of muscular rigidity. Only with the greatest amount of tact and by diverting the patient, can any information be derived from this part of the examination. Tenderness and muscular rigidity are sometimes shown by the child's disinclination to move either the trunk or lower extremities and by evidences of pain when he is moved by mother or nurse. When associated with vomiting, fever, and constipation, such symptoms are always suggestive.

Constipation is usually present, but by no means so regularly as in adults. Diarrhea is not at all uncommon, and, when associated with vomiting, tends to divert attention from the appendix to an ordinary gastro-intestinal attack. Abdominal distention, when present, is of much importance, taken with other symptoms. Fever is rather more apt to be high than in adults. But there are many exceptions, and, on the whole, the temperature is a very untrustworthy guide either to diagnosis or prognosis. The leucocyte count is of much assistance in diagnosis, at least in suppurative forms of appendicitis. A leucocytosis of at least 10,000 to 20,000 is usually present, with a polymorphonuclear percentage over 75. Some special symptoms may be seen in appendicitis which are quite misleading. We have on several occasions seen frequent micturition and other marked manifestations of vesical irritation, owing to the position of the appendix behind the bladder. Pain just before and during defecation is occasionally a striking symptom especially with infants. The rigidity of the thigh flexors seen in cases of appendicitis, which comes on with subacute symptoms, may give rise to lameness strongly suggestive of disease at the hip.

Course of the Disease.—A certain number of cases begin with definite

symptoms—pain, vomiting, fever, and constipation—and continue with slowly or rapidly advancing symptoms to increasing prostration, continued vomiting, constipation, rapid pulse, abdominal distention, rigidity, higher temperature, and death by general peritonitis at the end of five or seven days' illness. Others, with a similar onset, show a gradual abatement of all acute symptoms after a few days, and recovery at the end of ten days or two weeks, followed, perhaps, by another attack after a few months. These types are seen in children as in adults. But others are quite common. A child may be taken ill, sometimes abruptly, sometimes more gradually, with vomiting, which is repeated several times in a single day, afterward only occasionally. There is some pain; it is not very definite and not localized. The prostration is only moderate, the temperature not over 100° or 100.5° F. The examination shows little. Tenderness can not be definitely made out; the child is irritable, fretful, wishes to be left alone, and resists all efforts at abdominal palpation. The bowels are constipated, or they may be at first loose and afterward constipated. The child does not seem very sick. The attack is probably regarded as an ordinary one of acute indigestion. But things do not improve as they ought. The pulse becomes more rapid, the prostration greater, and the child begins to look seriously ill, though the temperature has not risen. The abdominal distention is now considerable and tenderness undoubted. An operation is decided on, and there is found a gangrenous appendix and a diffuse general peritonitis. Sometimes the grave symptoms develop with great rapidity in the course of a few hours, when previous symptoms had all been mild; sometimes so insidiously that the transition is almost imperceptible.

Prognosis.—The prognosis in young children is not good; of 132 collected cases in infants and very young children the mortality was 38 per cent. But in those over seven years old the outlook is rather better than in adults. The results depend much upon early diagnosis and proper treatment. General peritonitis, it is generally agreed, occurs much oftener in children than in adults; it is the cause of death in about 80 per cent of the cases. Of 43 fatal cases, nearly all of them from general peritonitis, only 6 died during the first three days, 19 from the fourth to the seventh day, 13 in the second week, and 5 in the third week. If general peritonitis occurs, the chances of recovery after operation are, however, usually better with children than with adults.

Diagnosis.—The diagnostic symptoms of appendicitis are a sudden onset with vomiting, sharp pain in the abdomen, and persistent acute localized tenderness in the right iliac fossa. Rigidity of any or all of the abdominal muscles is also significant. Constipation is more frequent than diarrhea, though the latter is not rare. There is almost invariably some elevation of temperature, but not often high fever.

Appendicitis may be confounded with colic, indigestion, and in infants with intussusception; in older children with abscesses due to psoitis. Colic is distinguished by the absence of localized tenderness and fever, by its short duration, and by the fact that the pain is generally less intense. Severe colic with fever in children over three years old should, however, always be regarded with suspicion. From acute indigestion the diagnosis of appendicitis is difficult at the onset, and it may be impossible for twenty-four hours. However, the pain of indigestion is rarely so severe, while the fever is usually higher. It should be remembered that the pain in appendicitis is not always localized, nor is the tumor always in the right iliac fossa. The presence of pain, vomiting, and localized tenderness, and the greater severity of the constitutional symptoms, indicate appendicitis. We have several times known the pleurisy accompanying pneumonia at the right base to be mistaken for appendicitis. With this there may be vomiting, severe localized pain, and sometimes also localized tenderness. Cyclic vomiting is distinguished by the history of previous attacks, the greater frequency with which the vomiting occurs, its abrupt cessation after twenty-four to seventy-two hours, the sunken abdomen, and the absence of pain, tenderness, and rigidity. The presence of early acetonuria is also characteristic. Intussusception, with its pain, colic, and vomiting, may suggest appendicitis, but is rare, except in infants; fever is absent early in the disease, and a tumor is usually present. Acute or subacute suppuration in the right iliac fossa is almost invariably due to appendicitis.

The leucocyte count may be of considerable assistance in differentiating appendicitis from colic, cyclic vomiting, ileocolitis, and intussusception. It should, however, be remembered that in some of the gravest cases the leucocytosis may be slight or there may be none at all. On the whole, while the presence of marked leucocytosis—i. e., above 20,000—may be of considerable assistance in the diagnosis, no inference can be drawn from a normal count or a slight leucocytosis if the child is greatly prostrated. Whenever, in children over two years old, there are symptoms pointing to acute peritonitis, no matter what their combination or variety, appendicitis should always be suspected.

Treatment.—Absolute rest in bed can not be too strongly insisted upon whenever appendicitis is suspected, no matter how mild the attack may appear. As a local application, the ice-bag is to be preferred. Opium should not be given. It does harm by obscuring important symptoms and increasing constipation. The colon should be kept empty by the daily use of enemata. After a thorough clearing of the bowels in the beginning, preferably by a saline, cathartics are to be avoided.

Appendicitis is a surgical disease, and surgical advice should be

sought early. In deciding as to the time of operative interference, it should be remembered that localization of the inflammation is less likely to occur with children than with older patients and that therefore the dangers of general peritonitis are much greater; that the progress of the disease is much less regular; that grave conditions are not revealed at once by grave symptoms; that the disease is an insidious one, and that to foretell the outcome even in the mildest cases is impossible. Taking all these things into account, we believe that immediate operation, once the diagnosis is made, is the course to be recommended in all cases of acute appendicitis in children. The younger the child the greater the urgency for operation.

INTESTINAL WORMS

Judging by published reports, intestinal worms are much more common in Europe than in the northern part of this country. In 18,000 patients treated for medical diseases in our dispensary services in New York and Baltimore there was positive evidence of worms in but 135 cases. Of these, 20 had tapeworms, 55 round worms, 56 thread worms and 4 both round and thread worms. In private practice among the better classes, worms are certainly rare.

Cestodes—Tapeworms.—Cestodes are usually introduced into the body by the ingestion of some form of food containing larvae (cysticerci). The larva of the *tenia solium* is most frequently found in pork; that of the *tenia mediocanellata* in beef: that of the *bothriocephalus latus* in fish; that of the *tenia cucumerina* inhabits dog or cat lice, being introduced into the intestinal tract accidentally by the hands. Several varieties of tenia are found in the human intestine.

Tenia Saginata or Mediocanellata—Beef Tapeworm.—Infection results from eating raw or partially cooked beef containing cysticerci. The worm is from twelve to twenty feet in length, and has a square pigmented head without hooks but provided with four suckers. The full-sized segments are from one-half to three-fourths of an inch long and about half as wide.

Tenia Solium—Pork Tapeworm.—This is a rare form in children, and comes from eating raw or partially cooked pork or sausage. It is from six to ten feet in length, the segments being nearly square. The head is about the size of a mustard seed and is pigmented. It also is provided with four suckers and a proboscis, surrounding which is a circle of about twenty-six hooklets.

Tenia Cucumerina or Elliptica.—The larvae of this form develop in a louse found on the skin of dogs and cats. Children who play with infected animals are the ones affected, the parasite being conveyed to

the mouth usually by means of the hands; it may thus be found even in young infants. This form of tenia is much smaller than either of the preceding varieties, the full length being only from six to twelve inches.

Bothriocephalus Latus.—This is a rare form except in the sea countries of northern Europe and Switzerland, where it is said to be very common. The larvae are harbored by certain fish, by eating which when insufficiently cooked they are introduced into the body. The full-grown worm is from twenty-five to thirty feet in length.

Tenia Nana.—The tenia nana, or dwarf tapeworm, is the smallest of all the cestodes. It is a narrow worm of one-half to three-fourths of an inch in length, and is composed of one hundred to two hundred segments. It has a slender neck and globular head which contains four suckers and twenty or thirty hooklets. The habitat of the nana is the upper part of the ileum where it is often found in immense numbers. A single stool may contain several hundred worms. The ova have two definite membranes within the inner one of which three pairs of hooklets are found. The cysticercus stage of this parasite is not known. It is probable that infection occurs from swallowing the ova themselves. As a similar parasite inhabits the intestinal tract of rats and mice it is possible that these animals play a part in transmission. From the observations of Schloss it seems probable that in the vicinity of New York this is the most frequent intestinal parasite of childhood.

Symptoms.—The only positive evidence of tapeworm is the discharge of the worms or separated segments, either singly or in groups. Occasionally worms pass into the stomach and are vomited. Various abdominal symptoms may be associated with worms, but most of these are very indefinite in character and are more often due to other causes. The most frequent symptoms are bad breath, various annoying sensations, colicky attacks, inordinate or capricious appetite, and diarrhea. Usually, if the patient is in good health, no constitutional symptoms are seen. Sometimes, particularly with the bothriocephalus latus, there is a very grave degree of anemia. The increase in the number of eosinophile cells in the blood is of considerable diagnostic value. They frequently form from four to ten per cent of the leucocytes, while in normal blood the usual number is less than two per cent. Many cases are on record, some of them in children, in which the symptoms of pernicious anemia have been present and have disappeared after the expulsion of the tapeworm. Nervous symptoms are not so often seen as with roundworms, and will be discussed in connection with them.

Treatment.—Prophylaxis requires the cooking of meat to a sufficient degree to destroy the cysticerci. There is especial danger in

eating raw pork or sausage; that from rare beef is much less. The list of drugs used for the expulsion of the worm is a long one; probably the most efficient is the oleoresin of male fern; it is, however, difficult to administer and it is very likely to provoke vomiting. It may be given in capsules containing ℥ x to ℥ xx, or in an emulsion made up with simple elixir and acacia, in which ℥ v to ℥ x are contained in one dram. For a child of four years at least one dram of the male fern should be given in the course of six to eight hours. The vermifuge should be preceded by several hours' fasting, and the bowels previously opened by a laxative. The following plan of administration has been found satisfactory: a light supper of milk, and in the morning a saline laxative on rising, but no breakfast; after the saline has acted freely the remedy is to be given, and following the last dose, half an ounce of castor oil or some other active purge. The effect of the cathartic is aided by a large injection of warm soap and water. Only milk should be given that day. The fragments passed should be carefully examined to see if the head has been expelled, as the worm is very likely to be broken at the neck. If this occurs it will grow again, and in about three months segments will appear in the stool. Other drugs useful for tenia are pumpkin seeds which are given in powdered form, infusion of pomegranate root, turpentine, and chloroform.

Nematodes.—Three varieties are found in the intestinal canal, the *ascaris lumbricoides,* the *oxyuris vermicularis,* and the *uncinaria Americana.*

Ascaris Lumbricoides—Roundworm.—This worm is usually found in the small intestine. It is much more frequently met with in children than is the tapeworm. It is exceedingly rare in infancy, but is usually seen between the third and tenth years. In over two thousand autopsies upon infants we have only twice found a roundworm in the intestine.

The roundworm resembles the ordinary earthworm; it is from five to ten inches long, the female being longer than the male. It is of a light gray color with a slightly pinkish tint, cylindrical, and tapering toward the extremities. The eggs are oval in form, about $\frac{1}{400}$ inch in diameter, and numbered by millions. These worms rarely exist singly; usually from two to ten are present, but there may be hundreds. When very numerous they coil up and form large masses, which may cause intestinal obstruction.

The migration of these worms is curious, and in some instances truly remarkable. They frequently enter the stomach and are vomited. Occasionally one may appear in the nose. They have been known to pass through the Eustachian tube into the middle ear and to appear in the external meatus. Entering the larynx they have produced fatal asphyxia. It is not very rare for them to enter the common bile duct

and produce jaundice. They may even enter in great numbers the smaller bile ducts and produce hepatic abscesses. They have been found in the pancreatic duct, in the vermiform appendix, and in the splenic vein. It has long been known that they would perforate an intestine which was the seat of ulceration, but well-authenticated cases have been reported in which they have perforated an intestine previously healthy, setting up a fatal peritonitis. In Archambault's case they perforated the stomach. In cases of a persistent Meckel's diverticulum, worms have been discharged from an umbilical fistula. They have been found in umbilical abscesses. Considering, however, the frequency of roundworms, migrations are rare.

Symptoms.—The symptoms of roundworms are of the most indefinite kind; often there are none until the worm is discovered in the stools. It is then fair to assume that other worms are also present. The most frequent abdominal symptoms are colic, tympanites, and other symptoms of indigestion, loss of appetite, disturbed sleep and grinding of the teeth at night. These symptoms are much more frequently due to other causes than to worms, but when all are present the existence of worms should be suspected.

A great variety of nervous symptoms may be associated with intestinal worms. They are more often seen with lumbricoids than with either of the other varieties. The symptoms may be of the most puzzling character, and may simulate very closely those of serious organic disease. There may be prolonged low fever, chills, headache, vertigo, hallucinations, hysterical seizures, epileptiform attacks, convulsions, tetany, transient paralyses such as strabismus, and even hemiplegia and aphasia. All these have been observed in connection with intestinal worms, and from the fact that the symptoms disappeared completely after the worms were expelled, there seems to be but little doubt that they were the cause of the symptoms. As in the case of the abdominal symptoms, however, intestinal worms are only one of the causes of such nervous disturbances, and certainly not a frequent one; but the possibility that nervous disturbances may depend upon worms should not be overlooked. The blood generally shows eosinophilia, as in patients with tapeworm.

The only positive evidence of the existence of roundworms is the discharge of a worm from the body, or the discovery of the ova in the stools. A microscopic examination of the stools is a valuable means of diagnosis, and one that is too infrequently employed. When worms are present the ova may be found in great numbers. Their continued presence, after the discharge of one worm, indicates that other worms remain.

Treatment.—An efficient agent for the removal of the worms is santonin. The same plan of administration may be followed as in the case of the tapeworm, viz., to give the drug on an empty stomach,

preceded by a laxative. Santonin is best given in powdered form mixed with sugar. For a child of five years as much as three grains are usually required. This amount should be given in three doses at intervals of four hours, soon followed by a purge of calomel or castor oil. Oil of chenopodium is somewhat easier of administration and is quite as efficient. It may be given as described under the treatment for Hookworm. The great difficulty with santonin is its tendency to provoke vomiting. Occasionally in susceptible children, even with ordinary doses, toxic symptoms may develop, such as yellow vision, dark-red or yellow urine, and nervous excitement or delirium.

Oxyuris Vermicularis—Pinworm—Threadworm.—The oxyuris resembles a short piece of white thread. The female is about one-third of an inch long, the male about one-half that length, but is less frequently seen. The worm tapers toward the tail. The ova are of slightly irregular size, and are considerably smaller than those of the roundworm.

The oxyuris inhabits the rectum, the cecum, and, according to Still, very frequently the appendix. These worms may be found also in the lower small intestine, in the stomach, and even in the mouth. If present in the rectum they are usually discovered by separating the folds of the anus. The number of worms is usually large. The irritation to which they give rise causes a great production of mucus, and frequently leads to a chronic catarrh of the colon of considerable severity. The worms are imbedded in the mucus; often they form with it small balls. According to Leuckart, they are incapable of multiplying *in situ*. Doubt has recently been thrown upon this view by the observations of Still. From the immature character and the large numbers of the worms found in the appendix (111 in one case), this writer believes that the appendix may be a breeding place. The ova as well as the worms are passed in enormous numbers with the stools. They attach themselves to the folds of the skin, the hairs about the anus, and even to the genitals. The patient may, through lack of cleanliness of the parts, continually re-infect himself. After discharge from the body, the ova may be carried by flies and deposited upon fruits, vegetables, or in drinking water.

Symptoms.—The principal local symptom caused by the oxyuris is itching of the anus or the genitals. This is caused by the migration of the worms from the bowel, and usually comes on at about the same hour at night, generally soon after the patient has retired. It is sometimes so intense as to be almost intolerable. It leads to frequent micturition, to incontinence of urine, in the male to balanitis, and in the female to vaginitis or vulvitis, and in both, but especially in the latter, it may be the cause of masturbation. Owing to the catarrhal colitis which is ex-

cited, there is discharged from time to time a large quantity of mucus. Severe colicky pains are often associated. The irritation may lead to prolapsus ani. Nervous symptoms are not so frequently associated as with the other varieties of worms, although we have seen at least one case of chorea in which they were almost certainly the cause. They have been known to excite convulsions. The general health is sometimes undermined and there may be marked and progressive loss in weight.

Treatment.—This is usually spoken of as a very simple matter, and no doubt in recent cases, or where the number of worms is small, this is true; but where the number is large, and considerable catarrhal inflammation of the colon is present, it is often a matter of the greatest difficulty to rid the bowel of these parasites. Cases frequently resist treatment by injection for months, even though thoroughly used. The reason for this is, that only the lower colon is reached by injections while the worms may be chiefly in the cecum or even in the appendix and small intestine. While, therefore, injections are important and indeed invaluable, they can not be relied upon exclusively. The most scrupulous attention to cleanliness is an absolute necessity as the first step in the treatment of all cases. It is well to bathe the parts about the anus after each stool, and even two or three times a day, with a bichlorid solution, 1 to 10,000. Itching is best controlled by the application of mercurial ointment to the folds of the anus at bedtime, this effectually preventing the escape of the worms from the bowel. The local application of cold will sometimes have the same effect. The most efficient of the injections is probably the bichlorid. The colon should first be thoroughly cleansed by an injection of lukewarm water containing one teaspoonful of borax to the pint, in order to remove the mucus. When this has been discharged, half a pint of the bichlorid solution of the strength mentioned should be injected high into the bowel through a catheter, and retained as long as possible. This should be repeated every second or third night. On other nights a simple saline injection may be employed. The infusion of quassia, asafetida, aloes, and garlic are also useful. Solutions of carbolic acid should never be employed.

When the worms are high in the colon, drugs by the mouth must be combined with injections. The most efficient remedies are santonin and the oil of chenopodium, which may be used as for roundworms. The expulsion of the worms is aided by saline cathartics; simple bitters, such as gentian and quassia, are also of some value. We have known one case, which resisted for over two years everything which had been tried, to be cured in two or three weeks by injections of a decoction of garlic, in connection with which garlic was given in liberal quantities by the mouth.

Uncinaria Americana or Hookworm.—This belongs to the class of nematodes. The males are one-fourth to one-half inch in length and the females slightly longer. The parasite resembles the *ankylostomum duodenale* of Europe. Infection usually takes place through the skin of the bare feet, more rarely that of the hands. It is possible, however, to contract the disease by eating dirty fruit or vegetables contaminated by the developing larvae; but infection does not occur from swallowing the ova or young larvae. After entering the skin the larvae find their way into the circulation and thus reach the lungs. From the lungs they may migrate or be coughed up into the mouth and then swallowed. They are not acted upon by the gastro-intestinal secretions, and in the upper part of the small intestine they develop into mature worms. These may exist in the small intestine for years.

The symptoms in the milder cases are minor digestive disturbances, general malnutrition with moderate anemia and arrested growth. In the more severe cases the anemia is very marked, the hemoglobin often falling to thirty per cent or below. The leucocytes are normal in number or slightly increased; but the percentage of eosinophiles is above the normal. In most patients the proportion reaches five or ten per cent; it may however be twenty-five per cent or even higher. Edema of the face is common and there may be general dropsy without albuminuria. Affected children besides being very backward in physical development, are dull, inattentive and entirely wanting in physical or mental energy. The appetite is sometimes absent; but more characteristic is the craving, not only for every kind of food, but for such articles as clay, dirt, chalk, etc. Death may be due to the progressive failure of nutrition or to intercurrent disease.

Prophylaxis in the individual consists chiefly in the protection of the feet of persons living in an infected district, by wearing shoes. The chief remedy for the hookworm is thymol. Its administration should be preceded by one or more full doses of the sulphate of magnesia or soda given after twelve hours' fasting. The quantity of thymol given to a child of five years should be six or eight grains in divided doses in the course of three or four hours. It may be administered either in capsule or in suspension. Two hours after the last dose, the salts should be repeated; but no food should be given until the cathartic has acted freely. Castor oil should not be used. A repetition of the treatment is often necessary before a cure is accomplished.

The oil of chenopodium is apparently quite as effective as thymol and has the advantage of being much cheaper. It may be administered dropped upon sugar. The usual dosage is one drop per year of age up to ten years. A dose of Epsom salts is given on the preceding day and three doses of the oil at two-hour intervals the next morning, the last

dose to be followed by castor oil. It should be remembered that chenopodium is toxic in over doses.

CHAPTER X

DISEASES OF THE RECTUM

PROLAPSUS ANI

Under this term are included two conditions. In the first, or partial prolapse, there is simply an eversion of the mucous membrane which protrudes beyond the sphincter. In the second, or complete prolapse, there is invagination of the rectal wall for a variable distance, usually two or three inches.

Etiology.—Prolapse is most common in children during the second and third years. Its frequency in early life is partly due to the lack of support furnished by the levator-ani muscles. It also occurs very readily when the ischiorectal fat is scanty; it is therefore often seen in children suffering from marasmus. The exciting cause may be anything which provokes severe and prolonged straining. This may be either the tenesmus accompanying inflammation of the rectal mucous membrane or chronic constipation. It may come from phimosis or stricture of the urethra, and it is a very frequent symptom of stone in the bladder.

Symptoms.—Prolapse usually occurs during the act of defecation. It is generally easily reduced, but shows a great disposition to return with every stool. In obstinate cases the bowel comes down at other times. The appearance of the tumor varies with its size. In the slighter form there is simply a ring composed of a fold of mucous membrane surrounding the anus. In the more severe form there is a flattened, corrugated tumor, usually about the size of a small tomato. The mucous membrane covering the tumor is of a deep purplish-red color, and bleeds readily. It may be the seat of catarrhal or membranous inflammation. The diagnosis in most cases is easy, although the tumor has been confounded with polypus and intussusception.

Treatment.—In most cases reduction is easily accomplished by laying the child upon his face across the lap, and making gentle pressure upon the tumor with oiled fingers. The application of cold, either by means of ice or cold cloths, is of assistance in cases which are not at once reduced by pressure. After reduction, in the milder cases the child should be kept upon his back for at least an hour. When the tumor tends to come down with every stool, special attention should be given at this

time. If an infant, the bowels should always move while the child lies upon his back, and during defecation the buttocks should be pressed together by a nurse. Older children should use an inclined seat placed at an angle of about forty-five degrees, but should never sit upon a low chair or assume any position in which straining is easy. After defecation the patient should lie down for at least half an hour. When there is constipation, the bowels should be kept free by means of laxatives. If there is diarrhea, tenesmus may be overcome by frequent sponging with ice water, or by the use of small injections of ice water and tannic acid, in the proportion of twenty grains to the ounce. In more severe cases it may be controlled by the use of suppositories of opium. When the bowel tends to come down frequently, this may be prevented by the use of an adhesive strap two or three inches wide, placed tightly across the buttocks. This is better in the milder cases than a T-bandage. The great majority of the cases are cured by these means in the course of a few weeks.

In the most severe cases the bowel not only protrudes during defecation, but also in the interval, and it may be down for days at a time. Such cases are rarely seen except in infants who have very flabby muscles, and but little adipose tissue at the floor of the pelvis. Reduction is sometimes difficult in cases when the prolapse has lasted a long time. It is often facilitated by painting the protruding part with a solution of epinephrin, and then dilating the sphincter by passing the finger into the central opening of the tumor. After reduction, suppositories containing from one-fourth to one-half grain of cocain may be inserted. They are more efficient than those containing opium or belladonna. A firm pad should be applied over the anus, held in position by a T-bandage. For several days at a time a short rubber tube may be kept in the rectum, held in place by adhesive plaster. The bowels should be kept freely open. Where all other measures fail, the protruding part may be touched with the Paquelin cautery, linear markings being made at intervals of an inch. Amputation or excision is not required in children.

FISSURE OF THE ANUS

This is not a very uncommon condition in children. The most frequent cause is the passage of a large, hard, fecal mass. Sometimes it results from traumatism inflicted with the nozzle of a syringe while giving an enema. It may be produced by the scratching excited by pinworms. In the beginning there is a simple tear at the margin of the anus. The laceration which is produced usually heals promptly; but if the cause is repeated, healing is prevented, and there is finally produced

a linear ulcer, or a true fissure, which may last for some time and be a source of great annoyance.

A fresh fissure has the appearance of any other tear at a mucocutaneous orifice. One of longer standing has a gray base, slightly indurated edges, often discharges a small amount of pus, and bleeds a drop or two with nearly every movement of the bowels. The most constant symptom is pain, which usually occurs with the act of defecation and continues for some time afterward. It is most severe when the fissure is just at the margin of the sphincter, and leads the child to resist every inclination to have the bowels move, so that it becomes a cause of chronic constipation, which condition again greatly aggravates the fissure. The pain is often referred to other parts in the neighborhood.

The treatment is simple and usually efficient. It consists in cleanliness, overcoming the constipation, and touching the fissure with nitrate of silver, preferably with the solid stick. If the case is not speedily relieved by such measures, the sphincter should be stretched as in adult patients.

PROCTITIS

Proctitis, or inflammation of the rectum, usually occurs with inflammation of the rest of the large intestine, but it may occur alone. It is to the cases in which only the rectum is involved that the term is generally applied.

The causes are for the most part local. A frequent one in infants is the use of irritating injections or suppositories, either for the relief of constipation or as a means of administering certain drugs. We have seen one obstinate case in an infant a year old, following the prolonged use of glycerin suppositories. It is sometimes caused by traumatism, especially by the careless giving of an enema. It accompanies pinworms. In certain cases it may result from direct infection through the anus. This may be from a gonococcus inflammation extending from the vagina or urethra, or from an infection due to other bacteria, particularly in cases of measles, scarlet fever, and diphtheria; or, finally, it may be due to syphilis. Proctitis may be catarrhal, membranous, or ulcerative.

Catarrhal Proctitis.—The pathological conditions are the same as in ordinary catarrhal inflammation of the intestinal mucous membrane. By the introduction of a speculum, or by simply everting the mucous membrane, it is seen to be reddened, swollen, and bleeds easily. There is a copious secretion of mucus. In cases of long standing there may be superficial ulceration appearing as a white or yellowish-white surface, usually just inside the sphincter.

The symptoms are chiefly local, although a condition of general irri-

tability may result from the local condition. There is heightened reflex action, so that the stool often comes with a spurt. There is pain with defecation, and mucus is discharged, usually as a clear, jelly-like mass, and sometimes in the form of a cast, but not generally mixed with the stool. There are usually traces of blood, sometimes quite large hemorrhages. In the most acute cases, tenesmus is present both during and after the stool. There may be prolapsus ani. The skin in the vicinity is irritated by the discharges, most frequently so in infants. If the cause is pinworms, there may be intense itching. The duration of the disease is indefinite, depending upon the cause. It may be a few days or many months. The inflammation may extend from the rectum to neighboring parts, leading to ischiorectal abscess.

Membranous Proctitis.—It has been customary to describe this as a complication of diphtheria, usually occurring with diphtheria of the external genitals. As few of these cases have been studied bacteriologically, it is impossible to say what proportion of them, if any, are to be regarded as true diphtheria. When the infection is from the intestine above, the rectum is never affected alone. When it is from below, this may be the case. The lesions are the same as in membranous inflammation occurring higher in the colon. The symptoms resemble those of the catarrhal variety with the addition that the stools contain pieces of pseudo-membrane. This can be made out only by repeatedly washing the discharges with water. If accompanied by prolapse, the pseudo-membrane may be seen. Membranous proctitis may be complicated by a membranous inflammation of the genitals or the perineum. Although it is usually acute, it may last for weeks.

Ulcerative Proctitis.—Ulcers of the rectum may be the result of a catarrhal inflammation; these, however, are usually superficial, affecting the mucous membrane only, and in most cases heal rapidly. Sometimes they extend more deeply into the submucous or even the muscular coat. They are then chronic, often very obstinate, and may last indefinitely. Follicular ulcers of the rectum are nearly always associated with the same condition in the sigmoid flexure. These are always multiple and usually small, rarely being more than a quarter of an inch in diameter. Sometimes the small ones coalesce, producing much larger ulcers. Single ulcers may be of tuberculous origin. Syphilitic ulcers are extremely rare in children.

The symptoms of ulcer of the rectum are mainly two—pain and hemorrhage. The pain is of variable intensity, and may be referred to the coccyx, or to any of the neighboring parts. The amount of bleeding may be small, the blood coming in clots, or it may be fluid and in so large a quantity as to produce general symptoms. It usually accompanies every stool. In addition the stool contains more or less pus, par-

ticularly in chronic cases. When the ulcer is low down, tenesmus is usually present and may be a prominent symptom. The duration of the symptoms is indefinite; often they last for many months and lead to a marked deterioration in the general health. A positive diagnosis of ulcer can be made only by examination with a speculum.

Treatment.—In cases of acute catarrhal proctitis injections of some bland fluids should be employed, such as a starch-water, limewater, a mixture of oil and limewater, or a warm one-per-cent saline solution. The local cause, if one exists, should be removed. In the most acute cases the patient should be kept in bed. When the tenesmus is severe, suppositories of opium may be used. In the more chronic cases saline injections should be given, and followed by a mild astringent like tannic acid, ten grains to the ounce, or a one-per-cent solution of hamamelis. Cases associated with pinworms are especially obstinate. Here the treatment is first to be directed to the worms, and afterward to the proctitis.

In the membranous cases the same measures are to be employed, and in addition the injection of a warm boric-acid solution two or three times a day.

Cases of ulcer require the most careful treatment. In many there is but little tendency to spontaneous recovery. An examination with the speculum should be insisted upon in all cases of chronic proctitis, to make sure of the diagnosis. Rest in bed is essential to a rapid improvement. The bowels should be kept freely open by the use of laxatives and injections of a boric-acid solution, or one or two ounces of liquid albolene may be injected every night and retained. If this does not relieve the patient, a weak solution of nitrate of silver (one grain to the ounce) may be injected daily after washing out the bowel with tepid water. If a stronger solution than this is used, it should be neutralized after half a minute by the injection of a saline solution.

ISCHIORECTAL ABSCESS

This is not a very rare condition even in infancy. Infection from the rectum, usually through the lymph channels, seems to be the most common cause, although sometimes the abscess may be traced directly to traumatism.

Essentially the same varieties of inflammation are seen in early life as in adults. Most of these cases recover promptly after simple incision and cleanliness, fistula being a rare sequel.

RECTAL POLYPUS

Polypi are rarely seen in children, but, when present, may be the cause of rather obscure symptoms. The most important one is hemorrhage. This at first occurs at intervals of days or weeks. The amount of blood lost is from a dram to an ounce or more. Later, the hemorrhages become more frequent and may be almost continuous, although rarely profuse enough to produce serious symptoms. The diagnosis of polypus is made only after a local examination. Sometimes the tumors are within the reach of the finger; in other cases a proctoscope must be employed. Spontaneous cure often takes place by the sloughing of the tumor, after which the bleeding soon ceases. In other cases operation is necessary.

HEMORRHOIDS

These, fortunately, are not often seen in children, although they occur in those as young as three or four years, and in some cases may even be congenital. The principal cause is chronic constipation, rarely diarrhea. The tumors are generally small and external, the chief symptom complained of being pain on defecation. Bleeding sometimes accompanies the pain, but the hemorrhages are usually small. The treatment is to be directed toward the underlying cause. In most of the cases this suffices to cure the condition. Operation is rarely required in young children, although neglect may make this procedure necessary.

INCONTINENCE OF FECES

Inability to control the fecal evacuations is seen in certain cases of paraplegia due to myelitis, after injury of the lumbar portion of the spinal cord, and in spina bifida. It may occur with the usual or with the occult variety, associated with incontinence of urine, when there is no paralysis of the extremities. It is also seen in acute disease, as in the coma of meningitis, and occasionally in the typhoid condition and in extreme adynamia, from any cause. It is quite common in severe attacks of chorea. It may sometimes be seen after operations for atresia of the anus or rectum. In all these conditions incontinence of feces is a symptom giving rise to much annoyance and needing careful attention. Uncleanliness with reference to excreta, seen in idiocy, can hardly be classed as incontinence.

Besides these familiar forms, the condition is sometimes seen from

causes somewhat resembling those of incontinence of urine. The tone of the sphincter becomes so feeble that it does not resist even the slightest impulse to evacuate the rectum. The discharge may take place with but little warning, and may occur either by day or night. In some cases a local cause exists, such as stretching of the sphincter by an old rectal prolapse. It has followed overdistention of the rectum from prolonged chronic constipation. Ostheimer reports a case in which a vesical calculus was present. It is sometimes seen after severe acute illness, as a result of a loss of general muscular tone. In certain children it has been known to persist from infancy until the age of ten or twelve years. It may come on as a somewhat acute condition in highly nervous patients with poor general nutrition. The causes are chiefly of local and nervous origin. The treatment is rather unsatisfactory, except in recent cases and in those due to local causes which can be removed. If constipation exists the rectum should be emptied daily, preferably by an enema. The remedies which have proven most successful are strychnia, ergot, and belladonna, but they must be given in full doses, sometimes advantageously by suppository as well as by mouth. The general health should receive careful attention.

CHAPTER XI

DISEASES OF THE LIVER

Aside from the different forms of degeneration which are seen in the various infectious diseases, the liver is not often the seat of serious disease in infancy and early childhood. In later childhood nearly all the forms seen in adult life are occasionally met with, although even then they are quite rare.

Size and Position.—The weight of the liver in the newly-born child, from one hundred and seven observations of Birch-Hirschfeld, is 4.5 ounces (127 grams), or about 4.2 per cent of the body weight. The accompanying table gives the results of one hundred and seventy-four ob-

Age.	Average.		Per cent of body weight.
	Ounces.	Grams.	
3 months	6.3	180	3.1
6 "	7.5	212	3.0
12 "	11.0	311	3.40
2 years	14.0	397	3.37
3 "	16.0	453	3.26

servations upon the liver in infancy in the autopsy room of the New York Infant Asylum.

In adults, according to Frerichs, the weight of the liver is about 2.5 per cent of the weight of the body.

The upper border of the liver is best made out by percussion. In the child, the upper limit of the liver dulness in the mammary line is found in the fifth intercostal space; in the axillary line, in the seventh space; posteriorly, in the ninth space. The lower border is best determined by palpation. This, as a rule, in the mammary line is found about one-half an inch below the free border of the ribs. According to Steffen, the left lobe is relatively larger in the child than in the adult. The liver may be displaced downward by contraction of the chest, as in rickets, or by an accumulation of fluid in the pleural cavity. It is frequently found lower than normal in conditions of great emaciation, owing to relaxation of the abdominal walls and its ligamentous supports. Upward displacement is much less frequent, and depends usually upon ascites or abdominal tumors.

Malformations and Malpositions.—Congenital malformations relate chiefly to the bile ducts. These have been considered in the chapter devoted to Icterus in the Newly Born.

The liver may be found upon the left side in cases of general transposition of the viscera. In diaphragmatic hernia it has been found in the thoracic cavity.

CATARRHAL JAUNDICE

This is due to a catarrhal inflammation of the common bile duct with which there is usually associated a similar inflammation of the duodenum and sometimes of the stomach also. The term *gastroduodenitis* is sometimes used synonymously with catarrhal jaundice. The jaundice in these cases is due to obstruction which is caused by swelling of the mucous membrane of the bile duct. Catarrhal jaundice is rare in infancy. In children from three to six years old it is not uncommon, and curiously occurs much more frequently in the fall months. This suggests an infectious origin. For the most part its causes are obscure.

It occasionally complicates malarial fever and may occur with any of the infectious diseases. Rehn has described a form which occurred epidemically.

The symptoms of the disease are quite uniform. When primary, the onset is like an ordinary attack of indigestion, with vomiting, pain, slight fever, and a moderate amount of prostration. The vomiting in some of the cases is repeated for several days. The pain may be quite

severe, and localized in the region of the duodenum. It may be associated with tenderness in this region. The bowels are usually constipated. After three or four days, icterus, which is the only diagnostic symptom, appears. It is first seen in the conjunctivae, afterward in the skin, varying in degree according to the severity of the attack, but in most cases not being very intense. It is accompanied by the regular symptoms of obstructive jaundice. The stools are gray, sometimes white: there is a marked amount of intestinal flatulence. The urine is very dark, of a yellowish-green or bronze hue, and stains the clothing. There is complete anorexia; the tongue is thickly coated with a white fur. Headache, dulness, and languor are present, and the patient feels wretchedly. The slow pulse and the itching skin are uncommon symptoms in children. The liver is usually found slightly enlarged, and sometimes tender on pressure. The duration of the disease is about two weeks, the general symptoms disappearing before the icterus. Recurrences and prolonged attacks are occasionally seen. The diagnosis rarely presents any difficulty, and the prognosis is invariably good.

The fats and starches of the food should be reduced to a low point or be entirely prohibited. Patients usually do much better upon a diet of rare meat, fruit, and skimmed milk, or buttermilk. If there is very much vomiting, food should be temporarily withheld and later skimmed milk should be given largely diluted with limewater. The amount of food given should be small, but water should be allowed freely, particularly the alkaline mineral waters. The bowels should be kept open, if necessary by means of cathartics. In most of the cases no other treatment is necessary. When the pain is severe it may be relieved by counter-irritation by mustard, turpentine, or even cantharides. The restricted diet should in all cases be continued for at least a week after the jaundice has disappeared.

NEW GROWTHS

New growths of the liver are rare in children and are usually secondary to deposits elsewhere, most frequently in the kidney. They are generally sarcomatous. Primary sarcoma of the liver has, however, been observed, and at so early an age as to make it practically certain that the condition was a congenital one. In most of the cases there is simply a slowly increasing abdominal tumor and progressive asthenia.

ACUTE YELLOW ATROPHY

This form of hepatic disease is rare in children. Greves has reported a well-marked case in an infant of twenty months, and has

collected seventeen other cases under ten years of age; the youngest was in an infant three months old. The symptoms and course of the disease are essentially the same as in adults. A condition closely allied to this is occasionally seen as a result of the administration of chloroform.

CONGESTION OF THE LIVER

Congestion of the liver occurs from the same causes in children as in adults. Acute congestion is not often seen. Chronic congestion is more common, and is usually secondary to general venous obstruction dependent upon congenital or acquired heart disease, atelectasis, or other pulmonary conditions, particularly chronic pleurisy, chronic interstitial pneumonia and emphysema. Chronic congestion of the liver causes no characteristic symptoms except a moderate enlargement of the organ with some pain and tenderness. The treatment is that of the primary disease.

ABSCESS OF THE LIVER—SUPPURATIVE HEPATITIS

In 1890 Musser found but thirty-four recorded cases of abscess of the liver in children under thirteen years. Since that time a few additional cases have been reported. In the above collection, there have not been included cases of suppurative hepatitis occurring in the newly born.

As in adults, abscess of the liver may result from traumatism, or it may be secondary to suppurative pylephlebitis, which depends upon a focus of infection in the umbilical vein, or in some part of the abdomen from which the branches of the portal vein arise. Pylephlebitis may follow appendicitis, it may follow typhoid fever directly, or be due to suppuration of the mesenteric glands or peritonitis following typhoid. In seven of the cases collected by Musser the disease was due to migration of roundworms from the intestine into the hepatic ducts. Menger (Texas) has reported one case following dysentery, the only one, we think, on record in this country. Very rarely great numbers of minute abscesses are found as a result of suppurative thrombosis of the jugular bulb following middle ear disease. In quite a number of cases no adequate cause can be found.

In the cases occurring in pyemia and in those associated with pylephlebitis there are usually several abscesses; in traumatic cases generally but one. If untreated, the majority of cases prove fatal either from exhaustion or from rupture into the pleura or peritoneum. In Asch's case spontaneous cure took place by rupture into the intestine.

Symptoms.—Occasionally abscess of the liver is latent, but in most of the cases the symptoms are marked and sufficiently characteristic to make the diagnosis a matter of no great difficulty. The most constant general symptoms are chills, which may be single, but are usually repeated; fever, which is commonly of the hectic variety and followed by sweating; prostration, vomiting, diarrhea, and cachexia. Jaundice is present in less than half the cases, and is rarely intense. The liver is almost invariably sufficiently enlarged to be easily made out by palpation or by percussion; the enlargement in most cases is chiefly downward. Pain is quite constant and frequently intense, but not always in the region of the liver. It may be in the epigastrium, at the umbilicus, in the lower part of the abdomen, and occasionally in the right shoulder. Tenderness over the liver is usually present. A positive diagnosis of hepatic abscess is to be made only by aspiration and the withdrawal of a fluid having the characteristics of "liver pus." Pulmonary symptoms usually exist with an abscess occupying the convexity of the right lobe. There may be cough and dyspnea from pressure, or pleurisy from extension of the inflammation through the diaphragm, or from rupture into the pleural cavity. The usual duration of abscess of the liver after the beginning of the symptoms is from one to two months. The prognosis will depend upon the cause of the disease. The pyemic cases are usually fatal. In Musser's collection, the proportion of recoveries was about thirty per cent. At the present time, with improved methods of treatment and earlier diagnosis, the outlook is somewhat better than this.

Treatment.—This is purely surgical, unless the abscess is due to an amebic colitis. In that case emetin hydrochlorid should also be given hypodermically as advised under amebic colitis. Cases have been reported where, after undoubted evidences of abscess have been present, recovery has ensued with the use of emetin alone. Without operation, however, the chances of recovery are slight. A small number of cases have been cured by aspiration, but in the vast majority of abscesses due to any cause only incision and drainage are to be depended upon, and, if the abscess is accessible, should be resorted to as soon as the diagnosis is established.

CIRRHOSIS

Cirrhosis of the liver is exceedingly rare in early life, although quite a number of cases are now on record between the ages of seven and fourteen years. Sixty-five have been collected by Howard and fifty-three by Laurè and Honorat. Nearly all the cases in these collections were between nine and fifteen years old. Cirrhosis in infancy is usually of syphilitic origin. Two-thirds of those in Howard's collection were males.

The etiology in most of the cases is obscure; in over half of those reported no cause could be discovered. Fifteen per cent of Howard's cases were traced to alcoholism, eleven per cent to syphilis, and eleven per cent to tuberculosis. Laurè and Honorat believe that the eruptive fevers sometimes play an important part as an etiological factor, and that at other times the cause is possibly malaria.

The anatomical features of cirrhosis in early life are essentially the same as in adults. The liver is sometimes enlarged, but usually it is smaller than normal. The connective tissue may be distributed around the lobules, along the bile ducts, in irregular patches, or in striations through the organ. Associated with this there is atrophy and fatty degeneration of the liver cells. In some of the cases reported there has been also a similar increase in the connective tissue of the spleen and kidneys.

Symptoms.—These are very much the same as in adult life. In the beginning there are the indefinite disturbances referable to the digestive organs, and the liver may be slightly enlarged; later there is ascites, enlargement of the spleen, and dilatation of the abdominal veins. Ascites is a pretty constant symptom, and is generally marked. Slight icterus is often present, but a marked amount is rare. There may be hemorrhages from the stomach, from the nose, or from other organs; in a few cases there is slight fever. The late symptoms are, a small liver, marked ascites with the consequent embarrassment of respiration, cachexia, and sometimes general dropsy. Diarrhea is a much more constant symptom than in adults. Death usually takes place from exhaustion. The course of cirrhosis in children is commonly more rapid than in adults, and the progress is steadily downward.

Treatment.—Medicinal treatment is of avail only with patients who are syphilitic. These should be put upon antisyphilitic remedies in full doses. The treatment in other respects is symptomatic and palliative. The ascites may require paracentesis as in adults.

AMYLOID DEGENERATION (*Waxy or Lardaceous Liver*)

From the experiments of Krawkow, Davidsohn, and others there seems now little doubt that amyloid degeneration can be produced by the prolonged action of the staphylococcus aureus, and probably by other organisms. Amyloid degeneration of the liver is associated with similar changes in the spleen and kidneys, and sometimes in the villi of the small intestine, and is usually seen in children after long-continued suppuration in chronic bone or joint disease, empyema, tuberculosis, or syphilis.

The liver is generally very much enlarged; in extreme cases a weight

of six or seven pounds may be reached. It is of a glistening, waxy appearance, very firm and hard. With a solution of iodin, a mahogany-brown reaction is obtained. The amyloid substance is deposited between the capillaries and the hepatic cells, leading to occlusion of the vessels and atrophy of the cells from pressure.

Amyloid liver *per se* produces few symptoms. Ascites is rarely present except in cases in which the liver is very large, and jaundice does not occur. In addition to the symptoms of the original disease in the course of which the amyloid degeneration occurs, there is the peculiar waxy cachexia which is seen in no other condition, but resembles somewhat that belonging to malignant disease. The face has the appearance of alabaster, and the skin has a singular translucency. The liver may be so large as to form a tumor, sometimes nearly filling the abdominal cavity. Not infrequently it extends to the umbilicus, and even to the crest of the ilium. The surface is smooth and hard, and the edges usually rounded. There is no localized pain or tenderness. The spleen is invariably enlarged. As a result of the associated amyloid degeneration of the kidney, there may be anasarca and albuminuria. Dropsy may occur from pressure of the large liver upon the vena cava, apart from the condition of the kidney.

Amyloid changes usually take place slowly, the whole course of the disease being marked by years, the patient dying from slow asthenia, from nephritis, or from some acute intercurrent disease. As a rule, cases go on steadily from bad to worse; but sometimes, after the disease has reached a certain point, the condition remains stationary for a long time.

The prognosis is always bad, although in a few cases improvement, and even cure, are stated to have occurred after the excision of the diseased joints upon which the amyloid degeneration depended. When due to syphilis, the usual antisyphilitic remedies should be given.

FATTY LIVER

Fatty infiltration of the liver is generally a secondary condition in early life, and causes no symptoms by which it can be positively recognized. Considerable discussion has of late arisen regarding its frequency in infants. From our records at the Babies' Hospital, Wollstein has tabulated 345 consecutive autopsies in which the condition of the liver was carefully noted. The liver was fatty in 201, or 58 per cent. Of these autopsies, 63 were cases of tuberculosis, in 43 of which, or 68 per cent, the liver was fatty.

The general nutrition of the 345 infants was as follows:

Wasted	188:	liver fatty,	104,	or 55	per cent—very fatty in	17.
Fairly nourished	80:	" "	52,	" 65	" " " " "	9.
Well nourished	77:	" "	45,	" 59	" " " " "	20.

These figures coincide very closely with the observations of Freeman at the New York Foundling Hospital, and indicate that fatty liver is not, as has been so often asserted, much more frequent in wasted infants than in others. The cause of this change in the liver is as yet but little understood.

The liver is moderately enlarged, smooth, with rounded edges, of a yellowish-red or a lemon-yellow color, and can be indented with the finger. A warm knife becomes coated with oil after cutting. Microscopically there is seen an accumulation of fat in the liver cells, usually irregularly distributed, but chiefly in the periphery of the lobule. Jaundice, ascites, and the other peculiar symptoms of hepatic disease are absent. The liver is moderately increased in size. Its functions are not interfered with in such a way as to be recognized by the symptoms. The treatment is that of the original disease.

HYDATIDS

Echinococcus disease of the liver, while rare among adults in this country, is almost unknown in children. We have been able to find but two recorded cases in America. From twenty-two European cases collected by Pontou, it appears that unilocular cysts are especially frequent in young subjects. If the upper surface is affected, pulmonary symptoms, cough and dyspnea, are usually present; if the under surface of the organ, there is pressure upon the portal vein, the vena cava, bile ducts, stomach, and intestines. This pressure may cause icterus, dilatation of the superficial abdominal veins, and sometimes ascites. The local signs are enlargement of the liver with a tumor, which is easily recognized in children because of the thin abdominal walls. The hydatid fremitus is usually obtained. By aspiration a clear fluid is withdrawn, showing under the microscope the presence of the hooklets, which establishes the diagnosis. Occasionally cure may take place by spontaneous rupture or suppuration of the cyst, but in most cases, when left to itself, the disease proves fatal. The treatment is surgical, and consists in aspiration or in incision, and the evacuation of the cyst.

BILIARY CALCULI

Up to the age of puberty calculi are extremely rare. Of twenty cases collected by Still, eleven occurred in newly-born infants or else gave

symptoms during the first month of life. The prominent symptom was intense and persistent jaundice. Nearly all died within the first month, the autopsy usually showing multiple calculi in the common duct.

The cases in older children do not differ from those in adults.

CHAPTER XII

DISEASES OF THE PERITONEUM

Inflammation of the peritoneum is seen at all ages, even in the first weeks of life; but is less frequent in children than in adults since most of the causes which are operative in later life either do not exist at all in childhood or are infrequent.

We shall consider separately acute, chronic, and tuberculous peritonitis.

ACUTE PERITONITIS

Acute peritonitis may occur at any period of infancy or childhood. It may even exist in intra-uterine life. In the newly born, peritonitis is not infrequent. After this time it is exceedingly rare during infancy, only four cases, including all varieties, being met with in 726 consecutive autopsies in the New York Infant Asylum. After the fifth year the disease is relatively much more common. Of the 187 cases above referred to, 25 per cent occurred in the newly born, 21 per cent between one and five years, and 54 per cent between the fifth and the sixteenth years.

Etiology.—In the newly born, peritonitis is seen as one of the frequent lesions of acute pyogenic infection. It is usually due to direct infection through the umbilical vessels. In infancy and childhood, peritonitis occurs both as a primary and secondary inflammation. The primary form is rare. It may be due to traumatism, such as falls or blows, or to surgical operations upon the abdomen; it has occurred after an injection for the cure of a congenital hydrocele. Very rarely the inflammation seems to have been excited by exposure, and it may follow severe burns. Cases of acute peritonitis are occasionally seen which are apparently primary. We have met with four in young children, two being due to the pneumococcus and two to the streptococcus.

The secondary form is more common. The most frequent of all causes is appendicitis, which should always be suspected in acute perito-

nitis occurring without definite cause. Extension of inflammation from the viscera to the peritoneum is very much less frequent in children than in adults. It is very rarely seen as a complication of dysentery. It is also rare in typhoid fever. It is occasionally due to abscess of the liver, ulcer of the stomach, acute intestinal obstruction from internal strangulation, intussusception, volvulus, and congenital atresia. It may extend from inflammation of the pleura. This may be in the form of an empyema which burrows through the diaphragm, or, without burrowing, the infection may take place through the lymph channels; or it may be secondary to a general pneumococcus septicemia. Peritonitis is infrequently due to infection through the female genital tract, especially in gonococcus vulvovaginitis in older girls. Extension of inflammation from the male genital organs is very rare. In one case at the New York Infant Asylum, fatal peritonitis in an infant started from a suppurative inflammation of the tunica vaginalis of unknown origin, the infection extending into the peritoneum through the inguinal canal. Any abscess in the neighborhood may rupture into the peritoneum and excite peritonitis. Those most frequent in children are connected with Pott's disease, perinephritis, and cellulitis of the abdominal wall. It is occasionally seen in pyemia from any cause, and quite frequently occurs as one of the complications of septic sore throat.

Of the acute infectious diseases, peritonitis is most frequently seen with pneumonia, and very rarely with scarlet fever. It is also seen as one of the complications of septic sore throat. When secondary to pneumonia, there is usually intense pleurisy and sometimes also pericarditis and meningitis; in other words a general pneumococcus infection is present.

The bacteria most frequently associated with acute peritonitis in children are: the streptococcus, especially in the newly born; the pneumococcus in cases complicating pneumonia or empyema; and the streptococcus associated with the *b. coli communis* in those following intestinal perforation.

Lesions.—In the fibrinous form there are changes similar to those occurring in inflammation of the pleura and the other serous membranes. The peritoneum is injected and fibrin is thrown out in considerable quantity, usually accompanied by a small amount of serum. The process is usually a localized one. The peritoneum lining the abdominal wall, as well as that covering the adjacent coils of intestine and the solid viscera, is covered by patches of yellowish-gray fibrin, causing adhesions between the various viscera and often matting the intestines together. In recent cases these adhesions are soft, and easily broken down; in old cases they are quite firm, and they may result in the formation of connective-tissue bands which are the source of subsequent trouble. In

other cases the serum is more abundant, usually clear, but it may be turbid or even bloody.

In the purulent form the products are serum, fibrin, and pus. When peritonitis results from perforation it is, as a rule, purulent from the outset, and the pus is foul and stinking. The amount of pus is proportionally larger than in adult cases. When the disease proves fatal in a few days there is found an extensive exudation of fibrin, with the formation of small pockets containing pus among the coils of intestine. Occasionally there may be larger collections of pus in the peritoneal cavity. In cases which have lasted a long time—generally those of localized inflammation—the process results in the formation of a peritoneal abscess. This consists in a collection of pus in some part of the peritoneal cavity, the situation depending upon the cause, but it is usually in one iliac fossa or in the pelvis. The abscess is shut off from the rest of the peritoneal cavity by a thick wall of fibrin. If left alone, such abscesses may open into the rectum, vagina, bladder, pelvis of the kidney, or externally—usually at the umbilicus. After the discharge of pus the cavity may contract and fill up by granulation, and the patient recover.

Inflammations of the other serous membranes, especially the pleura, are often associated with peritonitis.

Symptoms.—The symptoms of acute peritonitis in older children, as in adults, are usually well marked and sufficiently characteristic to enable one to recognize the disease easily; but not so in the case of infants. In them the symptoms are often obscure, and the disease may be found at autopsy when not suspected during life. The onset is nearly always abrupt, with fever and vomiting. As a rule, the temperature is high—from 103° to 105° F. Vomiting may occur only at the onset, but it often continues; the vomited matters are usually green. Older children complain of pain, which may be localized or general, and in younger ones this is indicated by crying and fretfulness. The abdomen very soon becomes swollen and tympanitic, this being one of the most constant features of the disease. The distention is generally uniform, but it may be irregular. There is tenderness on pressure, and usually marked rigidity of the abdominal walls. The pain causes the child to assume a fixed position and he cries if moved or disturbed. The posture is generally dorsal, with the thighs flexed. The bowels are in most cases constipated, but diarrhea is by no means rare. The abdominal distention causes dyspnea and thoracic breathing. There may be retention of urine or frequent micturition.

The general symptoms, almost from the beginning, are those of a serious disease. The pulse is small, rapid, and compressible. The prostration is great, from the very outset. The face is pinched, the

mouth is drawn, and the features indicate pain. In severe cases there may be hiccough, cold extremities, clammy perspiration, and collapse. The mind is usually clear. In infants there may be convulsions. A polymorphonuclear leucocytosis is almost invariably present, but is wanting in some cases of the gravest type.

In the most severe forms of general peritonitis the course is short and intense, and the disease goes on rapidly from bad to worse until death occurs. In infants this is often on the third or fourth day. The very severe forms of general peritonitis in older children run the same rapid course. In other cases the course is slower, lasting a week or ten days. If the patient lives longer than this the case is more hopeful, because the process is more apt to be localized. The development of peritoneal abscess is indicated by the continuance of the temperature, which may assume a hectic type, and be accompanied by chills and sweating. There are the local signs of an abdominal tumor.

Prognosis.—Acute general peritonitis, whatever its cause, is a very serious disease in childhood. Of eighty cases of all varieties under sixteen years of age, sixty-nine per cent were fatal. In the newly born and in infancy the disease is almost invariably fatal. In older children the outlook is not quite so hopeless, and depends upon the exciting cause.

Treatment.—The medical treatment of acute general peritonitis in children is extremely unsatisfactory, as the disease is almost always fatal unless it can be relieved surgically. Opium is indicated only for the relief of the single symptom, pain. It has, however, serious disadvantages in that it may mask important symptoms. Other medical treatment is symptomatic only and is to be employed in conjunction with surgical measures.

As a local application cold is usually to be preferred. It may be applied either by an ice-bag or by a Leiter's coil. If children rebel against the use of cold, heat may be substituted. Turpentine stupes may aid in relieving tympanites.

Feeding is always a difficult matter on account of the strong tendency to vomit; this is due to regurgitation from the intestine into the stomach, which in some cases is almost continuous. In such conditions great benefit may be obtained from washing the stomach shortly before feeding, repeating this several times each day. In this way vomiting may often be controlled and the stomach made ready for food. The diet should be milk, broth, or buttermilk.

In every case of acute peritonitis an immediate exploratory operation should be done if the child's general condition will permit. Appendicitis is often found to be the cause when least expected; and even when the peritonitis is due to some other cause operation gives the only chance

for recovery. Operation is also indicated in localized inflammations with the formation of peritoneal abscesses.

CHRONIC (NON-TUBERCULOUS) PERITONITIS

Peritonitis may occur in fetal life with the production of extensive adhesions, which may interfere with the development of the intestine and result in various malformations. These cases have been ascribed by Silbermann to syphilis.

Chronic peritonitis may follow the acute form, in which there are left adhesions which slowly increase owing to the production of new connective tissue. Such cases are sometimes chronic from the beginning.

The peritoneal abscesses which follow the suppurative form may run a chronic course. Chronic localized peritonitis may occur in connection with disease of any of the organs covered by the peritoneum.

Chronic Peritonitis with Ascites.—In most cases this is chronic from the outset and independent of the causes above mentioned. By far the most frequent form of inflammation is that due to tuberculosis, and by some writers the opinion is still held that chronic peritonitis with ascites is always tuberculous. After the observations reported by Henoch, Vierordt, Fiedler, and others, there seems to be little room for doubting the existence of a chronic non-tuberculous form of peritonitis with ascites, although it must be considered a rare disease.

Etiology.—Nearly all the cases thus far reported have occurred in children over six years old. The causes are for the most part obscure. Chronic peritonitis may be associated with disease of the intestines or the solid viscera of the abdomen, especially with new growths of the kidney, liver, etc.

Lesions.—The post-mortem observations thus far have been few. In the reported cases there has been found a large amount of greenish serum in the general peritoneal cavity, with a very moderate amount of fibrin and with adhesions, which are sometimes few and sometimes very numerous. Chronic pleurisy may be associated.

Symptoms.—The early symptoms are of a very indefinite character, but often nothing whatever is noticed until the swelling of the abdomen begins. The enlargement comes on rather gradually in the course of a few weeks. Pain is slight, or wanting altogether. There may be some abdominal tenderness. The abdomen is usually distended with fluid. The general symptoms are very few. In some cases there is a slight evening rise of temperature of one or two degrees. There may be general weakness, loss of appetite, and moderate anemia.

The usual course of the disease is for the fluid to remain for a time and then undergo slow absorption. In some instances there is no tendency to absorption of the fluid, the general health is gradually undermined, and the patients die from exhaustion or from some intercurrent disease. The diagnosis rests upon the presence of ascites, developing gradually without any signs or symptoms of disease in the heart, liver, or other organs. The points which distinguish it from tuberculous peritonitis are considered under that disease. The prognosis must be guarded on account of the difficulty in making a positive diagnosis from the tuberculous form.

Treatment.—The treatment is entirely symptomatic. The patient should be kept at rest, preferably confined to bed. When there is no tendency to absorption, and especially when the patient's general health begins to suffer, the fluid should be removed by paracentesis. If it continues to accumulate after repeated tapping, laparotomy may be performed, for in some cases this has the same beneficial effect as in tuberculous peritonitis.

TUBERCULOUS PERITONITIS

The peritoneum is quite frequently the seat of tuberculous inflammation in early life. It occurs especially between the ages of one and five years, but is infrequent during the first year. Of 100 cases observed by Still, the largest number were seen in the second year of life. In 255 autopsies upon tuberculous patients, most of them under three years old, of which we have records, the peritoneum was involved in 8.6 per cent; but in a majority of these the peritonitis was not the most important lesion nor the cause of death. Tuberculous peritonitis is apparently much more frequent in Europe than in this country. Thus, Still states that this was the cause of death in 16.8 per cent of his tuberculous patients under twelve years of age, and in 12 per cent of the deaths from tuberculosis under two years. In 105 autopsies, for the most part upon older tuberculous children, Ashby found the peritoneum involved in 36 per cent. In 883 collected autopsies upon tuberculous children of all ages, Biedert found the peritoneum involved in 18.3 per cent. These figures do not represent the number of cases of tuberculous peritonitis, as in many of them only a few miliary tubercles were present.

It is possible for peritonitis to occur as the primary lesion of tuberculosis, the bacilli entering by way of the intestine, causing no lesion of the mucous membrane; but in the great majority of cases it is secondary to tuberculosis of the intestine, the mesenteric glands, the pleura, or to that of more distant parts, such as the lungs, the bronchial glands,

etc. In a small number of cases there is a history of some local exciting cause, such as a fall or blow upon the abdomen. The bovine type of the tubercle bacillus is more frequently found in tuberculous peritonitis than in any other form of tuberculosis, possibly excepting cervical adenitis, which fact is strongly suggestive of milk as the source of infection.

Tuberculous peritonitis is usually associated with other abdominal lesions—tuberculosis of the mesenteric glands, intestinal ulceration, etc. It is very rarely acute, but usually occurs as a subacute or chronic disease.

The peritoneum may be involved as one of the lesions in acute or subacute general miliary tuberculosis. The lesions consist in a deposit of miliary tubercles, which are generally rather sparsely scattered over the peritoneum. The evidences of inflammation are very slight, or they may be absent altogether. These cases do not come under observation as cases of peritonitis, as there are no abdominal symptoms.

The principal anatomical and clinical varieties of tuberculous peritonitis are the ascitic and the fibrous forms.

The Ascitic Form.—This is much less frequent than the fibrous form. The peritoneum is thickly sown with miliary tubercles, both discrete and in conglomerate masses. They are found in the omentum and the mesentery, upon the surface of the intestines and the solid viscera. The peritoneum shows in varying degrees the changes of acute or subacute inflammation, with the production of a moderate amount of fibrin and a large amount of serum. In the most acute cases the fluid is in the general peritoneal cavity. In those of longer duration it may be sacculated. The fluid is usually abundant, but not excessive. It is most commonly a straw-colored serum, but it may be seropurulent, or even bloody. There are commonly other lesions of tuberculosis in the body, but they are usually less marked than those of the peritoneum.

Clinically, ascitic cases usually present the symptoms of a low grade of peritoneal inflammation. The onset is gradual, with indefinite general symptoms. There is usually some fever—100° to 101.5° F. There is general weakness, prostration, and some loss of flesh, but not rapid emaciation. Vomiting is not prominent, and pain and tenderness are often absent. There may be nothing distinctive until distention of the abdomen is seen. This at first is due to intestinal gas, but later to fluid, which may accumulate in sufficient quantity to fill the general peritoneal cavity. The bowels may be constipated or there may be diarrhea. In other cases there may be only a slowly developing ascites without any inflammatory signs, and the abdominal enlargement is practically the only symptom.

The ascitic form of tuberculous peritonitis may result fatally, death occurring from general tuberculosis or by slow exhaustion from the local disease; the duration under these conditions is usually from two to six months. At other times the fluid may gradually undergo absorption and recovery take place, or after absorption the fibrous form of inflammation may develop.

The Fibrous Form.—This is generally slower in its development and more chronic in its course than the ascitic form. There is a tuberculous inflammation, the products of which have undergone transformation to a greater or less extent into fibrous tissue. The most important feature of these cases is the production of extensive organized adhesions between the solid viscera and the intestines, between the intestinal coils, and between the intestines and the abdominal walls. The intestines may be compressed against the spine by bands.

These adhesions and their mechanical consequences are sometimes almost the only lesions present. In other cases there may be an accumulation of fluid, which may be sacculated or in the general peritoneal cavity. This may be serous, seropurulent, or purulent. The omentum may be greatly thickened. There are often present in the fibrous exudate covering the intestines, in the omentum, and in the mesentery, tuberculous deposits consisting of caseous nodules or larger caseous masses, which are frequently softened at the center. Tuberculous deposits are found upon the peritoneal surface of the intestine, and infiltrate the intestinal walls, often leading to perforation, and sometimes to fistulous communications between adherent intestinal coils. There may also be tuberculous infiltration of the abdominal walls, accompanied by cellulitis, resulting in abscesses, which may open externally, usually in the neighborhood of the umbilicus.

Clinically, these cases are distinguished by their slow, irregular course. They are the most chronic of all the forms. The onset is generally insidious, and fever is commonly absent. There is rarely vomiting. The bowels may be constipated or loose. For a long time the general health may remain good. The only characteristic symptom is the enlargement of the abdomen. In the early part of the disease this is chiefly from the tympanites, but later there may be some accumulation of fluid. It is rare that the inflammation remains entirely fibrinous. Ascites usually develops very slowly, but may be abundant. The adhesions of the intestines may give rise to irregularities in the outline of the abdomen. Ascites may be present for a time and then disappear spontaneously, and the general health may so improve that the patient is considered quite well. There may even be a permanent cure. In other cases, after symptoms have been absent for some time, relapses occur, and more fluid is poured out. In addition to these symptoms,

others are present depending upon the mechanical effects of pressure from the contracting adhesions. There may be more or less constriction of the intestine, pressure upon the vena cava, the renal or portal veins, the thoracic duct or its branches, or upon the stomach. These conditions may give rise to dyspeptic symptoms, emaciation, edema of the lower extremities, and albuminuria. In some cases tuberculous peritonitis is entirely latent, and it is discovered at autopsy when there have been either no abdominal symptoms during life, or only colicky pains of an indefinite character. The course of this form of peritonitis is slow and irregular; it generally lasts for from six to twelve months, although with intermissions and exacerbations it may extend over several years.

If softening and breaking down of inflammatory products take place, well-marked constitutional symptoms are usually present. These are partly from the peritonitis and partly from general tuberculosis. Fever is regularly present, the temperature usually ranging from 99° to 102° F., though it is occasionally much higher. There is progressive emaciation, anemia, prostration, and sweating. Diarrhea is frequent and the intestinal discharges may at times be bloody. The abdomen is large, but not so much distended as in some of the other forms; the superficial veins are frequently prominent. Ascites often can not be made out by percussion, even though fluid is present. Areas of dulness and tympanitic resonance are irregularly distributed. Nodular masses of various sizes and irregular shapes may be felt anywhere in the abdomen, but they are more frequently in the region of the umbilicus and in the right iliac fossa than elsewhere. The epigastric region may be occupied by a smooth, hard tumor—the thickened omentum—which may resemble the liver. There may be the signs of phlegmonous inflammation of the abdominal wall in the neighborhood of the umbilicus, and even an abscess, which, after opening, may leave a fistulous communication with the peritoneum. There are usually some signs of disease in the lungs, and the pulmonary symptoms may mask those of the abdomen. The course of the disease, when softening and breaking down have taken place, is steadily progressive, the usual duration being from three to six months. Death results from the pulmonary disease, from tuberculous meningitis, from exhaustion, and occasionally it is due to accidents associated with perforation.

Diagnosis.—The essential symptoms of tuberculous peritonitis are an enlarged abdomen, often with evidence of fluid, wasting, colicky pains, irregularity of the bowels, nodular masses in the abdomen, and usually slight but continuous fever. In young children chronic ascites with fever usually means tuberculous peritonitis. Pouting of the navel, with induration and redness about it, is suggestive, and any chronic abscess

in the neighborhood of the umbilicus is suspicious. If the abdominal effusion is sacculated instead of diffuse, the probabilities of peritonitis are much increased. If there are added physical signs pointing to disease of the lungs or the evidence of tuberculosis elsewhere, and a positive cutaneous tuberculin reaction, the diagnosis is almost certain. Cirrhosis of the liver is practically unknown in infancy and early childhood. When ascites is absent, tuberculosis of the peritoneum may be suspected if there are irregular nodules or masses in various parts of the abdomen, with tenderness, emaciation, colicky pains, and, in the later stages, fever. But fever may be absent for a long time, even though local symptoms are marked. The epigastric tumor due to omental thickening may be mistaken for the liver; but it generally extends quite across the abdomen, and the upper as well as lower border can often be felt. Fecal masses may resemble tuberculous deposits, but are removed by cathartics and enemata.

Abdominal paracentesis to establish the presence of fluid or to allow of its examination is not justifiable. The danger of injury to the intestines even when a considerable accumulation of fluid is present is too great.

Prognosis.—Tuberculous peritonitis is always a serious disease, but by no means a hopeless one. The younger the child as a rule the more rapid the progress of the disease and the worse the outlook. The prognosis is especially bad during the first three years of life; at this period most of the cases terminate fatally. Many cases occurring in older children recover spontaneously and entirely. The most hopeful ones are those with ascites. But even in the fibrous form some apparently complete recoveries take place, the adhesions disappearing by absorption to a degree truly remarkable. The most unfavorable cases are those in which there is strong evidence of the breaking down of tuberculous deposits, with continuous fever and wasting.

Treatment.—The general treatment of tuberculous peritonitis is similar to that of tuberculosis in other parts of the body. The essentials are, rest, which should be invariably in the recumbent position, a climate mild enough to permit the patient to remain out of doors the greater part of the time, and very careful attention to feeding, with the purpose of improving the general nutrition. Heliotherapy, or the direct exposure of the abdomen to the sun's rays, has been much vaunted as a remedy and merits a trial as it can be employed in conjunction with the measures just mentioned. Beginning with a few minutes' exposure the time may be gradually lengthened to two or three hours. Under general treatment a very considerable number of patients recover, especially those who are over three years old. Such a termination is more likely if the diagnosis has been made early and if the

disease is limited to the peritoneum. Drugs play but a small part in the treatment of these cases. The value of tuberculin in tuberculous peritonitis has not yet been established.

In cases not progressing favorably under medical treatment, the question of operation should be considered. This was for a number of years a very frequent procedure and was employed in almost all cases. The results were not, however, such as to make it advisable as a routine measure. Hygienic treatment alone accomplishes in general as much if not more. In certain circumstances, operation is advisable. The most favorable cases are those of the ascitic variety. It may be useful also with localized or general suppuration and for the relief of intestinal obstruction occurring in the course of the disease. Operation affords temporary relief in some cases when the distention is very great. In the fibrous form not much is to be expected from it. Operation may be done for the relief of recurring colicky pains due presumably to constriction by bands. The existence of other foci of tuberculosis does not contraindicate operation except when these are chiefly intestinal, or when there is advanced general tuberculosis. In deciding the question of operation, its unfavorable results should also be borne in mind. A not uncommon consequence is injury to the intestine from the breaking up of adhesions, which may result in fecal fistulae. For the surgical aspect of the treatment the reader should consult works upon surgery.

ASCITES

Ascites consists in an accumulation of fluid, usually clear serum, in the general peritoneal cavity. It is a symptom of the various forms of peritonitis, especially the chronic varieties described in the preceding pages. It may be due also to portal obstruction from cirrhosis of the liver, or pressure upon the portal vein by peritoneal adhesions or large lymphatic glands. It is occasionally seen in all forms of abdominal tumors. Ascites may occur in general dropsy from cardiac disease, or from any condition causing pressure upon the vena cava. It is also seen in the general dropsy of renal disease. A moderate amount of ascites is often met with in extreme anemia or leukemia.

Small accumulations of fluid in the peritoneal cavity are difficult of detection. Large amounts are generally easily made out. There is a uniform smooth distention of the abdomen and dilatation of the superficial veins, especially about the umbilicus. On palpation, the wave of fluctuation can be obtained by placing one hand against the abdomen upon one side and giving the opposite side a sharp tap. A similar wave may be felt when there is tympanitic distention. The two are, however,

distinguished by having an assistant make pressure with the edge of the hand along the linea alba while the test is being made; this obstructs the wave transmitted through the abdominal wall, but does not affect that through the fluid. On percussion in the sitting posture, there is dulness below and resonance above. When the patient is recumbent, there is resonance in the median line and dulness or flatness in the lateral portion of the abdomen.

The prognosis and treatment of ascites will depend upon its cause.

Chylous Ascites.—This term is applied to certain cases in which the abdominal fluid contains fat. The color may be milky-white or light brown, and the fluid, after standing, may have at its surface a thick, creamy layer. The amount of fat present has been as high as five per cent. This condition is rare in childhood. The exact pathology is as yet not well understood. In the cases which have thus far come to autopsy there has usually been found chronic peritonitis, sometimes simple, sometimes tuberculous. The lymph vessels in some of the cases have been empty, and often no obstruction of the lymph circulation could be discovered. The fat is believed by some to be derived from fatty degeneration of the products of chronic inflammation, but this seems hardly sufficient to explain the large amount of fat sometimes found. In some of the cases it has been due to a wound of the thoracic duct. The amount of fluid is frequently very large. The prognosis is usually bad, although Pounds has reported a case in a girl of ten years, where recovery followed laparotomy. Tuberculous peritonitis was present.

SUBPHRENIC ABSCESS

In the group of cases of localized peritonitis or peritoneal abscess, must be included subphrenic abscess. This is a rare condition in childhood, and consists in an accumulation of pus just beneath the diaphragm and above the liver. Its cause may be either in the thorax or in the abdomen. It may complicate acute pneumonia, usually of the right lower lobe, by a direct extension of infection through the lymph channels. Sometimes it has been associated with phthisical cavities. In the abdomen it results from the extension of some focus of suppuration, such as an abscess around the appendix or abscess of the liver. The accumulation of pus is sometimes very great, so that the diaphragm is crowded high into the thorax.

The symptoms and physical signs closely resemble those of empyema, and most of the cases have been operated upon with the belief that the surgeon was dealing with empyema. Meltzer has reported a case in a child of two years which followed pneumonia of the right base. At the

operation only a few drops of pus were found in the pleural cavity; but there was discovered a pinhole opening in the diaphragm, from which the pus had escaped, and a large subphrenic abscess. This was evacuated, and the patient recovered perfectly. Subphrenic abscesses may contain air; they are then likely to be mistaken for pneumothorax. These abscesses require incision and drainage like other forms of peritoneal abscess.

SECTION IV

DISEASES OF THE RESPIRATORY SYSTEM

CHAPTER I

NASAL CAVITIES

ACUTE RHINOPHARYNGITIS

(*Acute Nasal Catarrh—Coryza*)

Although the symptoms of acute nasal catarrh are chiefly nasal, the principal seat of the pathological process is the rhinopharynx.

Etiology.—Certain children are predisposed to attacks of acute nasal catarrh. This predisposition, as it sometimes extends to entire families, may be inherited; but more frequently it is acquired, and usually by the following mode of life: It is seen in children who get very little fresh air, because they are kept indoors unless the weather is perfect; who live in houses always overheated; whose sleeping rooms are kept carefully closed at night for fear they may take cold; who are for the same reason so overloaded with clothing that they can not engage in any active play without being thrown into a profuse perspiration. These conditions after a time result in a great sensitiveness of all the mucous membranes, but especially those of the nose and pharynx, which is much increased by residence in a damp, changeable climate. Young infants and those who are rachitic, are frequent sufferers from acute nasal catarrh. Attacks are often brought on by insufficient covering for the head, by wetting the feet, by cold and exposure, especially to street dust and the raw winds of winter and spring, accompanied by the dampness which occurs with melting snow. In susceptible children the exciting cause is often a very trivial one. A draught of cold air for a few minutes may be sufficient to excite sneezing and a nasal discharge. Atmospheric conditions are probably not the only cause of acute nasal catarrh. Microörganisms certainly play an important part. The staphylococcus, streptococcus and pneumococcus are commonly found associated with this condition, much less frequently the influenza bacillus.

Recent observations of Tunnicliff showed the presence of a new organism called the "bacillus rhinitis" in 98 per cent of the cases of acute rhinitis studied and in 66 per cent it was the only organism present. It is a Gram-negative anaerobic bacillus. Acute catarrh may be sporadic or epidemic; certain forms are contagious, being communicated by children using the same handkerchief, occupying the same bed or simply by close contact.

Acute nasal catarrh may be a symptom of measles, nasal diphtheria, or influenza, and it may accompany erysipelas of the face.

Symptoms.—In the mild form the changes in the mucous membrane of the nose are not great, and are usually secondary to those of the rhinopharynx, being in a large measure due to the discharge. There is redness and slight swelling. The nasal passages may be for the time quite occluded by the discharge, which is usually profuse, at first seromucous, and later mucopurulent. The symptoms may be very transient, sometimes passing away in a few hours, in which case there is only a vasomotor disturbance; or they may continue and develop into a true inflammation. The discharge may excoriate the nostrils and the upper lip. At the onset there is usually sneezing, and in infants often a slight fever.

In older children there is no rise of temperature except in the most severe cases. The obstruction to nasal respiration causes mouth-breathing, and the dryness and discomfort which result from it produce disturbed sleep, snuffling and difficulty in nursing, this being in severe cases almost impossible. The inflammation may extend to the lachrymal duct, involving the eyes in a mild conjunctivitis. The process often extends to the larynx and bronchi, with hoarseness and cough. There may be closure of the Eustachian tubes, causing deafness and otalgia. The chief complication for which the physician should watch is otitis.

The severe form in infants is often attended by marked constitutional symptoms; the temperature may be as high as 104° or 105° F. and sometimes fluctuates widely. The discharge soon becomes mucopurulent and is very profuse, pouring from the anterior nares and filling the pharynx. The cultures in this form frequently show the pneumococcus. Severe symptoms often continue for a week or more, the child being seriously ill. Complications are almost always present. In most cases there is cervical adenitis and otitis. If the child is a delicate one bronchopneumonia is apt to develop. Retropharyngeal abscess is not infrequently seen.

Diagnosis.—It is important to distinguish between a simple acute catarrh and one due to measles, influenza, nasal diphtheria, or hereditary syphilis. Measles and influenza usually cause more fever and general constitutional disturbance than does simple catarrh. Nasal diphtheria

may be present when there is only a profuse discharge tinged with blood. When such a discharge persists for two or three weeks this is always to be suspected, even though the constitutional symptoms may be very slight. The only positive means of excluding diphtheria is by cultures. A persistent acute nasal catarrh in a young infant should always suggest syphilis, and the patient should be carefully watched for the development of other symptoms.

Treatment.—A young child suffering from acute coryza should be kept indoors in a room with an even temperature of about 70° F., the bowels freely opened, and the amount of food somewhat reduced. The only drug which seems to have much influence upon the secretion is belladonna.

Useful local applications are liquid albolene, oleostearate of zinc, or alkaline sprays, such as Seiler's solution, to clear away the secretions. If the nasal obstruction causes great interference with respiration or nursing, epinephrin diluted with a saline solution may be used with a medicine dropper.

The upper lip and nostrils should be protected by vaseline or some simple ointment. Under no circumstances should irritating or astringent injections be given. In older children inhalations of spirits of camphor may be used with some advantage.

The severe cases require more active treatment. For most of them nasal irrigation with a warm saline solution is to be advised. This should be done as in diphtheria. After cleansing the rhinopharynx a few drops of a five-per-cent solution of argyrol may be dropped into the nostrils two or three times daily.

Prophylaxis consists in solving the perplexing question, so often put to the physician, of how to prevent children from "taking cold." This is a matter of the utmost importance, and follows what has been previously said under the head of Etiology. No amount of cod-liver oil and iron will remove this tendency to catarrh so long as bad hygienic conditions continue. Sleeping rooms should be large and well ventilated, and a window should be kept open at night, except in very severe weather or during acute attacks. The temperature of the house during the day should be kept from 65° to 68° F., but not above this. Children should be accustomed to go out of doors unless the weather is especially bad. So firmly rooted in the minds of the laity is the idea that acute catarrhs come from cold, that the habit of coddling delicate children is always likely to be carried to an extreme.

With every delicate and "catarrhal" child one should begin in the summer by having him live in the open air as much as possible, sleeping in a room with free ventilation, with moderate covering, and continuing the same practice into the fall and early winter. If begun

gradually in this way there is little difficulty in continuing throughout the winter.

The next point to be insisted on is cold sponging immediately upon rising in the morning, especially about the chest, throat, and spine. The use of chest protectors, cotton pads, and extremely thick clothing should be prohibited. Woolen underclothing should be worn upon the chest throughout the year, and upon the legs also in winter; the very lightest in summer, and only a medium weight in winter.

Frequently repeated attacks point to the presence of adenoid vegetations in the pharynx, and no measures are of much avail until these are removed.

CHRONIC NASAL CATARRH

This term is rather loosely used to designate a chronic nasal discharge. Such a discharge is common both in infancy and childhood. It is a condition frequently neglected by physicians. Patients are too often subjected to routine constitutional treatment by cod-liver oil and preparations of iodin, with the idea that such cases are "scrofulous," while local treatment is either neglected altogether, or consists only of the use of the nasal douche or syringing with a saline solution. Permanent damage to the organs of hearing, smell, speech, and respiration may result from neglecting or ignoring chronic nasal catarrh in childhood.

Chronic nasal catarrh is not to be regarded as a disease, but only as a symptom which may be due to any one of a variety of pathological conditions, each of which requires very different treatment, viz., adenoid growths of the pharynx, foreign bodies in the nose, polypi, deviation of the septum or any other congenital deformity of the nasal passages, the various forms of chronic rhinitis, and syphilis, which causes a form of rhinitis peculiar to itself.

Adenoid Growths of the Pharynx.—These are more fully discussed elsewhere. They are by far the most frequent cause of chronic nasal discharge in infants and young children, and should be first suspected. The nasal discharge accompanying adenoid growths is due to a chronic rhinopharyngitis. Treatment is without avail unless the growths are removed. After this is done the nasal discharge usually disappears quite promptly.

Foreign Bodies in the Nose.—This condition should be suspected whenever there is an abundant mucopurulent discharge limited to one nostril. Foreign bodies in the nose are quite frequent in young children. Peas, beans, beads, or shoe buttons are most frequently lodged there. The efforts at removal on the part of the child, or the parents, generally result in pushing the body farther into the nose. It first sets

up a mechanical irritation, accompanied by pain, swelling, sneezing, and sometimes hemorrhage. This is followed by a catarrhal inflammation which in the course of a few days becomes purulent and may last indefinitely. The discharge is generally quite abundant. The symptoms point to an obstruction of one nostril, and an examination with a probe readily detects the presence of the foreign body.

In recent cases the removal of the foreign body may sometimes be accomplished by compressing the empty nostril and having the child blow his nose strongly. Often the sneezing which the foreign body excites is sufficient to remove it. Before any attempt is made to seize the body with forceps, cocain should be used, not only for the purpose of preventing pain, but in order to contract the mucous membrane so as to allow better manipulation. In many cases general anesthesia is necessary. In most circumstances ordinary foreign bodies can with proper forceps be extracted without difficulty. No subsequent treatment is required, except the use of some mild antiseptic to keep the nose clean for a few days, as the inflammation quickly subsides after the removal of the cause.

Nasal Polypi.—These are among the infrequent causes of chronic nasal discharge in childhood. They are especially rare before the seventh year, but both mucous and fibrous polypi are seen. The symptoms are those of a chronic nasal catarrh with partial or complete obstruction of one or both sides. Polypi increase in size with the occurrence of every acute coryza, and are always especially troublesome in damp weather. They may be accompanied by reflex symptoms, such as cough, sneezing, and even by attacks of asthma. There may be headache, and sometimes disturbances of smell, taste, and hearing. The symptoms are of much longer duration than in the case of obstruction from a foreign body, the discharge is not so abundant, and is not purulent. The diagnosis is made only by local examination.

Polypi may be removed with the forceps, but this is best accomplished by the use of the wire snare. When they have been present for a long time the accompanying chronic rhinitis may require subsequent treatment.

Deviation of the nasal septum, and other congenital deformities which may cause narrowing of the nasal respiratory tract, are conditions which belong to the specialist.

CHRONIC RHINITIS

Simple Chronic Rhinitis.—Simple chronic rhinitis existing alone is of rare occurrence in young children. In the cases so classed the symp-

toms are usually due to rhinopharyngitis, which almost invariably depends upon adenoid growths. The growth may be a small one, so that the symptoms of obstruction are slight or absent. A frequent complication is chronic enlargement of the cervical lymph nodes.

The only constant symptom is an excessive nasal discharge which is usually mucous but which may be mucopurulent. It is easily removed by blowing the nose if the child is old enough to be taught to do this. Children too young to clear the nose in this way suffer from almost constant discomfort. The amount of discharge depends upon the severity of the case. It frequently causes irritation of the upper lip, which may be the seat of eczema or impetigo, especially in infants. The lip may be swollen and prominent. The condition of the external parts is aggravated by the constant disposition to pick the nose, which may be overcome by the application of a short anterior splint to each elbow.

Epistaxis sometimes occurs. The duration of the disease is indefinite; it may last for months or even for years, the symptoms in summer being insignificant, but returning every cold season. It may terminate in recovery, or, in children with flabby tissues and delicate constitution, it may be followed in later childhood by hypertrophic rhinitis.

Treatment.—Prophylaxis is important. The main purpose should be to prevent attacks of acute nasal catarrh by the measures mentioned in the discussion of that disease. The general treatment should not be routine, but based upon the indications of each case. General tonic treatment is required in most cases.

Local treatment consists first in cleanliness, and, secondly, in the use of astringents. In infants, if the discharge is abundant, an efficient method of getting rid of it is by nasal syringing. This is attended by some risk of forcing materials into the middle ear; but if very carefully done, the danger seems to be less than that of allowing the discharge to remain. All solutions are to be made with sterile water and used warm, either with a nasal douche or syringe. No force should be employed. Either Dobell's or Seiler's solution may be employed, diluted with an equal amount of water. Recently there have been introduced several devices for removing abundant secretion by means of suction, which obviate the risks attendant upon the syringe and are even more efficient. Ordinarily, the nose should be cleansed thoroughly twice a day, more frequently in very severe cases. Harm is often done by the overzealous use of local treatment in these conditions.

Syphilitic Rhinitis.—Rhinitis is seen both in early and late hereditary syphilis. Coryza, or snuffles, is one of its earliest and most constant symptoms. It usually begins between the third and sixth weeks of life, rarely after the third month. The pathological condition is a subacute catarrhal rhinitis, sometimes with the formation of superficial

ulcers or mucous patches. The disease is usually attended by a profuse nasal discharge of seromucus or mucopus, occasionally tinged with blood. It may continue from a few weeks to two or three months. It usually requires only constitutional treatment and protection of the nostrils and lips by the use of the ointment of the yellow oxid of mercury diluted with four parts of vaseline. When the discharge is very abundant any one of the cleansing solutions previously mentioned may be used as a spray.

The rhinitis of late hereditary syphilis is a very different pathological condition. There are here gummatous deposits which break down, and form ulcers of the mucous membrane and deeper tissues. There is also periostitis, with extension of the disease to the cartilages and bones of the nasal fossae, particularly of the septum. There may be perforation of the triangular cartilage, necrosis of the vomer or nasal bones, perforation of the hard or soft palate, and at times extensive ulceration of the alae nasi and the face. Cicatrization may follow, causing stenosis of the nostril. These lesions in the nose are generally accompanied by deep ulceration of the pharynx and soft palate. They usually occur in children who have presented the early symptoms of hereditary syphilis, but are occasionally seen when no such history can be obtained. Such was the case in a patient recently under observation in the Babies' Hospital, who had perforation of the nasal septum and of the floor of the nasal fossae, causing a free communication with the mouth. These are cases of true ozena. The odor from the discharge is at times almost intolerable. When neglected these cases go on from bad to worse and may continue for years, producing unsightly deformities.

The constitutional treatment is that of hereditary syphilis in general and is discussed in the chapter upon that disease.

Locally there may be used a spray of one of the cleansing solutions already mentioned, or black wash, or a solution of bichlorid of mercury, 1 to 10,000. Although improvement may take place quite promptly, the results of treatment in the late cases are often unsatisfactory, as the disease has usually progressed so far before treatment is begun that some deformity of the nose results, usually a sinking in of the bridge and flattening of the alae, giving rise to the so-called "saddle-back" deformity.

EPISTAXIS

The hemorrhage may come from any part of the nasal fossae, but it is generally from the anterior nares, and most frequently from the vessels of the septum. Epistaxis is a rare symptom in the hemorrhages of the newly born, and when present suggests syphilis. It is infrequent

throughout infancy, but in childhood it is quite common, occurring in boys more frequently than in girls. In the latter it is especially common about the time of puberty. Children who are kept much indoors in overheated apartments, and who have susceptible mucous membranes and flabby tissues, are particularly prone to it. The exciting cause may be a local one, like a fall or blow; epistaxis may be due to picking the nose, or to any kind of mechanical irritation; it may be associated with nasal catarrh; and it is often caused by a small ulcer upon the septum. An attack may be brought on by mental or physical excitement. It occurs as an occasional, often an early symptom, in typhoid or malarial fever, in measles, or during severe paroxysms of pertussis. It is seen in the hemorrhagic form of all the eruptive fevers, in certain cases of diphtheria, in hemophilia and scorbutus, in grave anemia, leukemia, and in diseases of the heart and blood vessels.

Symptoms.—Epistaxis is frequently preceded by a sense of fulness or pain in the head, which is relieved by the bleeding. The blood is usually from one nostril, and comes slowly by drops. The amount lost is generally small, but it may be large enough, when repeated, to produce a serious grade of anemia even in strong children; the hemorrhage may even prove fatal. Epistaxis may be overlooked if the blood finds its way into the pharynx and is swallowed. In most of the cases the hemorrhage ceases spontaneously in from ten to twenty minutes, recurring at longer or shorter intervals, according to the nature of the cause. Hemorrhage from adenoid growths of the pharynx may closely resemble that from the nose, but otherwise there can rarely be any difficulty in recognizing epistaxis.

Prognosis.—This depends upon the cause. In the great majority of the so-called idiopathic cases epistaxis is not serious. Occurring early in the course of one of the infectious diseases, it does not ordinarily affect the prognosis unless it is very severe. When it occurs late, however, it is always a bad sign, and particularly so in diphtheria. It may be serious in any of the hemorrhagic diseases or in diseases of the blood, when it is not infrequently a cause of death.

Treatment.—To remove the predisposition, a child should receive general tonic treatment, especially plenty of outdoor exercise, and every means should be taken, by the use of cold baths, friction, and proper food, to tone up the vascular system.

An efficient means of arresting the hemorrhage is compression of the nose between the thumb and finger. This may be combined with the application of ice over the nose, and sometimes small pieces of ice may be introduced into the nostrils. The application of cold to the back of the neck or its use in the mouth may be of service by exciting reflex contraction of the capillary vessels. All tight clothing or bands about

the neck should be loosened, and the patient kept quiet in the sitting posture. After the hemorrhage has ceased the child should not blow his nose for some time. Epinephrin is one of the most efficient local means of checking the bleeding. Another valuable remedy is the peroxid of hydrogen, used full strength. If bleeding continues in spite of all the above measures, the anterior nares should be plugged, and if this does not control it, the posterior nares should be plugged. Usually very little effect is seen from drugs given internally, although in frequently recurring hemorrhages where no local cause can be discovered, calcium lactate should be tried; from thirty to sixty grains a day should be given to a child of five years.

The subcutaneous use of horse serum often has a very decided effect in controlling these hemorrhages which do not yield readily to the usual treatment. From 20 to 30 c.c. may be given to a child of five years and repeated every few hours if bleeding continues. Human serum is even more efficacious. In very severe hemorrhages transfusion may be necessary. In severe cases of nasal hemorrhage recurring at short intervals without any apparent cause, ulcer of the septum should be suspected, and, if present, should be touched with chromic acid.

CHAPTER II

DISEASES OF THE LARYNX

The characteristic feature of laryngeal disease in infants and young children is the association of muscular spasm with every form of inflammation. Often it is the laryngeal spasm, rather than the inflammation, which gives rise to the principal symptoms. This spasm is only one expression of the great reflex irritability of young children.

CATARRHAL SPASM OF THE LARYNX

(Spasmodic Laryngitis; Spasmodic Croup; Catarrhal Croup)

The term *catarrhal spasm* is fairly descriptive of this disease, which is characterized by a very mild degree of catarrhal inflammation associated with marked laryngeal spasm.

Etiology.—It is not often seen during the first six months, but is frequent from this time up to the third year. After five years it is rare. It occurs rather oftener in children who are well nourished. Certain chil-

dren have a predisposition to such attacks, those who have had one attack are likely to have others. The condition has many points of resemblance to spasmodic asthma which may replace it in later childhood. Heredity seems to have some influence in producing this extreme susceptibility of the air passages. Catarrhal spasm of the larynx is very frequently associated with enlarged tonsils and adenoid growths of the pharynx, sometimes with an elongated uvula. The exciting cause may be exposure to cold, especially to high winds, or an attack of indigestion. There is no doubt that catarrhal spasm of the larynx is seen at the present time much less frequently than formerly; the reason for this is not clear.

Lesions.—The catarrhal inflammation of the larynx affects chiefly the parts above the cords; there is congestion and dryness, and later increased secretion of mucus. To this there is added a spasm of the muscles of the larynx. There is no submucous infiltration, and no tendency to edema of the glottis.

Symptoms.—The attack may be preceded for several hours by slight hoarseness, or by a nasal discharge. During the day the child may appear perfectly well. Usually there is heard during the evening a hollow, barking cough, at first infrequent and not severe. About midnight this is apt to increase in severity, and there is now difficulty in breathing. As soon as this becomes marked the child wakes, and presents the characteristic symptoms of an attack. In the mild cases the dyspnea is not sufficient to waken the child. In severe cases there is marked dyspnea, especially on inspiration, and a loud stridor as the air is drawn through the narrowed opening of the glottis. This may often be heard in an adjoining room. There is seen on inspiration deep recession of the suprasternal fossa, the supraclavicular spaces, and the epigastrium; also depression of the intercostal spaces, and even of the walls of the chest. Any excitement increases the spasm and aggravates the dyspnea. The distress may be great; the breathing usually slow and labored; the voice hoarse, but rarely lost; the cough stridulous, hoarse, and metallic; the pulse rapid; the temperature normal or slightly elevated, rarely over 101° F. There may be slight lividity of the fingertips and of the lips, and sometimes considerable prostration. In the course of three or four hours the attack slowly wears away and the child falls asleep. During the following day, aside from slight hoarseness and occasional cough, he is apparently well. Most of the cases are not so severe as this; there are the croupy cough, the hoarseness and general discomfort, but not marked dyspnea. On the second night there is a repetition of the experience of the first, usually quite as severe unless affected by treatment; and on the third day a remission similar to that of the day previous. On the third night the attack, if it occurs at all, is generally a mild one. Slight hoarseness persists for several

days, but otherwise the child is apparently well. Many children have such attacks every few weeks in the course of the cold season, the slightest exposure or an indiscretion in diet being sufficient to induce one.

Prognosis.—This is good, the disease never proving fatal, although nothing is more alarming, at least to parents, than to witness for the first time one of these severe attacks of catarrhal croup.

Diagnosis.—Catarrhal spasm may be confounded with laryngismus stridulus, acute catarrhal laryngitis or with membranous croup. Laryngismus stridulus occurs only in infancy. In it there is not simply stridulous breathing, but periods of complete arrest of respiration. These may be repeated many times during the day, and may continue for weeks, being often complicated by carpopedal spasm, sometimes by general convulsions.

From acute catarrhal laryngitis and membranous laryngitis, catarrhal spasm is distinguished by its sudden onset, the mildness of the symptoms of inflammation, the spasmodic character of the dyspnea, and the daily remissions. The history of previous attacks will often aid in diagnosis. In case of doubt, a positive diagnosis can often be made by allowing the child to inhale a little chloroform. This at once relieves dyspnea due to spasm, while it has scarcely any effect upon that due to inflammation or membrane.

Treatment.—The purpose of treatment during the attack is to produce relaxation of the laryngeal spasm. This is accomplished by the use of emetics, steam, and hot fomentations over the larynx. To produce vomiting, ipecac is the safest drug. This may be given in the form of the syrup, one-half teaspoonful every ten or fifteen minutes to a child of two years until vomiting occurs, or it may be combined with ten or fifteen drops of the wine of antimony. The latter should not be repeated more than once or twice as it may produce serious depression. When given at longer intervals these remedies are useful in relaxing spasm without causing emesis.

Emetics have a double value if the attack is due to indigestion. If there is constipation, an enema should be given. Following the free vomiting there is generally some improvement in the symptoms, but there may be a recurrence of the spasm unless other means are employed. To prevent this, antipyrin is one of the most useful drugs. One grain may be given to a child one year old. This may be repeated every two hours if necessary. Quite as much relief as that obtained from the drugs mentioned is seen from the use of steam inhalations. For this purpose the child should be placed in a closed tent, and steam introduced from a croup kettle. This may be used in conjunction with other measures, and continued as long as necessary. Poultices or hot fomentations over the larynx are also useful. In one case in which severe

spasm had recurred for eight successive nights in spite of everything that was tried, the child being in great distress from the dyspnea, intubation was performed with instant relief. Tracheotomy, however, would scarcely be advisable.

During the day following the first night attack, the child should be kept in a warm room, and it is well to continue the ipecac in doses too small to produce vomiting. After 6 P.M. the doses should be doubled, and at bedtime two grains of antipyrin given. If so treated, the symptoms may not recur upon the second night, or there may be only the cough without the severe dyspnea. The child should be confined to the house for two or three days after one of these attacks, the drugs being gradually reduced; but the antipyrin should be given at bedtime for three or four successive nights.

To prevent a repetition of the attacks and remove the tendency to them, it is most important that the child should have plenty of fresh air and cold bathing, especially cold sponging about the neck and chest. Everything which experience has shown to bring on the attack should be carefully avoided. Local causes, such as adenoid growths and hypertrophied tonsils, should receive appropriate treatment. Generally it is not necessary to exclude fresh air from the sleeping room. Although an open window on a cold, damp night may sometimes excite an attack, plenty of fresh air regularly given tends rather to diminish the susceptibility. If the child's condition is poor, general tonic treatment is to be employed.

ACUTE CATARRHAL LARYNGITIS

Acute laryngitis is not so frequent as the disease just described, although it is much more severe, and may even be fatal. It occurs especially in children from one to five years of age, usually in the cold season. Predisposition to attacks is induced by the same conditions as in the case of acute rhinitis. Catarrhal laryngitis may be primary, when it is usually excited by cold or exposure,[1] or it may be secondary to measles, influenza, scarlet fever, or other infectious diseases. It may also be of traumatic origin, from the inhalation of steam or irritating gases.

[1] The following case is a good illustration of a severe attack excited by cold: A rather delicate infant, eight months old, was taken out, with very scanty covering, on a raw December day. In a few hours hoarseness and stridor were noticed, and the temperature was 101° F.; three hours later it was 103° F., and in spite of the usual remedies which were employed the dyspnea had reached such a degree as to require intubation. The tube was worn only three days and the child made a prompt recovery.

Lesions.—There is a moderately intense congestion of the laryngeal mucous membrane, sometimes general and sometimes localized. This may be seen with the laryngoscope, but is not always visible after death. With the congestion there are swelling and dryness, followed by increased secretion. In the milder cases the process is limited to the mucosa. In the more severe cases it involves the submucosa also, which is congested, edematous, and may be infiltrated with cells. The changes are especially marked in the lymphoid tissue of the subglottic region. The swelling may be sufficient to produce a very marked degree of laryngeal stenosis. In many mild and in all the severe cases there is associated catarrhal inflammation of the trachea, and often of the larger bronchi. In young children there is very little tendency to edema of the glottis.

Symptoms.—In the mild form, such as that which is usually seen in older children, there is hoarseness, or even loss of voice, and a laryngeal cough which is sometimes hard and teasing and always worse at night. There may be pain and soreness over the larynx. Constitutional symptoms are mild or absent, the patient not usually being sick enough to go to bed, and often rebelling even at being kept indoors. The duration of the disease is from four to ten days, with a strong tendency to relapses from slight causes.

The severe form of catarrhal laryngitis is sometimes preceded by acute coryza, or there may be mild laryngeal symptoms for a few days before the development of the more severe ones. In other cases the disease develops rapidly and severe symptoms are present within a few hours from the onset.

When the case is fully developed the voice is metallic and hoarse, and occasionally but not usually lost. There is a hoarse, dry, barking cough, which is very distressing, and sometimes almost constant. The cough, like the voice, is stridulous, and more or less stridor is present on inspiration. There is a slight amount of constant dyspnea, but this is scarcely noticeable unless the chest is bared. Severe dyspnea occurs in paroxysms, usually at night. Then, we may get the signs of obstructive dyspnea similar to those mentioned in severe attacks of catarrhal spasm. This dyspnea is chiefly inspiratory, but in some cases it increases steadily from the beginning of the attack, and may be indistinguishable from that due to membrane. Constitutional symptoms are usually present and may be severe. The temperature ranges in most cases from 101° to 103° F., but may go to 104° or 105° F. The pulse is rapid and full and respiration is accelerated. Children sometimes complain of pain in the larynx and trachea which is increased by coughing. The symptoms are severe for two or even three days, the fever continuing with moderate prostration and paroxysms of dyspnea, sometimes even attacks of suffocation and cyanosis. Usually after two or three days there is a grad-

nal subsidence of the dyspnea and the inflammatory symptoms, and the case goes on to recovery. At other times the inflammation extends downward to the large and then to the small bronchi, and finally results in bronchopneumonia. The attack may prove fatal from laryngeal obstruction due to swelling and spasm.

Diagnosis.—This disease is chiefly to be distinguished from membranous laryngitis. The onset of the two diseases may be very similar, and for the first twelve hours we have no absolute means of distinguishing between them, except possibly by the use of the laryngoscope, which is often conclusive in older children but not usually so in infants. All cases, therefore, should be looked upon with a degree of apprehension. The temperature in the catarrhal is usually higher than in the membranous form. The dyspnea is mainly paroxysmal, with daily remissions and nightly exacerbations, and is chiefly inspiratory, while that of membranous laryngitis is constant, steadily and often rapidly increasing, and is present both on inspiration and expiration. In catarrhal laryngitis the voice is not usually lost, but in the membranous form this is the rule. There can be little room for doubt when there are enlarged glands, membranous patches on the tonsils, and nasal discharge. Very often, however, all these evidences of diphtheria are wanting, the really difficult cases being those in which the process begins in the larynx. The prevalence of diphtheria and a known exposure count for something in favor of membranous laryngitis. If cultures from the pharynx show the presence of Klebs-Loeffler bacilli, diphtheria of the larynx is certain; but no conclusions can be drawn from negative cultures. In catarrhal as well as in membranous laryngitis there may be extreme dyspnea, cyanosis, pallor, prostration, and even death.

Prognosis.—This depends somewhat upon the cause of the disease and also upon the age of the patient. It is much worse when it is secondary to measles or scarlet fever. It is better in children over three years of age than in infants, also when the general condition of the child is good. The prognosis in severe catarrhal laryngitis should always be guarded, not only on its own account, but also because it is impossible at first to be certain that the case is not one of membranous laryngitis.

Treatment.— In all cases children affected are to be kept in bed, and the temperature of the room should be between 70° and 72° F. The diet should be light and fluid, and the bowels should be freely opened. A hot mustard foot bath should be given at the outset. Antipyrin (one grain every two hours to a child two years old) is useful if there is much spasmodic dyspnea. For this symptom emetics are beneficial, given as in catarrhal spasm. The use of ipecac and squills in smaller doses than is required for emesis (five drops each of the syrups of ipecac

and squills every two hours) may give relief, especially in the early stage, when the cough is dry, hard, and severe.

All the remedies mentioned are to be regarded as accessories to the essential treatment, which consists in the use of inhalations. The child should be placed in a tent into which steam is introduced from a croup kettle. Simple steam may be used, or pine needle oil, compound tincture of benzoin, lime-water, or creosote may be added. In moderately severe cases inhalations should be used for fifteen minutes every two hours; in very severe ones they should be continued the greater part of the time. Poultices or hot fomentations may be applied over the larynx. Relief is sometimes obtained by using counter-irritation by mustard. In our experience the local use of cold is very unsatisfactory, on account of the difficulty of applying it properly, and the objection to it on the part of young children. Stimulants may be required late in the disease, the amount of prostration being the guide to their use.

In cases of extreme dyspnea operative interference may be needed. It is required more often in infants and young children than in those who are older. Opinions will of course differ as to when the dyspnea has reached the danger point. One should not wait for general cyanosis. If pallor, marked prostration, and steadily increasing dyspnea are present the case should not be allowed to go on without interference, even though one may be perfectly sure that it is one of catarrhal inflammation only. The severity of the dyspnea is the only guide; cases at autopsy may turn out to be catarrhal, which were regarded during life as undoubtedly membranous. If intubation is done, the tube can generally be dispensed with in two or three days. Convalescence is usually rapid, but there is danger of recurring attacks during the remainder of the cold season.

SUBMUCOUS LARYNGITIS—EDEMA OF THE GLOTTIS

These two conditions are not quite identical, although they are closely associated and may be conveniently considered together. They are both rare in early life. In true edema of the glottis there is simply a dropsical effusion into the submucous cellular tissue of the aryteno-epiglottic folds, causing them to project as large rounded swellings on either side of the superior isthmus of the larynx. They may be of sufficient size to cause serious or even fatal obstruction to respiration. With the laryngoscope they appear as pale-red tumors, lying usually in contact near the base of the tongue. By the finger their presence can be quite readily distinguished. Edema of the glottis occurs principally in the late stages of nephritis.

In the inflammatory form of edema, or true submucous laryngitis, there is the same sort of swelling of these structures, but in this case it is due to some active inflammation in the neighborhood. The swelling is partly from the edema and partly from cell infiltration. Usually all the parts surrounding the upper opening of the larynx are in a state of acute inflammation. The epiglottis may be swollen to the thickness of a finger and easily seen by depressing the tongue.

The exciting causes may be the mechanical irritation of a foreign body, the inhalation of steam or irritating gases, erysipelas of the neck, primary catarrhal laryngitis, or retropharyngeal abscess.

The symptoms consist of great inspiratory dyspnea with attacks of suffocation, while expiration may be quite easy. In true edema there are in addition the symptoms of the primary disease. In the inflammatory form there are the evidences of local inflammation—hoarseness, cough, pain, and difficulty in swallowing. A positive diagnosis may be made by a digital examination. The symptoms may develop with great rapidity in either variety, and frequently prove fatal in a few hours.

The treatment of true edema consists in scarification or multiple puncture, the application of ice externally, and even the swallowing of ice; in the inflammatory form, in addition, local blood-letting by leeches and, as a last resort, tracheotomy. Intubation is useless in either form.

CHRONIC LARYNGITIS

The following varieties are seen: (1) A simple form usually associated with adenoid vegetations of the pharynx; (2) tuberculous; (3) syphilitic; (4) that associated with new growths.

1. **With Adenoid Growths of the Pharynx.**—This is not uncommon. A slight superficial catarrhal inflammation develops, the symptoms of which may continue for many months. These cases are often treated for a long time unsuccessfully by the use of sprays, inhalations, etc., but the symptoms disappear rapidly after the removal of the adenoid growths. Similar symptoms may be associated with hypertrophic rhinitis. In this also the treatment should be directed to the primary condition.

2. **Tuberculous Laryngitis.**—This belongs to later childhood, and is rare even then. In infancy it is almost unknown. Rheindorf has reported a case in a child of thirteen months, which was regarded during life as syphilitic, but was shown by autopsy to be tuberculous. Of sixteen cases in children, reported by Rilliet and Barthez, none occurred during the first three years, and only four before the seventh year. The

larynx alone may be affected, or the larynx and trachea, or the larynx, trachea, and lungs. Pulmonary tuberculosis is usually found to be present at autopsy, even though there may have been no pulmonary symptoms. Demme has reported a case of tuberculous laryngitis in a boy of four years whose lungs were healthy, death resulting from tuberculous meningitis.

The symptoms are hoarseness, aphonia, laryngeal cough, and mucopurulent, sometimes bloody, expectoration. The sputum may contain tubercle bacilli. With the laryngoscope tuberculous deposits may be seen, but more frequently there is tuberculous ulceration of the mucous membrane. In children this is usually superficial, the deep destructive ulceration seen in adults being very rare.

It is to be differentiated from syphilis chiefly by the general symptoms, as the laryngoscopic appearances may be very similar. Local treatment is seldom necessary and only with older children. It should be in the hands of a specialist.

3. **Syphilitic Laryngitis.**—In the early stage of syphilis the larynx is often the seat of a catarrhal inflammation, which presents nothing especially characteristic except its protracted course. The laryngitis of late hereditary syphilis is quite rare, and is likely to be overlooked because of the difficulties in the way of a thorough examination, and because the disease is usually painless.

Strauss has collected fourteen cases between the ages of three and fifteen years, and added three of his own. He states that deep seated processes are much more rare than among adults. The parts most frequently affected are, first, the epiglottis; secondly, the aryteno-epiglottic folds; thirdly, the posterior laryngeal wall. The epiglottis was involved in twelve of fourteen cases. Usually there was only perichondritis; in the more severe cases there was partial or complete destruction of the cartilage. In four cases papillomatous masses were seen. In five cases the process extended from the epiglottis to the epiglottic folds of one or both sides. In several instances the superior vocal cords were thickened from hyperplasia, and occasionally small tumors were formed. In only one case was there ulceration of these folds. Changes in the vocal cords and the arytenoid cartilages were rare, occurring only with extensive inflammation. The symptoms are those of chronic laryngitis: hoarseness, sometimes aphonia, and in a few cases chronic laryngeal stenosis. The diagnosis can be made only by means of the laryngoscope. In most of the cases there are present ulcerations of the palate or uvula, or scars from previous ulcers; sometimes the disease extends into the nose. Serious symptoms often result when to old syphilitic lesions there is added acute laryngitis or edema.

In addition to the usual constitutional remedies for syphilis, and

to the means ordinarily employed for the relief of chronic laryngitis, intubation may be required in these cases for the relief of laryngeal stenosis. The tube must usually be worn for many months.

NEW GROWTHS

New growths of the larynx are not very rare in children. Excluding the granulations which follow the use of the tracheal canula, the only one that is likely to be met with is papilloma. This may occur even in infancy. According to Rauchfuss, the majority of the cases begin during the first year. Boys are more frequently affected than girls.

The symptoms depend upon the size and location of the tumor. The earlier manifestations are usually ascribed to chronic laryngitis. There is hoarseness, sometimes loss of voice, and a paroxysmal cough; later, dyspnea develops which often increases by paroxysms. The symptoms are slowly progressive, and it may be several months before they are sufficiently severe to attract special attention. A positive diagnosis is made only by the laryngoscope. There is seen a whitish granular tumor or tumors, sometimes pedunculated, sometimes with a broad base, which may be attached to any part of the larynx. The prognosis is usually serious on account of the danger of bronchopneumonia after operation.

The treatment of these cases belongs to the specialist. Operative removal of these papillomata usually results in their recurrence in increased numbers. For this reason operations through the mouth have been largely given up. Papillomatous tumors will often disappear entirely if complete rest for the larynx is secured by means of tracheotomy; but the tube must be worn for from six months to a year. Radium has been used in a few instances with brilliant results, the tumor disappearing after a single application and not recurring; but extensive cicatrization has also been reported.

FOREIGN BODIES IN THE LARYNX AND BRONCHI

The aspiration of foreign substances into the larynx is not an uncommon accident in children. It usually happens from an attempt to cough, laugh, or cry while the child has something in his mouth. If the body is sharp and irregular, like a pin, the shell of a nut, or a fragment of bone, it is liable to become impacted in the larynx. If smooth, like a pea or a bead, it is usually drawn into one of the bronchi, generally the right.

When the body enters the larynx there is immediately excited a

violent paroxysmal cough, with dyspnea amounting almost to suffocation. Often the body is dislodged by this initial attack of coughing. If it becomes impacted in the larynx, it may cause sudden death by occluding the glottis; elsewhere it may excite acute laryngitis, usually of considerable severity.

The impaction of a foreign body in one of the primary bronchi, or one of the lobar divisions, is indicated by cough and a severe localized pain in the chest. There may be expectoration of blood. On auscultating the chest, there is found an absence of respiratory murmur over one lung or one lobe, according to the situation of the foreign body. Percussion usually gives marked dulness, the signs thus suggesting pleural effusion; or there may be increased resonance, which may even be tympanitic, owing to diminished tension in the part of the lung involved and to the emphysema which rapidly develops in the surrounding lung. If the foreign body remains impacted in one of the bronchi, it usually excites a localized inflammation, which may terminate in the formation of an abscess. This may result fatally, or there may follow a prolonged illness, with hectic symptoms resembling pulmonary tuberculosis; and finally, after weeks or months, the foreign body may be expelled by an attack of coughing, and the patient recover completely. In other cases no abscess develops but there are repeated attacks of acute pneumonia which never entirely resolve so that chronic pneumonia of an intense degree develops. The general health is greatly interfered with and the child usually succumbs to one of the recurrent acute attacks.

The diagnosis of a foreign body in the larynx is made by the suddenness of attack and the violence of the early symptoms. In older children the body may be seen with the laryngoscope, but in young children this is very difficult. The position of a metallic or solid body may be revealed by the X-ray. The prognosis is always doubtful, and depends upon the nature of the foreign body and the point at which it has been arrested. The usual cause of death either with or without operation is bronchopneumonia.

The first thing to be tried is inversion of the patient. By this means, assisted by the cough, the foreign body is not infrequently expelled even though it has passed below the larynx. The symptoms of laryngeal obstruction may call for immediate tracheotomy or laryngotomy, intubation not being applicable to these cases. If, after tracheotomy, the foreign body can be located in the larynx, but can not be extracted through the tracheal wound, the thyroid cartilage should be divided in the median line. The removal of a foreign body from the bronchi or the tracheal bifurcation should be attempted only by one skilled in bronchoscopy.

CHAPTER III

DISEASES OF THE LUNGS

THE PECULIARITIES OF THE LUNGS IN INFANCY AND EARLY CHILDHOOD

Thorax.—The general shape of the thorax is somewhat cylindrical, the conical or dome-shape of the adult thorax not being attained until puberty. The antero-posterior and the transverse diameters are nearly equal in the newly born, but after the third year the transverse diameter is always greater, the difference increasing steadily up to adult life. On account of the shape of the chest, the lungs are situated rather more posteriorly in the infant than in the adult.

The thoracic walls are very elastic and yielding, owing to the cartilaginous condition of a large part of the framework. They are relatively thinner than in the adult, chiefly from the imperfect development of the thoracic muscles. The greater part of the thickness of the thoracic walls is due to the deposit of fat, generally abundant in well-nourished infants; but where the fat is scanty the walls are extremely thin. The capacity of the thorax is considerably encroached upon by the high position of the diaphragm, the large size of the thymus gland, and the frequent distention of the stomach and intestines.

Respiration.—According to Uffelmann, the rapidity of respiration during sleep at the different ages is as follows:

At birth	35 per minute.
At the end of the first year	27 " "
At two years	25 " "
At six years	22 " "
At twelve years	20 " "

During waking hours this rate is very materially increased, and from comparatively slight disturbance it may be nearly twice as rapid.

The type of respiration in infants is diaphragmatic, and it continues to be chiefly so until after the seventh year, when the costal element gradually becomes more and more prominent. The rhythm of respiration is easily disturbed. In very young infants the regular rhythm is seen only in sleep. The lungs do not always expand equally; at certain times and in certain positions respiration may be carried on for a few moments almost entirely with one lung. For some moments it may be very superficial, and then quite deep. The length of the interval between

inspiration and expiration varies much at different times. Regular rhythmical respiration is not fully established before the end of the second year. After this time disturbances of rhythm are due chiefly to pulmonary or cerebral disease; but in infancy quite marked irregularity may have little or no significance. It is very common in all asthenic conditions.

Structure.—As compared with the adult, the trachea of the young child is larger; the bronchi are larger and occupy a greater space; the air cells are much smaller and occupy less space; and the interstitial tissue is much more abundant.

Physical Examination.—This requires tact and time, but yields results which are quite as satisfactory as in adults. It should be undertaken only in a room having a temperature of about 70° F., or before an open fire.

Inspection.—This should be made with the chest bare. There should be noted, the shape of the chest, the presence of deformities from rickets, the want of symmetry in the two sides, bulging of the intercostal spaces, whether the two lungs expand equally or not, also variations in rhythm, and the presence and extent of any recession of the soft parts or bony walls as an indication of obstructive dyspnea.

Palpation.—This also should be made upon the bare skin, always with the hand well warmed. Although we can not get the fremitus of the ordinary voice, we can get that of the cry. This is usually more intense than in adults, on account of the thinness of the chest walls. We frequently get a bronchial fremitus—a vibration produced by mucus in the tubes. The position of the apex beat of the heart should be determined, it being remembered that in infancy this is normally in the mammary line, or just inside of it, and usually in the fourth intercostal space.

Percussion.—For the examination of the back, the child may be laid face downward upon the nurse's lap, or be seated upon her arm. For the front and the lateral regions of the chest, the child is most conveniently placed upon his side across a hard pillow. The percussion blow must be light, either with a single finger or a small percussion hammer. Percussion should be made both during inspiration and expiration. The normal percussion note is somewhat tympanitic, this being due to the relatively large bronchi and the thin chest walls. This note is exaggerated in the interscapular region and beneath the clavicle, especially upon the right side. Here cracked-pot resonance may be obtained even in health.

Auscultation.—This may be practiced with the naked ear or with the stethoscope. A stethoscope is absolutely necessary for a thorough examination of the apices of the lungs in front and the axillary regions.

Most children are less frightened by the instrument than by the head of the physician during anterior auscultation.

The normal respiratory murmur of the infant is generally described as "puerile." In quality this has been likened to the bronchial breathing of the adult, but the resemblance is not a very close one. It is rude, rather loud, and seems very near the ear. Its peculiar character is due to the fact that the tracheal and bronchial sounds are more distinct, because not transmitted through so thick a layer of lung and chest wall. It is especially loud in the regions where the bronchi are superficial, as between the shoulder-blades and beneath the clavicles, particularly of the right side. A careful comparison of the two sides of the chest will generally enable an observer to avoid errors. The irregularity of rhythm which occurs from slight causes should be remembered, and the infant's position changed several times during auscultation, to avoid the mistake of attaching too much importance to a feeble respiratory murmur of one side.

On account of the thinness of the chest walls, there is difficulty in distinguishing between râles produced in the bronchi and pleuritic friction sounds. Before drawing any inference from the auscultatory signs, both lungs must be examined for several minutes, changing the child's position, and often inducing a cry or compelling a deep inspiration by other means, in order to bring out signs which otherwise may be overlooked. As auscultation is extremely difficult or impossible in a crying infant, this part of the physical examination should be made first if the child is quiet, since upon it we must chiefly depend for diagnosis. Inspection and percussion can be deferred until later.

Peculiarities in Disease.—There are several peculiarities connected with the respiratory organs in infancy and early childhood which must be constantly borne in mind in studying their diseases. The muscular development of the thoracic wall is feeble. The soft, yielding character of the thoracic framework causes the chest to sink in readily from atmospheric pressure whenever there is obstructive dyspnea. On account of the small size of the air vesicles, acute congestion may interfere with their function almost as completely as does consolidation. Because of the delicate walls of the air vesicles, emphysema is readily produced in obstructive dyspnea, but it is rarely permanent. There is a tendency to collapse, either on the part of lobules or groups of lobules, but very rarely of an entire lobe. This is a much less important factor in the production of symptoms in acute pulmonary disease than many writers would lead us to suppose. The tendency of inflammation to spread from the large to the small bronchi is much greater than in adults. In all forms of pulmonary disease the rapidity of respiration is much greater than in adults. Areas of consolidation often exist without appreciable

changes in the percussion note, because they are superficial and are surrounded by healthy or emphysematous lung. Flatness should always suggest the presence of fluid.

Probably the most common mistakes are to confound bronchial râles with friction sounds, exaggerated puerile breathing with bronchial breathing, and to overlook the existence of fluid because of the presence of bronchial breathing.

ACUTE CATARRHAL BRONCHITIS

Acute catarrhal bronchitis is one of the most frequent conditions for which the physician is called upon to prescribe in children. It occurs at all ages, from early infancy up to puberty. Its frequency, however, diminishes steadily after the second year. The predisposition to acute bronchitis exists with the same constitutional conditions, and is acquired in the same manner as the predisposition to the acute catarrhal inflammations of the upper respiratory tract. (See Acute Rhinopharyngitis.) Bronchitis is very common in children who are suffering from rickets and malnutrition. It is much more frequent in the cold months, especially in the late winter and early spring, when there are sudden atmospheric changes and high winds. The presence of large tonsils and adenoid vegetations of the pharynx are important predisposing causes.

Bronchitis may be a primary or a secondary disease. The primary form is excited by cold, exposure with insufficient clothing in severe weather, wetting of the feet, or chilling of the surface in any manner. Under these conditions it may occur alone, or be associated with or preceded by acute catarrh of the nose, pharynx, or larynx. In rare cases it is caused by the inhalation of irritants. Bronchitis is an almost invariable accompaniment of measles and influenza. It is very common in pertussis, in scarlet and typhoid fevers, and diphtheria, and may occur in any acute infectious disease; it also complicates pneumonia and pleurisy. The microörganisms associated with bronchitis are chiefly the staphylococcus aureus and the pneumococcus, often in combination; next in importance are the streptococcus and, especially in protracted cases, the influenza bacillus.

Lesions.—Acute catarrhal bronchitis is an inflammation of the mucous membrane of the bronchi. As a rule it is bilateral, both sides being involved to the same degree. Localized bronchitis is secondary to some other pathological process in the lungs, usually tuberculosis, old pleuritic adhesions, or pneumonia. In acute bronchitis only the larger tubes may be affected, this usually being complicated with inflammation of the trachea (ordinary tracheobronchitis); or, in addition, the process

may extend to the medium-sized tubes (severe bronchitis); or, in infants especially, it may extend to the smallest tubes (capillary bronchitis). In the last-mentioned form there are invariably changes in the zones of air vesicles surrounding the bronchi, and these cases are therefore more properly classed as bronchopneumonia. In the first form the inflammation is superficial, and affects only the mucous membrane of the bronchi. In the second form it may involve the entire thickness of the bronchial wall, and in the third form it does so regularly.

The pathological changes consist in congestion and swelling of the mucous membrane, desquamation of the epithelium, and an exudation of mucus and pus cells. At autopsy the injection of the mucous membrane is usually distinct; pus and mucus cover the surface of the larger bronchi, and by pressure ooze from the cut extremities of the smaller tubes. The chief lesion of the walls of the bronchi consists in an infiltration with leucocytes. In infants dying from bronchitis, the lungs are much more frequently emphysematous than collapsed. In fact the readiness with which emphysema occurs in bronchitis is one of its distinguishing features in infancy. However, this is rarely permanent but usually subsides rapidly after the acute attack is over. There is swelling of the lymph nodes at the root of the lungs, which in most of the acute cases is slight, but in protracted cases, and after recurring attacks, may be quite marked.

Symptoms.—It is convenient to consider separately the symptoms in infants and in older children.

The Bronchitis of Infants.—1. *The Mild Form* (*Bronchitis of the Larger Tubes.*)—The onset is generally gradual, and the symptoms of bronchitis may be preceded by those of catarrh of the nose, pharynx, or larynx. The change in the character of the cough, the slightly accelerated breathing, and a further rise in temperature, indicate an extension to the bronchi. The cough may be constant and severe, or very slight. There is no expectoration. The secretions are usually coughed up into the mouth or pharynx, and swallowed. This sometimes excites vomiting. At other times the mucus is coughed only into the trachea or larynx, and aspirated again into the lungs. The respirations are from forty to fifty a minute, and often accompanied by a rattling sound, due to mucus in the large bronchi or trachea. The general symptoms are not severe, and unless the infant is very young or very delicate no apprehension need be felt as to the outcome. The temperature is generally from 100° to 102° F. for two or three days, then below 100° F. A moderate amount of restlessness dependent upon the severity of the cough, anorexia, and sometimes vomiting and diarrhea, are usually present.

The physical signs in the first stage are dry, sonorous râles over the whole chest. A little later these give place to coarse mucous râles heard

everywhere, but especially distinct between the scapulae and in the infraclavicular regions. On palpation there is usually a marked bronchial fremitus. Often there is not enough dyspnea to cause recession of the soft parts of the chest. Unless the disease extends to the smaller bronchi and the air vesicles, the illness usually lasts about a week. Coarse râles in the chest may remain for some time after the symptoms have subsided. Relapses are exceedingly common. In a delicate or rachitic child, or in one whose surroundings are bad, one attack is likely to be followed by a succession of others, so that the child may not be really well until warm weather comes. The general health may suffer from the prolonged confinement to the house, although the patient may never have been seriously ill.

2. *The Severe Form* (*Bronchitis of the Smaller Tubes*).—This differs from the preceding variety mainly in the greater severity of all its symptoms. The onset may be like that just described, the severe symptoms not appearing until the patient has been sick two or three days, or they may be severe from the outset. If the latter, it is indistinguishable from bronchopneumonia. There is cough, dyspnea, accelerated breathing, fever, and moderate, sometimes severe, prostration. The cough is tighter, and more frequently of a short, teasing character than severe and paroxysmal. There is difficulty in nursing. Dyspnea may be quite marked and is shown by the active dilatation of the alae nasi and the recession of all the soft parts of the chest on inspiration. The respirations, as a rule, are from 50 to 80 a minute. The temperature for the first day or two is usually 100° or 102° F., but it may be 103° or 104° F. So high a temperature does not continue unless pneumonia develops. The prostration is in most cases more closely related to the dyspnea and the rapidity of respiration than to the temperature. Often there is slight cyanosis.

In the beginning the chest is filled with sibilant and sonorous râles. In twelve or twenty-four hours these are wholly or in part replaced by moist râles—coarse or fine, according as they are produced in the large or medium-sized tubes. The râles are always best heard behind, but they are present all over the chest. The sibilant and sonorous breathing may persist throughout the attack and for a week or two thereafter. This prominence of the spasmodic or asthmatic element in bronchitis is characteristic of infancy and early childhood. The respiratory murmur is feeble; the resonance on percussion is normal or slightly exaggerated. As the case progresses toward recovery, the finer râles are the first to disappear.

At the onset of such a case it is impossible to say whether the disease will be limited to the medium-sized bronchi or will extend to the smallest bronchi and air vesicles. In young or very delicate infants, and dur-

ing measles, it is very common for the disease to spread rapidly to the air vesicles. In other cases, usually in infants under six months old, there may develop attacks of respiratory failure or suffocation. These may occur in a severe case at any time, and, because of the infant's inability to empty the tubes of secretion, the dyspnea steadily increases until the respiratory muscles are exhausted. The symptoms which follow are usually ascribed to pulmonary collapse. It is, however, by no means certain that this is the correct explanation, for at autopsies made in such cases the lungs are usually found to be the seat of acute emphysema. The clinical picture is a clear one. There is no disposition to cough or cry; the pulse is feeble; the respiration very rapid, superficial, often irregular; the skin cyanotic, and often clammy. Finally, there may be added to the other signs, dulness, apathy, and stupor. Such attacks may come on quite suddenly even in robust infants, and unless the treatment is energetic, death often follows in a few hours, being frequently preceded by convulsions.

The usual course of the disease in infants previously in good health is that the severe symptoms continue for two or three days only, after which the temperature falls to 100° or 100.5° F., and gradually becomes normal. The constitutional symptoms usually decline with the temperature, and, except during the first thirty-six hours, they rarely give cause for anxiety. Recovery almost invariably occurs unless the disease extends to the finer bronchi.

Bronchitis is principally to be distinguished from bronchopneumonia. The differential diagnosis is more fully considered under that disease. The most important points are that in pneumonia the temperature is higher and more prolonged, the prostration greater, the râles very often localized—being heard only behind, often over only one lung—the duration is more protracted, and all the symptoms are more severe. In nearly all cases of severe bronchitis in young children some pneumonia is present.

The Bronchitis of Older Children.—This is not nearly so serious as in infants, because the same danger does not exist of extension of the inflammation to the finer bronchi and air cells.

1. *The Mild Form.*—This is very common. The constitutional symptoms are slight, and often entirely absent after the first day. The patient is never sick enough to go to bed. The first symptoms are cough and soreness or a sense of oppression beneath the sternum. The cough is always worse at night. It is at first tight, hard, and racking; later it is loose, and in children over five years old there is usually expectoration—first of white, frothy mucus, but after a few days it becomes more abundant, and of a yellow or yellowish-green color, from the presence of pus. The physical signs are only coarse râles, at first dry, and later moist, but

heard over both sides of the chest, in front and behind. There may be some disturbance of digestion, anorexia, constipation, or diarrhea. The usual duration of the attack is from one to two weeks. If the patient is not kept indoors the disease may pass into a subacute form, lasting for several weeks as a protracted "winter cough," but without any other important symptoms.

Such prolonged or recurring attacks of bronchitis of a subacute form should suggest influenza or tuberculosis. A positive cutaneous tuberculin reaction renders tuberculosis probable. A careful search for bacilli in the sputum should then be made. Although not found at first, if present repeated examinations will usually disclose them. Influenza can be diagnosticated with certainty only by sputum cultures.

2. *The Severe Form.*—The onset is abrupt, with fever, chills, pains in the back, headache, cough, and sometimes pain in the chest. There is a feeling of tightness or constriction beneath the sternum. The onset resembles that of pneumonia, except that the symptoms are less severe. The temperature for the first two or three days ranges betwen 100° and 103° F. It is generally highest in the first twenty-four hours. The cough resembles that of the mild form, but it is usually more severe. The expectoration is more profuse, and occasionally, in the early stage, it may be streaked with blood.

The coarse râles of the mild form are present, and in addition there are finer râles—at first dry, and later moist—heard all over the chest. Frequently, wheezing râles are heard on expiration. The duration of the attack is ordinarily from two to three weeks, the patient being sick enough to be confined to bed for three or four days only. There is frequently a cough for some time after all physical signs have disappeared. Relapses are easily excited by any indiscretion before the patient has quite recovered.

The prognosis in the primary cases is good, such almost invariably terminating in recovery, and very exceptionally passing into bronchopneumonia; but this not infrequently happens when the attack complicates measles or pertussis.

Treatment of Bronchitis.—To remove the predisposition to bronchitis the same means should be employed as those mentioned in Acute Rhino-Pharyngitis. Children with tuberculous antecedents, and those who are especially prone to pulmonary disease, should, if possible, spend the winter in a warm climate. The sleeping apartments of susceptible infants should not be too cold—never below 55° F.—but they should be well ventilated. It is important in infants and young children that mild attacks of bronchitis should not be neglected.

Every young child who has an acute catarrh of the nose, pharynx, larynx, or bronchi should be kept indoors. In every such catarrh accom-

panied by fever the child should be kept in bed while the fever lasts, even if the temperature does not go above 100.5° F., and is accompanied by no other constitutional symptoms. A very large number of the cases will recover promptly when no other treatment is employed than to keep the child in bed. Fresh air is indispensable. But the advantages of cold air have not yet been demonstrated. According to our experience, the wide-open windows have no place in the treatment of acute bronchitis in infants or young children in the winter and spring season. The temperature of the room should be about 70° F. The room should be well ventilated and frequently aired, the child meanwhile being removed to another room. There is a great advantage in changing the child's position in the crib and from the crib to the nurse's arms. Careful attention should be given to feeding and to the condition of the bowels. A cathartic, preferably castor oil, should be administered at the outset.

Poultices are objectionable and should not be employed. Counter-irritation is very valuable. In infants, good results are obtained by the frequent use of a mustard paste (see chapter on General Therapeutics). The paste may be repeated, according to indications, from two to five times a day. If properly used, it will not injure the skin.

Inhalations may, in the great majority of cases, take the place of the administration of drugs by the mouth, a very great advantage in infants. They may be used by means of the croup kettle, the child always being placed in a tent. In the early part of the disease inhalations, like simple aqueous vapor or lime-water, may be used. Later turpentine, creosote, benzoin, terebene, or eucalyptol may be added. Of these, creosote usually gives the most satisfaction. Inhalations are to be used for ten or fifteen minutes from four to eight times a day.

In infancy, expectorants may advantageously be dispensed with. For older children, antimony and ipecac may be used in the first stage. When the secretion is more abundant, creosote, turpentine, or terebene may be given. Small, frequently repeated doses usually give the best results. Opium should be given cautiously to infants. The dry, harassing cough of the early stage sometimes yields to nothing so quickly as to small doses of Dover's powder (e. g., one-tenth of a grain every two hours to a child of one year). The use of emetics to get rid of bronchial secretion is not to be advised. Stimulants are not required in most of the cases. The indications for them are the same as in pneumonia. When there is much dyspnea of the asthmatic type, nothing works as well as epinephrin. It should be given intramuscularly; the dose is two to five minims of the 1-1,000 solution. The effects are almost immediate, but often only transient.

Should attacks of suffocation and respiratory failure occur in infants, the indications are to excite respiratory movements and to get as much

blood as possible to the surface and the extremities. Flagellation or spanking and the use, alternately, of hot and cold douches to the chest will sometimes induce the deep respiratory efforts desired. Other useful measures are the hot mustard bath and the mustard pack applied to the entire body. Probably the most effective of all remedies is dry cupping. The chest should be cupped front and back for five or ten minutes every few hours. Oxygen should be administered. As these symptoms are likely to recur every few hours for a day or two, a repetition of the treatment may be needed. For such patients cold air is injurious. They should be kept in a room with a temperature of 70° to 72° F.

In the non-febrile cases in older children, confinement in bed is unnecessary, but they should be kept indoors. In the early stage, with hard, dry cough, one of the best remedies is brown mixture (the mistura glycyrrhizae composita of the U. S. P.). It will be found advantageous in most cases to have the formula made up with one-half the usual amount of opium. When the cough is especially hard and dry inhalations of steam are indicated. In the second stage, muriate of ammonia may be added to the brown mixture; or terebene, two or three drops upon sugar, may be given four or five times a day, and inhalations should be used several times a day.

In the more severe cases the patients should be kept in bed and a counter-irritant to the chest employed. For the general discomfort, pain, headache, etc., nothing is better than phenacetin and Dover's powder (two grains of the former to one-half grain of the latter to a child of five years), repeated every three to six hours. All patients should be kept in bed as long as the temperature is above normal.

After all physical signs and constitutional symptoms have disappeared, a cough continues sometimes for weeks. Expectoration is scanty, or is wanting altogether; the cough is hard, dry, often paroxysmal, and in some cases occurs at night only. For this condition the best remedies are cod-liver oil and creosote. When these measures are not effective, a change of climate should be advised.

FIBRINOUS BRONCHITIS (*Bronchial Croup*)

Fibrinous bronchitis is usually seen in diphtheria, as an extension from the larynx or trachea. There is, however, another form of bronchitis attended by a fibrinous exudate, which occurs as a primary disease. This is very rare in children. Weil has, however, collected twenty cases of the primary form. The etiology is obscure. It is seen at all ages, from infancy up to puberty, and it may be either acute or chronic. From the cases thus far reported it would appear that the acute form is rela-

tively more common in children than in adults. The disease may be confined to certain branches of the bronchial tree, or it may affect all the bronchi, even to the minute subdivisions. The fibrinous membrane is found loose in the tubes or adherent. There are generally associated other pulmonary changes, such as emphysema, atelectasis or bronchopneumonia.

The acute form somewhat resembles ordinary catarrhal bronchitis. The diagnostic features are, the severity of the dyspnea and the expectoration of tube casts from the larger bronchi, or elongated cylinders from the smaller ones, the former resembling macaroni, the latter, vermicelli. The expectorated masses are often in balls or plugs, and their peculiar character is not recognized until they are placed in water. The casts are dissolved by alkalis, especially by lime-water. After the expulsion of a large cast, improvement in all the symptoms occurs. They, however, return as the exudate reappears. The ordinary duration of acute cases is from one to three weeks.

In the chronic form there are no constitutional symptoms, but only dyspnea and cough, often recurring in paroxysms, with the expectoration of fibrinous casts. The patient may have these attacks at intervals of a few days or weeks, extending over a period of months, or even years. There are no characteristic physical signs. The diagnosis rests upon the peculiar character of the expectoration. The prognosis in acute cases is unfavorable, the mortality being 75 per cent (Weil). Chronic cases are not dangerous to life.

Treatment.—This is quite unsatisfactory. To loosen the membrane and facilitate its expulsion, the most efficient means are inhalations of the vapor of lime-water. Pilocarpin is too dangerous for use with small children. Occasionally emetics are of value. Improvement in some of the chronic cases has resulted from the use of iodid of potassium.

CHRONIC BRONCHITIS

Chronic bronchitis is not a very common disease in children, particularly in young children, one reason being that chronic emphysema, so frequently an associated condition in adults, is rather rare in early life. Chronic bronchitis always accompanies chronic pulmonary tuberculosis and chronic interstitial pneumonia, with or without the occurrence of bronchiectasis. It is seen in chronic cardiac disease, especially with lesions of the mitral valve. It may occur as a late symptom of hereditary syphilis. Excluding the varieties mentioned, it usually follows attacks of acute bronchitis, the process becoming chronic because of the patient's constitutional condition or his unhygienic surroundings. The acute at-

tack may be primary, but it often follows measles and whooping-cough. Deformities of the chest, the result either of rickets or of Pott's disease, are occasionally a cause.

Symptoms.—The only constant symptom is cough, which is persistent, obstinate, and nearly always worse at night or early in the morning. It often occurs in paroxysms strongly suggestive of pertussis. Expectoration is not generally abundant, but in older children it is usually present, and in a few cases it is profuse. A copious morning expectoration of fetid pus or muco-pus indicates bronchiectasis. There is no fever, little or no dyspnea, and although the patients are thin, they are not emaciated, and in many cases the general health is not much affected. There may be coarse mucous râles, or no physical signs whatever. The duration of the disease is indefinite, depending upon the cause. All these patients are better in summer than in winter, and suffer frequently from exacerbations of acute or subacute bronchitis.

The diagnosis is to be made mainly from pertussis and tuberculosis. From mild attacks of pertussis the diagnosis may be impossible except by the course of the disease. Tuberculosis may be suspected if the thermometer shows regularly a slight evening rise of temperature, if there is much anemia, and steady loss of flesh. It may, however, be present without any of these symptoms. A positive cutaneous reaction is suggestive, but a certain diagnosis can be made only by the discovery of tubercle bacilli in the sputum.

Treatment.—The first indication is to treat the primary conditions upon which chronic bronchitis may depend. Attention should be directed to the general condition—rickets and malnutrition each receiving its appropriate treatment. In many cases a change of climate is the only thing which is really curative. The general health should be promoted as much as possible by a tonic plan of treatment which may advantageously include the use of cod-liver oil. The results obtained from drugs are not very satisfactory but the following may be employed: potassium iodid, creosote and terebene, the last two being given both by mouth and by inhalation. For the relief of cough opiates are to be avoided as much as possible.

ASTHMA

Asthma is characterized by attacks of severe spasmodic dyspnea, which may be preceded, accompanied, or followed by a bronchitis of greater or less severity. In infancy, the association of asthma with bronchitis is a very close one, and the cases present quite a different clinical picture from the disease as seen in older children, which differs in no essential points from the asthma of adults.

Writers differ very much in their statements regarding the frequency of asthma in early life, mainly because of a want of agreement in regard to what shall be included under this term. The asthmatic attacks of infants are considered by some as a stage of bronchitis, by others as distinct from that disease. Typical attacks resembling those of adult life are rare in children, and extremely so before the fifth year. However, of 225 cases of asthma reported by Hyde Salter, the disease began before the tenth year in nearly one-third the number.

Etiology.—The general or constitutional causes are the same in children as in adults. Asthma is often hereditary. It frequently occurs in children who in infancy have suffered from eczema. The local cause may be any form of irritation in the nose or pharynx—hypertrophic rhinitis, adenoid growths of the pharynx, hypertrophied tonsils, or elongated uvula—or in the bronchial mucous membrane, as a result of previous attacks of acute bronchitis. It is probable that it may also be caused by the irritation of enlarged bronchial glands. In susceptible children, a paroxysm may be excited by high winds, dust, cold and damp air, indigestion, constipation or the inhalation of substances such as the pollen of certain plants, especially rag-weed, golden-rod and roses. Contact with animals, especially horses, cats and dogs may also initiate an attack. It has been recently shown, by Schloss and Talbot particularly, that certain foods, especially eggs and rarely milk, pork and other meats are responsible for attacks in certain children. There can be no doubt that this susceptibility to the pollen of plants, to contact with animals and to various foods is a phenomenon closely allied to that of anaphylaxis. Cutaneous and intracutaneous tests have shown not only the production of urticarial wheals, at the site of the test, but have also initiated attacks of asthma. In certain instances the susceptibility to these protein substances is inherited; in others it is perhaps the result of an active sensitization, but in many instances there is no sufficient explanation as to how the child has become sensitized. The constriction of the bronchi, which causes many of the symptoms of asthma, is probably chiefly due to the contraction of the unstriped circular muscular fibers in the walls of the bronchi. Swelling of the mucous membrane, either by dilatation of the blood vessels or by exudation of the serum into the mucous membrane itself undoubtedly is a factor of importance in some instances.

Symptoms.—Four quite distinct clinical types of asthma are seen in children: (1) Cases which in their onset simulate attacks of bronchitis; (2) those in which asthmatic symptoms follow an attack of bronchitis, continuing for weeks or months, but not necessarily recurring; (3) hay fever, or the periodical form which occurs every summer; (4) that which resembles the ordinary adult asthma, with the nervous element predominating. The prominence of the catarrhal symptoms is char-

acteristic of all forms of asthma in children, the first two varieties mentioned being peculiar to early life.

Attacks Resembling Acute Bronchitis.—These cases are rare, but may be seen even in infants. The onset is sudden, with moderate fever, incessant cough, severe dyspnea, and sometimes cyanosis, prostration, and cold extremities. The chest is filled with sonorous, sibilant, and soon with subcrepitant râles. Instead of running the usual course of bronchitis of the finer tubes, the symptoms may pass away very rapidly, and in forty-eight, sometimes in twenty-four, hours the patient may be quite well. It is only by the course of the disease and by recurring attacks that their true nature can be recognized. In infants this form of asthma may be fatal.

Cases Following Attacks of Bronchitis—Catarrhal Asthma.—This form is not uncommon, though it is frequently designated by some other term than asthma—sometimes as spasmodic bronchitis, or catarrhal spasm of the bronchi. The symptoms are, however, indistinguishable from asthma, and they evidently belong in the same category. This form is usually seen in infants, being rare after the third year. Many of the patients are rachitic; others have large tonsils, or adenoid growths of the pharynx; while in still others there is every reason to suspect the presence of large bronchial glands. Usually there is nothing peculiar about the antecedent bronchitis; in most cases it is not especially severe, and is limited to the larger tubes. The febrile symptoms subside in a few days, but the cough continues, as do also the dyspnea and wheezing. When the symptoms are fairly established they are very uniform and characteristic. The respiration is accelerated, usually to 50 or 60, sometimes to 70 or 80, a minute. The temperature from time to time may be very slightly elevated, or it may remain normal. The respiration is noisy, labored, and accompanied by distinct wheezing.

On auscultation, there is prolonged expiration accompanied by loud, wheezing and sonorous, or sibilant râles, and occasionally coarse moist râles are heard. In cases which have lasted some time a moderate amount of emphysema can be inferred from the prominence of the infraclavicular regions, and exaggerated resonance over the chest in front and the depression of the bases posteriorly.

These symptoms and signs often continue for three or four weeks. While they are constantly present, they vary in intensity from time to time, being usually much worse at night. The symptoms are always increased by exposure to a cold, damp atmosphere, by any fresh accession of bronchitis, and often by trivial digestive disturbances. The cough is not usually severe, and expectoration in most cases is absent. The general health is often but little affected. With recovery from the asth-

matic symptoms the emphysema usually disappears gradually, although we have seen severe cases in which it persisted.

What proportion of these children afterward develop ordinary asthma, we are unable from personal experience to say. Some undoubtedly do, but in others which we have been able to follow, recovery has seemed to be permanent. This would appear more likely in those cases closely associated with rickets, or with other causes which disappear spontaneously with time or as a result of treatment.

Hay Fever.—This is very rare before the seventh year and but few well-marked cases are seen before the tenth year. In its clinical aspects it does not differ essentially from the disease as seen in adults, except possibly by the greater prominence of the bronchial catarrh.

Ordinary Attacks of the Adult Type.—These usually occur at intervals of a few weeks or months, depending upon the nature of the exciting cause. The beginning is usually at night with dyspnea, a short, dry cough, and loud, wheezing respiration. Deep recession of the soft parts of the chest is seen, as in laryngeal stenosis. There is prolonged expiration, accompanied by loud, sonorous, sibilant and wheezing râles, and the vesicular murmur is very feeble. Later, moist râles may be heard. After many attacks emphysema is present. This occurs more rapidly than in adults, and may be extreme, giving rise in marked cases to serious thoracic deformity. On account of the loss of sleep and interference with nutrition, the general health may become seriously impaired. Urticarial wheals are not infrequently present at some time during an attack.

Diagnosis.—Typical attacks of asthma are easily recognized. Some of the catarrhal forms seen in infancy, however, present some difficulty, and a positive diagnosis may be impossible except by the progress of the case. The presence of urticaria speaks strongly for asthma. The blood picture in asthma is characteristic and of much value in diagnosis. The important thing is the presence of a large number of eosinophile cells. They may form as high as 15 to 20 per cent of the leucocytes. In a series of cases examined in one of our clinics by Wile, the average was 10.7 per cent; the highest observed being 26 per cent. The eosinophilia is greatest at the height of the attack. The blood examination serves to differentiate asthma from simple bronchitis and from tuberculosis. The existence of marked eosinophilia definitely establishes the asthmatic character of some of these attacks in infancy. Eosinophile cells are commonly found in the sputum. Charcot-Leyden crystals and Curschmann's spirals may also be seen but much less frequently and usually only in the sputum of older children.

Prognosis.—This is best in the cases of catarrhal asthma in infants, and in older patients when it depends upon some local cause which can

be removed, as when the disease is due to reflex nasal or pharyngeal irritation. In the majority of other cases, asthma is likely to become chronic unless the child is removed to some climate in which the attacks do not occur. The younger the child, the shorter the duration of the disease, and the less marked the hereditary tendency, the better the prognosis. In those children that are sensitive to the pollen of plants and to certain foods there is reason to believe that specific treatment by immunization may be of benefit. The results with hay fever have been encouraging and Talbot was able to prevent, or diminish greatly, the attacks in some of his patients especially those who were sensitive to egg.

Treatment.—The nose and the rhinopharynx should be carefully examined in every case of asthma, and any pathological condition there present should receive attention as the first step in the treatment. Importance, in children, should be attached to the removal of adenoid growths of the pharynx. We must admit, however, to have seen very few cases of asthma cured or even greatly improved by this means. During attacks, the best means of relieving the symptoms is the inhalation of fumes of nitre paper or stramonium leaves. Most of the proprietary remedies contain these ingredients. The sleeping room may be filled with the fumes of these substances, or the child may be placed in a tent into which the fumes are introduced. Emetics may be employed when the attack is brought on by indigestion. To prevent the recurrence of night attacks, antipyrin is valuable given in full doses at bedtime—four grains at five years and six grains at ten years. Between the attacks the syrup of hydriodic acid (for a child of five years one-half teaspoonful, t.i.d.) or potassium iodid (gr. ii to gr. iv, t.i.d.), may be given for a number of weeks. Tonics are often useful. Those especially valuable in asthmatic patients are cinchonidia (gr. ii, t.i.d.) and arsenic (gr. 1-100, t.i.d.). They may be advantageously combined. Cocain used locally in the throat and opium by mouth or hypodermically will often cause a cessation of attacks but are objectionable with older children on account of the tendency to the formation of a drug habit. On account of their susceptibility to the drug, cocain is dangerous with infants and very young children.

In the severe acute attacks nothing gives so much immediate relief as the use of epinephrin intramuscularly—dose ♏v to ♏viii, for a child of three years.

In the cases of catarrhal asthma following bronchitis, expectorants and ordinary cough remedies are useless. Cod-liver oil and the iodid of potassium are valuable in some of the cases. Others are greatly relieved by the regular use of creosote inhalations several times a day, with a nightly dose of antipyrin. The fumes of nitre and stramonium often

afford no relief, and sometimes the cases are made distinctly worse by them. The best of all measures is to send the child at once to a warm, dry climate.

Very careful attention should be given to the diet; articles to be avoided with most asthmatic children are cream, eggs, and all sweets. For all children who have had repeated attacks, whether in the form of hay fever or for those whose asthma is chiefly in the winter and spring and excited by attacks of bronchitis, the most important thing is removal to a place where they do not have the disease, and a residence there long enough to break up the tendency to recurrence. This will usually require several years. The region best suited to most asthmatics is one which is high, dry, and moderately warm. Some do exceedingly well at the seashore; others much better in the mountains. Patients often suffer less in cities than in the country.

Children who are susceptible to the odor of animals should be kept from contact with them. Those who are sensitive to proteins of certain foods should have these eliminated from the diet after it has been determined which are the proteins responsible. Immunization by the ingestion of very small quantities of the proteins has been practiced by Schloss and Talbot with marked success in some instances. The method is still in the experimental stage and should be employed with caution.

Those children who are sensitive to pollen should spend the weeks in which the plants are in bloom in the mountains or at some place where they are not exposed. Distinct benefit has been obtained by immunization against pollen in the hay fever and asthma of adults. It is as yet too early to say what the effect of this form of treatment will be with children. It should be attempted only by one trained in the methods of immunity.

CHAPTER IV

DISEASES OF THE LUNGS—(Continued)

PNEUMONIA

In early life the lungs are more frequently the seat of organic disease than any other organs in the body. Pneumonia is very common as a primary disease, and ranks first as a complication of the various forms of acute infectious disease of children. It is one of the largest factors in the mortality of infancy and childhood.

Cases of acute pneumonia are divided, from an anatomical point of view, into two principal groups: (1) bronchopneumonia, also known as catarrhal and as lobular pneumonia; (2) lobar pneumonia, also known as croupous and as fibrinous pneumonia. These differ little from each other in etiology, but considerably in the products of inflammation, the distribution of the disease in the lung, and somewhat as to the parts involved and the nature of the changes in them.

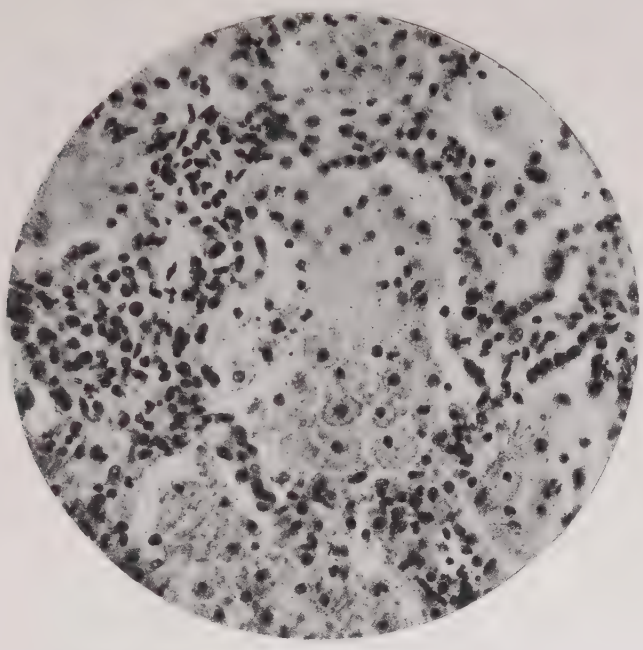


FIG. 46.—BRONCHOPNEUMONIA. The picture shows at its center one entire air vesicle, and at its margin parts of four or five other vesicles; they are filled with large epithelial cells having small nuclei. There are also seen leucocytes with intensely black nuclei and narrow protoplasm. Between the cells is a finely granular material, which is the exudation fluid coagulated during the hardening process. The alveolar septa are somewhat infiltrated.—From Karg and Schmorl.

In bronchopneumonia the large bronchi are the seat of a superficial inflammation, while in those of small size the entire bronchial wall is affected; the exudation into the air vesicles is mainly cellular, being made up of epithelial cells, leucocytes, and red blood-cells (Fig. 46), fibrin being either absent, or present only in small amount. In many cases there are marked changes both in the alveolar septa and in the interstitial tissue of the lung; resolution is often imperfect, and there is a strong tendency for the inflammation to pass into a chronic form, involving the connective-tissue framework of the lung. The lesion is widely and often irregularly distributed, usually being most marked in

the vicinity of the small bronchi from which the inflammation spreads, and in the most superficial lobules of the lung.

In lobar pneumonia, bronchitis, when present, is usually superficial, the walls of the bronchi being very slightly or not at all affected; the same is true of the alveolar septa. The principal product of the inflammation is fibrin (Fig. 47), which fills the alveoli and the terminal bronchi, the cells being relatively few and chiefly leucocytes. The process is

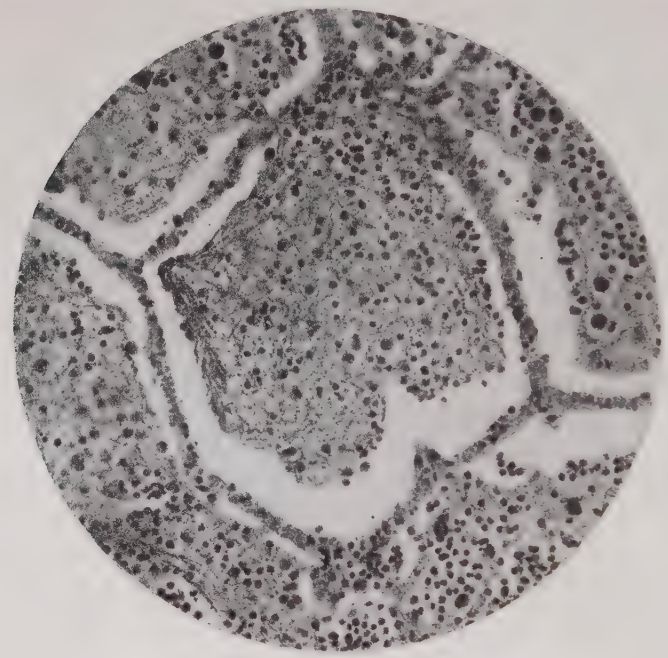


FIG. 47.—LOBAR PNEUMONIA. In the air vesicle shown in the picture there is a firm, close network of fibrin, in the meshes of which are leucocytes. At the lower part the exudation has contracted away from the wall in consequence of the process of hardening.—From Karg and Schmorl.

usually sharply circumscribed, involving an entire lobe or a part of a lobe. In most cases it clears up rapidly and completely, there being but little tendency to involve the framework of the lung in a chronic process.

While in typical cases the two forms of inflammation are quite distinct, there are seen many intermediate forms which partake of the characters of both, and one may be in doubt, even after a microscopical examination, in which group to place a case. It not infrequently happens that both varieties of pneumonia are present in different parts of the same lung or in both lungs at the same time. These mixed forms are especially frequent during the second and third years; but during the first year, and after the third, the types are usually well marked.

The following table shows the relative frequency of lobar and bronchopneumonia in three hundred and seventy cases,[1] nearly all taken from one institution (New York Infant Asylum). They include all the cases of acute primary pneumonia occurring during seven years:

Under six months,	bronchopneumonia,	73	cases;	lobar	pneumonia,	11	cases.
Six to twelve "	"	96	"	"	"	29	"
Second year,	"	73	"	"	"	40	"
Third "	"	19	"	"	"	23	"
Fourth "	"	0	"	"	"	6	"
Totals,	"	261	"	"	"	109	"

Thus it will be seen that, of the cases of acute pneumonia occurring during the first two years, twenty-five per cent were lobar and seventy-five per cent were bronchopneumonia.

When we come to a consideration of the microörganisms with which the different forms of pneumonia are associated, we find that they do not correspond to the anatomical varieties. Lobar pneumonia is regularly associated with the presence of the pneumococcus, rarely with Friedländer's bacillus, but in a large number of cases other organisms are also found. In bronchopneumonia there is almost always a mixed infection. In the primary cases the pneumococcus is usually the predominant organism, but it is commonly associated with the staphylococcus aureus. In the secondary cases, especially when pneumonia follows measles or scarlet fever, the streptococcus is usually present, such cases being generally of a severe type. In the pneumonia of diphtheria, besides the streptococcus the diphtheria bacillus is frequently found. In winter the bacillus of influenza may be the only organism present, but it is usually associated with the pneumococcus. The organisms mentioned are found in all possible combinations, sometimes one and sometimes another predominating. With any of them the bacillus of tuberculosis may be found.

Much interest has recently been aroused in the different types of pneumococci which are found in acute pneumonia.[2] Of the cases studied thus far in young children, type IV of Cole's classification, has been much the most frequently present; but all the forms found in adults have been observed. In a series of 50 cases studied at the Babies' Hospital nearly 75 per cent were type IV.

[1] The division was here made according to the predominant clinical or pathological features. Most of the doubtful cases were classed as bronchopneumonia.

[2] According to the researches of Cole of the Rockefeller Institute pneumococci may be divided into four groups or types. Nos. I, II and III have definite individual characteristics; IV includes the remainder or unclassified group. The differentiation is made by animal inoculation and requires from twelve to twenty-four hours. For type I he has produced a serum from immunized horses

Some idea of the nature of the infection in pneumonia may be gained from the following table. The sputum cultures represent the pneumonias of one winter and spring in the Babies' Hospital, and the post-mortem cultures from those of two seasons in the same institution:

	Sputum cultures from 124 cases of pneumonia.	Post-mortem cultures from the lungs in 76 cases of pneumonia.
Staphylococcus aureus	in 116 cases	36 (alone in 8)
Pneumococcus	" 94 "	26 (" " 4)
Streptococcus	" 63 "	17 (" " 1)
Bacillus influenzae	" 47 "	19 (" " 2)

Why the same exciting cause in one case produces bronchopneumonia and in another lobar pneumonia may be in part owing to the difference in the structure of the lung at the different ages, especially the relatively large size of the bronchi in infancy. Again, in very young and in feeble children, the process tends to become diffuse and the products are chiefly cellular; in those who are older and more vigorous it is likely to be circumscribed, with fibrin as its chief product; in the intermediate ages and intermediate conditions the types are often mingled.

The immediate source of infection of the lungs is the mouth or the rhinopharynx. All the forms of bacteria found in pneumonia may be found in these cavities, some of them constantly, others only at certain times, especially during an attack of any of the acute infectious diseases. Provided the other conditions are favorable, pneumonia may be excited by direct contagion. This plays a small part in inducing primary pneumonia; there seems, however, to be little doubt that the secondary forms, especially the pneumonia complicating measles, diphtheria and influenza, are not infrequently communicated in this way.

---

which has been shown to have distinctly curative effects. Thus far no satisfactory serum for the other groups has been produced. As the serum is not effective in infections due to other types than I, it is of little assistance in the pneumonias of young children since few of the cases of pneumonia at this age are due to this type of organism. The serum is not yet available for general use.

The pneumococci of types I and II are seldom found except in the mouths of persons suffering from pneumonia or those in contact with them. Type IV is the form which is most widely diffused and is frequently found in the mouths of healthy persons. The pneumonia associated with type IV in adults is usually of the mildest variety seen. The fact that this is the type of organism usually found in the pneumonias of children probably accounts for the low mortality from primary pneumonia in patients over two years of age. In infants and young children, however, pneumonia associated with type IV may be very severe.

The different forms of pneumonia which will be considered are: (1) Acute bronchopneumonia; (2) acute lobar pneumonia; (3) acute pleuropneumonia; (4) hypostatic pneumonia; (5) chronic bronchopneumonia.

Tuberculous bronchopneumonia will be discussed in the chapter devoted to Tuberculosis.

ACUTE BRONCHOPNEUMONIA

(*Catarrhal Pneumonia; Lobular Pneumonia; Capillary Bronchitis*)

This is essentially the pneumonia of infancy. Under two years, the great majority of the cases of primary pneumonia are of this variety, and throughout childhood nearly all the cases of secondary pneumonia. The term bronchopneumonia describes a lesion rather than a disease, several quite distinct forms of infection being included under this head. Its mortality is high, because of the tender age of the patients in which the primary cases occur, and also because when secondary it complicates the most severe forms of the acute infectious diseases of children.

Etiology.—The distribution, according to age, of 426 cases of bronchopneumonia was as follows:

During the first year	224	cases, or 53	per cent.
" " second year	142	" " 33	" "
" " third "	46	" " 11	" "
" " fourth "	10	" " 2	" "
" " fifth "	4	" " 1	" "
	426	100	

After four years bronchopneumonia is infrequent as a primary disease, although it is seen throughout childhood as a complication of the infectious diseases.

Of the cases referred to, 38 per cent occurred during the winter months, 31 per cent during the spring, 13 per cent during the summer, and 18 per cent during the autumn. While, therefore, nearly 70 per cent of the cases occurred in the cold months, bronchopneumonia is seen throughout the year.

Bronchopneumonia affects all classes, but is most frequent in children having poor hygienic surroundings, especially in inmates of institutions, and in those previously debilitated by constitutional or local disease. In 246 consecutive cases of primary pneumonia, 110 were in good condition prior to the attack, and 126 were delicate, rachitic, or syphilitic.

The following table gives a good idea of the conditions with which acute bronchopneumonia is most frequently seen; 443 cases were classed as follows:

Primary [1]	164
Secondary to bronchitis of the large tubes	41
Complicating measles	89
" pertussis	66
" diphtheria	47
" acute ileocolitis	19
" scarlet fever	7
" influenza	6
" varicella	2
" erysipelas	2
	443

A large number of the patients had previously suffered from one or more attacks of bronchitis, and fifteen previously had bronchopneumonia.

As an exciting cause, exposure to cold must still be classed among the potent factors of primary pneumonia. The organisms concerned in bronchopneumonia have been discussed in the previous pages.

Lesions.—The term bronchopneumonia is now generally adopted as a generic one, and it is to be preferred either to lobular or catarrhal pneumonia, as it gives prominence to the bronchial element in the inflammation. The process may begin in the larger tubes and gradually extend to those of smaller caliber, finally involving the pulmonary lobules in which these tubes terminate; or it may extend to the air vesicles which surround the tube in its course through the lung, so that in whatever direction the lung is cut, there are seen, surrounding the small bronchi, zones of pneumonia (Fig. 48). In other cases the process seems to begin almost at the same time in the small bronchi and the air vesicles, as both are found involved, even when death occurs within a few hours of the first symptoms.

There are, however, cases in which the parts of the lung affected bear no relation to the bronchi—where there are found simply smaller or larger areas of pneumonia irregularly scattered through the lung, usually near the surface (Plate VIII). From the distribution of the lesions such cases might better be termed lobular than bronchopneumonia.

Much has been said in the past about pulmonary collapse from obstruction of the small bronchi, as a condition antecedent to this form of pulmonary inflammation. So far as our observations go, there has been adduced but little evidence that this is the rule, or, indeed, that it often occurs. Even in autopsies made very early in the disease, but little collapse is found, most of the cases supporting the view of Delafield, that when the disease extends from the bronchi to the air cells it involves those surrounding the tube quite as regularly as those to which the tube leads.

[1] It is probable that a number of cases complicating influenza were included among these primary cases.

Acute Bronchopneumonia

Primary pneumonia in a child two years old, showing the irregular distribution of the consolidation and its incomplete character. A is the pleura somewhat thickened; B, lung tissue which is practically normal; C C are consolidated areas, scattered through which are groups of air vesicles still containing air. (Slightly magnified.)

The following observations are made from a study of 170 autopsies of which we have records, microscopical examinations having been made in about one-third of the number.

Seat of the Disease.— In eighty-two per cent of the autopsies extensive

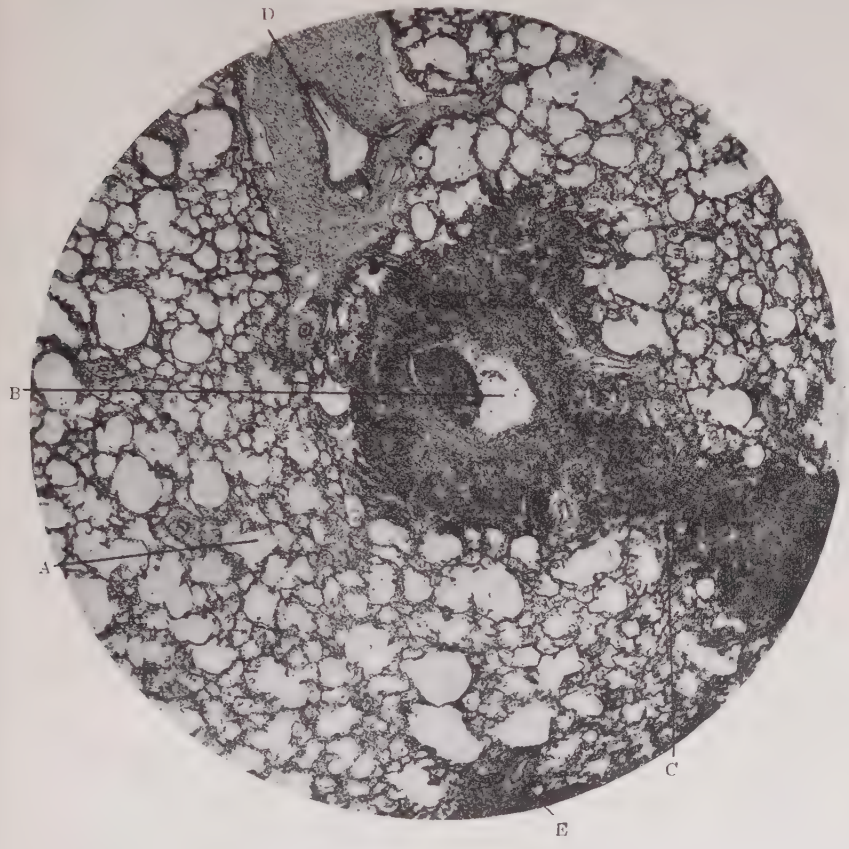


FIG. 48.—BRONCHOPNEUMONIA, WITH THICKENING OF A BRONCHUS. In the center of the picture is seen a small bronchus, B, which is cut somewhat obliquely; the degree to which its wall, C, is thickened is well shown. It is partially filled with pus, its mucous membrane is nearly destroyed, and its walls greatly thickened from infiltration with leucocytes. This infiltration extends to the lung tissue in the neighborhood; it forms a peri-bronchitic zone of pneumonia. Elsewhere in the picture the lung tissue, A, is practically normal. D is a small blood-vessel. E is another smaller bronchus. Throughout the lung everywhere accompanying the small bronchi similar changes were seen, in addition to which there were present some large areas of consolidation. The disease was of four and a half weeks' duration; the child, five months old.

disease was found in both lungs. The parts most affected were the lower lobes posteriorly; next to this the posterior part of both the upper and lower lobes. The left lower lobe was more extensively diseased than the

right in over two-thirds of the cases. If the pneumonia is in front only, the right apex is the most frequent seat.

There are a certain number of cases which appear to follow tolerably well-defined stages of congestion, consolidation, and resolution; but the disease may be arrested at any of the stages and the child recover, or death may occur at any stage and there may be found at autopsy different portions of the lung representing all the stages mentioned. In considering, therefore, the lesions of bronchopneumonia, it seems best to describe the condition in which the lungs are found at the various periods when death is likely to occur, rather than to attempt to describe the different stages of the disease, as in lobar pneumonia.

1. *The Acute Congestive Form (Acute Red Pneumonia).*—This is the condition in which the lung is usually found if death occurs during the first two or three days of the disease. In the cases severe enough to cause death in the first twenty-four hours, very little can be seen by the naked eye except acute congestion. The vessels of the pleura are distended, and there may be small superficial hemorrhages. Both lower lobes are usually heavy and dark colored. There is to the naked eye no consolidation. All, or nearly all, the lung can be inflated. On section, there is found intense congestion with some edema. When the process has lasted a little longer the affected areas are more sharply defined. These, usually the posterior portions of both lungs, are of a brownish-red color, and appear partially consolidated, although with a little force they may in most cases be inflated. After section, pus and mucus flow from the divided bronchi, and the whole lung may be more or less congested or edematous.

The microscope alone reveals the fact that these are not cases of simple pulmonary congestion or bronchitis of the finer tubes. In one case in which death occurred twelve hours from the first symptoms, well-marked evidences of inflammation of the air vesicles were found. In these hyper-acute cases, the microscope shows great distention of all the small blood-vessels of the affected area, and small or large extravasations of blood just beneath the pleura, into the alveoli and interstitial tissue of the lung. In some cases these hemorrhages form the most striking feature of the lesion. The air vesicles are partially, some almost completely, filled with red blood-cells, swollen and desquamated epithelial cells, and a few leucocytes (Fig. 46). The red blood-cells predominate. The inflammation may be diffuse, involving nearly a whole lobe, or in small areas in the neighborhood of the small bronchi. The mucous membrane of the large and small bronchi is the seat of catarrhal inflammation, and the walls of the latter are infiltrated with round cells.

When the process has lasted from twenty-four to forty-eight hours all the changes described are more marked, but the red color of the

inflammatory products still persists. Such cases give during life only the signs of congestion and bronchitis.

2. *The Mottled, Red and Gray Pneumonia.*—This is the usual appearance when the disease has lasted somewhat longer, and is found in most of the cases dying between the fourth and fourteenth days. There are usually at this time quite large areas of consolidation, sometimes affecting nearly an entire lobe, so that at first sight the case may resemble

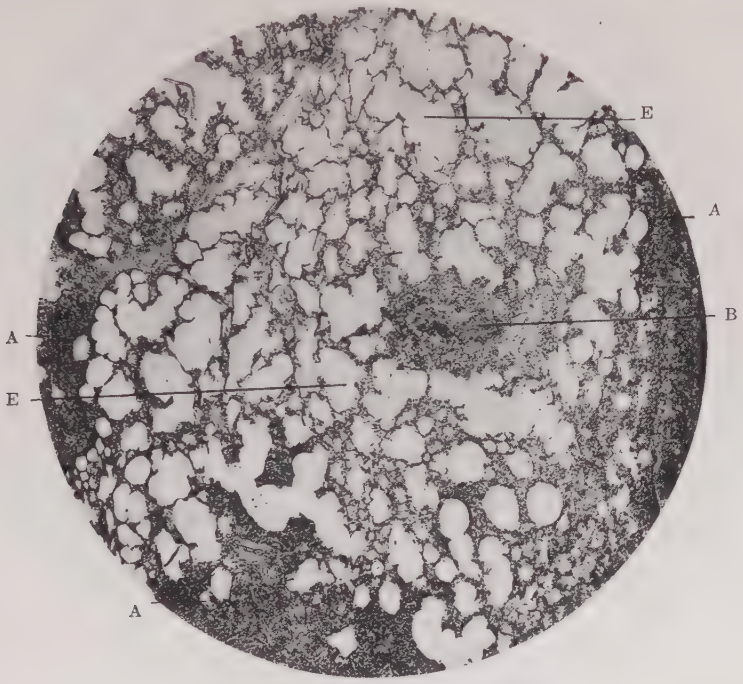


FIG. 49.—ACUTE BRONCHOPNEUMONIA. In the center is shown a small bronchus, B, with a zone of pneumonia about it. The greater part of the section is made up of emphysematous lung tissue, E E, showing dilatation of the alveolar spaces and rupture of some of the alveolar septa. At the border, A A A, are seen the margins of consolidated areas of lung.

lobar pneumonia. This is sometimes described as the "pseudo-lobar" form. The extent of these areas depends largely upon the duration of the disease. In most cases there is pleurisy over the consolidated portions. This may cause the lung to adhere to the chest wall, the firmness of the adhesions depending upon the duration of the process. The surface of the lung is usually of a mottled red and gray color; it often has a coarsely granular feel, due to the consolidation of some of the superficial lobules of the lung. On section, it is rarely found that an entire lobe is consolidated, the superficial portion being most affected, while

the central part is normal or only congested. The color is mottled, like that of the surface. In some places the consolidation appears complete; in others the consolidated areas are separated by healthy, congested, or emphysematous lung tissue (Fig. 49). The gray areas surround the small bronchi and vary in size. The smallest ones look very much like

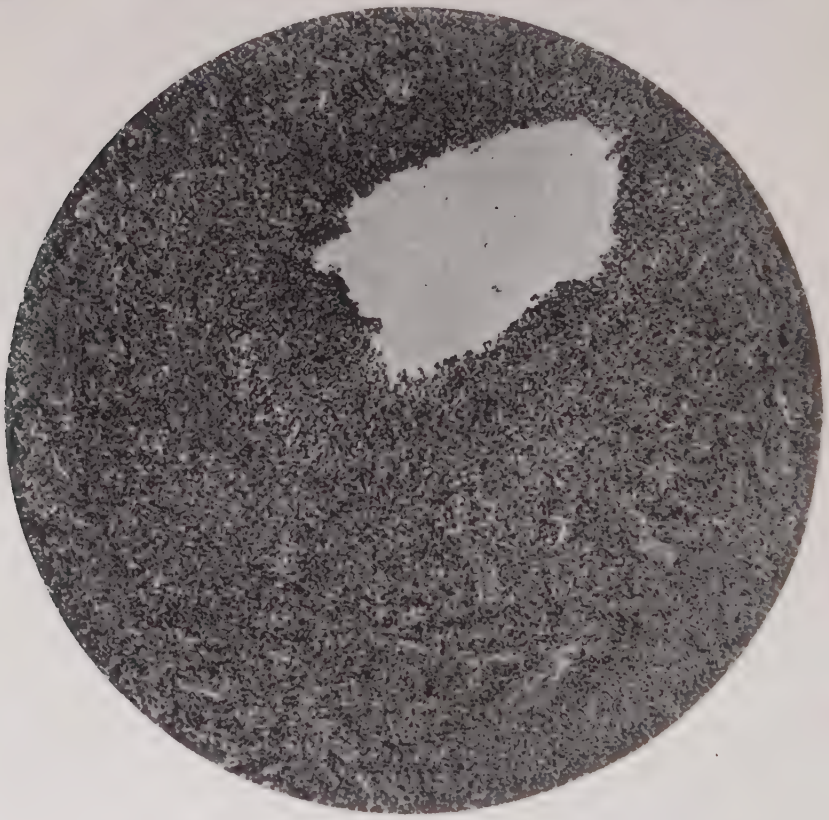


FIG. 50.—BRONCHOPNEUMONIA. Dense infiltration of pus cells in and about a small bronchus; under a low power. The cavity shown in the specimen is a cross-section of one of the small bronchi, which is partially filled with pus cells; the epithelium is destroyed. The bronchial wall and the pulmonary tissue in the neighborhood are so densely infiltrated with leucocytes that almost every trace of normal structure is effaced. Child fifteen months old, disease of four weeks' duration. Extensive areas like this were found in both lungs.

miliary tubercles. The larger ones are seen where the process has existed for a longer time and has gradually invaded the contiguous air cells. If the lung is cut parallel with the bronchi, there may be seen small gray striae of pneumonia along their course (Fig. 48, C). From the cut bronchi, pus flows quite freely on pressure. The bronchial walls are often seen to be thickened even by the naked eye. The parts affected

are usually the posterior portions of the lower lobe of one or both sides, the remainder of the lobes being congested or edematous, while in front the lung is emphysematous.

Under the microscope the smaller bronchi (Fig. 48) are seen to be much thickened and infiltrated with leucocytes. The gray areas surrounding the bronchi are made up of groups of air vesicles, which are packed with leucocytes (Fig. 50). Fibrin is sometimes seen in small amount, also red blood-cells and desquamated epithelial cells, but the leucocytes predominate. Surrounding the areas densely infiltrated are groups of air vesicles which are normal or congested, or which show only the earlier stages of the inflammatory process.

3. *Gray Pneumonia* (*Persistent Bronchopneumonia*).—This form is seen in protracted cases when there have been continuous symptoms usually for from three to six weeks. The pleuritic adhesions are more general and firmer. The amount of lung involved may be very great, often nearly the whole of both lungs posteriorly. The affected lung appears completely consolidated and slightly enlarged. On section, it is of a nearly uniform gray color, sometimes of a yellowish-gray. On pressure, pus exudes from the smaller and larger bronchi. The bronchial walls are markedly thickened, and in some places there may be a slight dilatation of the smaller bronchi. The part of the lung not consolidated may be almost white, owing to vesicular emphysema. In some cases there is also interstitial emphysema. Small cavities containing pus may be found in the lung. The bronchial glands are frequently swollen to the size of a large bean, and are of a reddish-gray color.

The microscope shows that the air vesicles of the consolidated portions are distended chiefly with leucocytes, but there are also epithelial and connective-tissue cells. The alveolar septa may be so much thickened as to encroach upon the alveolar spaces (Fig. 51). Complete resolution is then impossible.

Termination.—Death may occur at any stage, or the pathological process may be arrested at any stage and the case go on to recovery. Resolution may take place before any consolidation recognizable by physical signs has occurred; in such cases it is usually rapid and complete. If there has been consolidation, resolution may take place after two or three weeks and be complete, or it may be delayed for five or six weeks and still be complete. In many cases, especially those in which it is delayed, resolution is only partial, and there are relapses or recurring attacks. After the first, or after several attacks, there may develop a chronic interstitial pneumonia; or simple pneumonia may be followed by tuberculosis. Such cases as these are to be carefully distinguished from the much more frequent ones in which the bronchopneumonia is tuberculous from the outset.

Associated Lesions of the Lungs.—*Pleurisy* is almost invariably found over every large area of consolidation, and in cases of more than three or four days' duration; while in most of those fatal within the first few days the pleura is normal or only congested. It is seen in all grades of severity, from a slight gray film of fibrin that can hardly be stripped off, to a yellowish-green exudation one-fourth of an inch

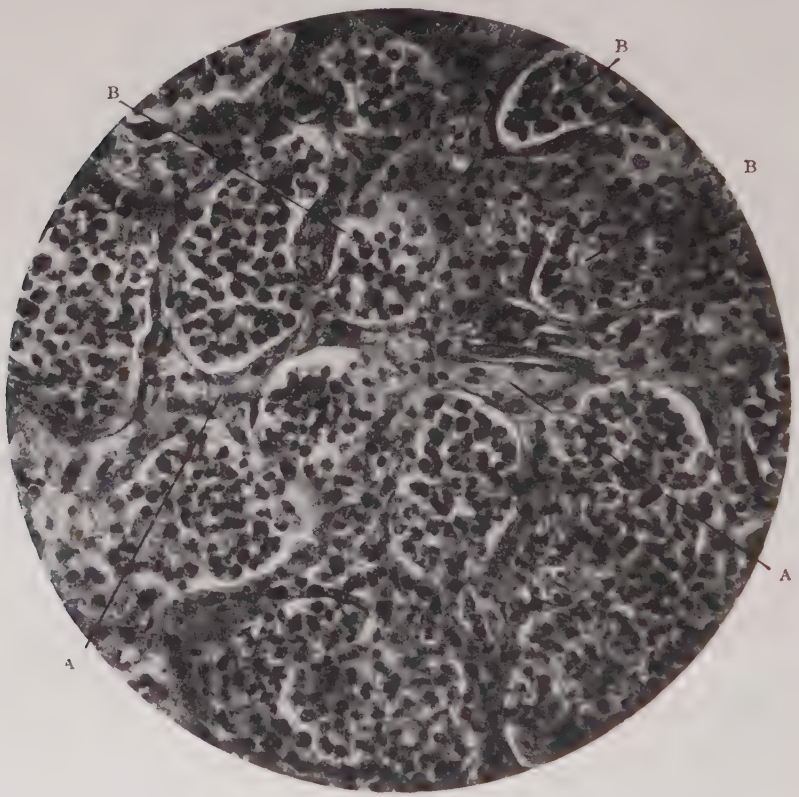


Fig. 51.—Persistent Bronchopneumonia; Highly Magnified. There is shown at A A marked thickening of the alveolar septa, encroaching upon the alveolar spaces. All the alveoli, B B, are densely packed with leucocytes. A similar condition also through nearly the whole of the affected lung. (For history and temperature, see Fig. 60.)

thick. A small amount of serum—two or three ounces—in the pleural sac is common, but a large serous effusion is very rare. Cases in which there is an excessive inflammation of the pleura are considered elsewhere under the head of Pleuropneumonia. Empyema occurs both during the stage of acute inflammation of the lung and while this is subsiding, but it is less frequent than in lobar pneumonia.

Bronchial Glands.—In all the recent acute cases these are swollen

and red; the usual size is that of a pea or a bean. They show microscopically the usual changes of acute hyperplasia. In protracted cases, and after repeated attacks, they may be two or three times the size mentioned, and of a gray color. It is rare that they are large enough to give rise to symptoms unless they become the seat of tuberculous deposits.

Emphysema.—This is one of the regular and striking features of acute brochopneumonia in infancy, it being especially marked in the protracted cases. It is usually vesicular, involving the greater part of the upper lobes in front and the anterior margin of the lower lobes. Occasionally interstitial emphysema is seen, forming either large striae upon the surface of the lung, or blebs of considerable size along the anterior margin. This may occur even in cases uncomplicated by pertussis or by laryngeal stenosis.

Gangrene.—Gangrenous areas were found in six cases of the series mentioned. In four of these the pneumonia was primary, in one it followed diphtheria, and in one ileocolitis. It occurred in scattered areas of a grayish-green color, varying from one-fourth of an inch to two inches in diameter.

Abscesses of the lung are by no means uncommon. They were noted in seven per cent of the autopsies. They are usually minute and multiple, varying in size from one-sixth to one-half inch in diameter. Sometimes a portion of a lobe is fairly honeycombed with minute abscesses. In one case a large abscess was found occupying the greater part of a lobe, the symptoms resembling those of empyema. Abscesses are usually found in regions where the inflammatory process has been especially intense. They may be found in prolonged cases, in those of unusual severity, as shown by excessively high temperature and rapid extension of the disease, and in very delicate subjects. The microscope shows that these abscesses usually begin as an accumulation of pus in the small bronchi, whose walls become softened and break down on account of the intensity of the inflammation. They may be superficial, but are more commonly in the interior of the lung; they contain yellow pus and sometimes broken-down lung tissue. Small abscesses can not be recognized clinically; the large ones give the symptoms and signs of empyema. They are discussed more fully elsewhere. In several instances they have been successfully operated on, though wrongly diagnosticated.

The lesions in other organs will be considered under Complications.

Symptoms.—Bronchopneumonia has no typical course. The cases differ from each other very markedly, but they may be divided into a few quite distinct groups.

1. The Acute Congestive Type.—This may be seen at any age, but is more frequent in young infants. It may be either primary or sec-

ondary, being not uncommon in either form. Its symptoms are few and irregular, and the disease is often unrecognized. The entire duration may be only twenty-four hours. High temperature, extreme prostration, cyanosis, and rapid respiration may be the only symptoms. The temperature varies between 104° and 107° F., usually rising steadily until death occurs. The prostration is extreme from the outset, the patient being overwhelmed by the suddenness and severity of the attack. Cyanosis is frequently present, and is almost always seen shortly before death. The respirations are from 60 to 80 a minute, but in most cases not strikingly labored. Cough is frequently absent. Cerebral symptoms are often marked—dulness and apathy, sometimes quite profound stupor, and not infrequently convulsions just before death. The physical signs are few and inconclusive. There is often nothing abnormal except very rude breathing over both lungs behind; sometimes the breathing on one side is feeble, and on the other much exaggerated. There may be no râles whatever, and no change in the percussion note.

The suddenness and severity of these symptoms are something which it is hard for one who has not observed them to appreciate. We have known an infant to die in twelve hours from the time in which he was apparently in perfect health, and had an opportunity to confirm the diagnosis of pneumonia by a microscopical examination of the lung. The diagnosis can not be positively made during life, and in most of the cases the disease passes under some other name. It is often regarded as malignant scarlet fever or measles with suppressed eruption, or cerebro-spinal meningitis.

If the children are sufficiently strong to withstand the onset of violent symptoms, they may recover completely in four or five days, the lung clearing up very rapidly. In other cases these grave symptoms may abate in a day or two, to be followed by those of ordinary broncho-pneumonia, which runs its usual course.

The symptoms of some of these cases may be explained by the sudden intense engorgement of the lung, which, owing to the small size of the air vesicles, interferes with its function almost as much as does consolidation. In other cases the symptoms are due not so much to the pulmonary condition as to a general pneumococcus infection. We have seen cases of pneumonia fatal in less than two days in which the pneumococcus was found by post mortem cultures to be disseminated through the organs of the body.

2. Acute Disseminated Bronchopneumonia (Capillary Bronchitis).—Although the symptoms in this class of cases are chiefly due to the bronchitis, there are always evidences of pneumonia to be found post mortem. These are not very common cases. The process begins as an inflammation of the medium-sized and small bronchi, but not of

the finest bronchi. The onset is acute, with fever, very rapid and labored breathing, severe cough, moderate prostration, and in most cases cyanosis.

The temperature is not high, usually only from 100° to 102° F., and it often continues so for three or four days. The pulse is rapid, and at first is full and strong. The respirations are exceedingly rapid, often from 80 to 100 a minute. There is dyspnea with marked recession of all the soft parts of the chest during inspiration. Cough is always present, usually severe, and sometimes almost incessant. The prostration is not so great as in the cases previously described, and the development of the symptoms is much less rapid.

There are at first sibilant and afterward subcrepitant râles over the entire chest, with which are usually mingled coarser moist râles. There are no evidences of consolidation. The respiratory murmur is everywhere feeble, but not otherwise altered. Percussion generally gives exaggerated resonance, owing to the emphysema which is present, the note being sometimes almost tympanitic.

The symptoms may gradually increase in severity until death takes place by the third or fourth day, from respiratory or cardiac failure. There is usually marked cyanosis, and toward the end rapidly increasing prostration. Just before death the temperature often rises rapidly to 106° or 107° F. At the autopsy there are found evidences of bronchitis of the tubes of all sizes, and minute zones of pneumonia about the smaller bronchi. The lungs are generally in a state of hyper-inflation, on account of which they do not collapse on opening the chest. There may be in addition extensive congestion or edema, the development of which has been the immediate cause of death.

In cases which do not prove fatal there is usually by the third or fourth day great improvement in the general symptoms; the finer râles may disappear, and the coarse ones become more and more prominent. By the end of a week there may be complete recovery. Instead of this, there may be a continuance of the constitutional symptoms, and disappearance of the fine râles in front only, while behind there are gradually added to them the signs of consolidation in one of the lower lobes near the spine. From this time the case may progress as one of ordinary bronchopneumonia.

The prognosis in this class of cases is very much better than in the congestive variety, recovery being probable unless the patients are very young or delicate infants.

3. Bronchopneumonia of the Common Type.—When primary, this usually begins suddenly with symptoms not unlike those of lobar pneumonia. This is the mode of onset in about two-thirds of the cases. In only about ten per cent is the pneumonia preceded by bronchitis of the

large tubes. In these the symptoms of bronchitis may slowly or rapidly merge into those of pneumonia. When the onset is sudden it is marked by high fever, frequently by vomiting, rarely by convulsions. In addition there are rapid respiration, cough, prostration, and sometimes cyanosis. The symptoms are more distinctly pulmonary than is generally the case in lobar pneumonia.

The temperature, as a rule, is high; rarely is it continuously so, but it is of a remittent type. The daily fluctuations often amount to four or five degrees. The fever usually continues from one to three weeks, and subsides gradually rather than by crisis, though crises are by no means rare. Although, as a rule, we expect a high temperature with acute pneumonia, this is not invariable. Primary cases may run their course, and even terminate fatally, although the temperature has not been above 101° F. We have records of several such cases. A low temperature is more often seen in young and delicate infants than in those who are older and more robust.

The respirations are frequent and labored; there is real dyspnea. On inspiration, there are marked recessions of all the soft parts of the chest, and the alae nasi dilate actively. The usual rapidity of the respirations is from 60 to 80 per minute; very often, however, it rises to 100, and on several occasions we have seen it even 120. Respiration generally seems more embarrassed than does the action of the heart, and respiratory failure is a more frequent cause of death than cardiac failure. The pulse is always rapid—from 150 to 200 a minute—and when so it is often irregular. The pulse rate is of much less importance than its character. Early the pulse is full and strong, but soon it becomes soft, compressible, and weak.

The prostration is usually moderate for the first day or two, but steadily increases as the lung becomes more and more involved, and toward the close of the disease may be extreme.

Cough is much more constant than in lobar pneumonia, and more distressing; sometimes it is almost incessant. It disturbs rest and sleep, and may cause vomiting if the paroxysm occurs soon after eating. There is no expectoration. Mucus is sometimes coughed up into the trachea, or even into the pharynx, to be swallowed again, or more frequently aspirated into the lung. If during a severe paroxysm the patient is turned upon his face or inverted, much of this mucus may be dislodged. A strong cough is a good symptom; suppression of the cough is a bad symptom, indicating a loss of the reflex sensibility of the bronchial mucous membrane and of the respiratory center.

Pain in the chest is not common, and is rarely an annoying symptom. Cyanosis is present at some time in most of the severe cases. It may occur at the onset, or at any time during the course of the disease.

It is usually due to sudden congestion of a portion of the lung not previously involved. Even when slight, it is always a danger-signal of respiratory failure, and when present only in the finger tips or lips indicates that the patient must be carefully watched and energetically treated. In the severe cases the whole body may be of a dull leaden hue.

Nervous symptoms at the onset are not so frequent as in lobar pneumonia, convulsions being rare; but late convulsions, particularly in the pneumonia which complicates pertussis, are frequent, and when present the disease is usually fatal. Delirium may occur at any time during the attack. In infants this shows itself by excitement and inability to recognize the nurse or mother. Occasionally patients present marked cerebral symptoms throughout the disease closely simulating those of meningitis. As elsewhere stated, the nervous symptoms depend less upon the location of the disease than upon its extent, the intensity of the infection, and upon the susceptibility of the patient, such symptoms being especially common in rachitic children and in those suffering from pertussis.

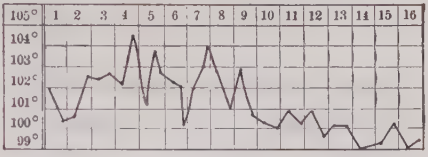


FIG. 52.—TEMPERATURE CURVE IN TYPICAL BRONCHOPNEUMONIA OF THE MILDER FORM. *History.*—Male, sixteen months old; delicate child; previous bronchitis; onset gradual; signs of consolidation at left base on fifth day, but fine râles over both lower lobes behind; resolution slow, râles persisting for a long time in both lungs.

Gastro-enteric symptoms are frequent in infancy, and are of much importance. Often there are from four to six stools a day, of a green color, containing mucus and undigested food. These symptoms depend upon the feeble digestion which is associated with the febrile process, and are often aggravated by improper feeding and overmedication. Vomiting and diarrhea add much to the danger of the attack. In summer this complication is more frequent and is likely to be more severe. Distention of the stomach or intestines from gas may be the cause of distressing symptoms, owing to the added embarrassment of respiration produced by this upward pressure. In infants it may lead to attacks of cyanosis and even to convulsions.

The blood in acute bronchopneumonia shows regularly the changes of a moderate secondary anemia, which in protracted cases becomes very marked. A leucocytosis is almost invariably present. In an average case this ranges from 20,000 to 40,000. It sometimes is excessively high without any apparent reason. We have several times seen it over 100,000. The increase is chiefly in the polymorphonuclear cells which usually form from sixty to eighty-five per cent of the total leucocytes. With

the fall in temperature the leucocytosis in most cases rapidly disappears. A rapid diminution in the leucocytosis may indicate a marked loss of resistance in the patient; and may be seen with either a high or a low temperature. In the pneumonia which complicates pertussis, the increase in the white cells may be chiefly of the lymphocytes.

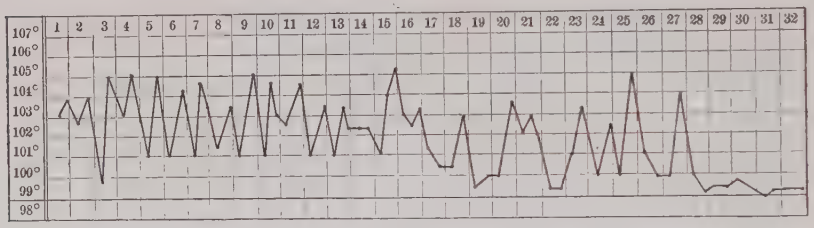


Fig. 53.—Temperature Curve of Bronchopneumonia with a Prolonged Course; Recovery. *History.*—Female, eighteen months old; in fair condition; sudden onset. Early signs were localized, fine râles over left base; on fifth day signs of consolidation at left base, with râles on both sides behind. General symptoms of moderate severity. Signs of consolidation disappeared about a week after cessation of fever; râles persisted nearly two weeks longer.

Positive blood cultures were obtained in 75 of 315 consecutive cases of bronchopneumonia studied at the Babies' Hospital. The pneumococcus was found in 47, the streptococcus in 15 cases.

The urine in most cases is scanty, high-colored, and loaded with urates. A trace of albumin is often present when the temperature is

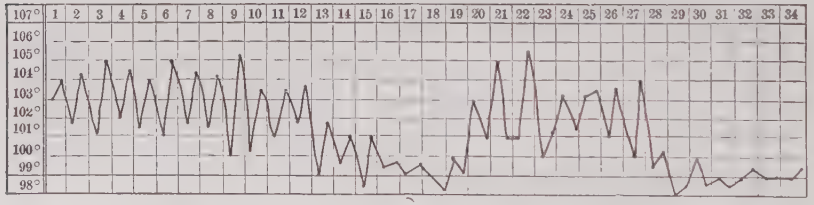


Fig. 54.—Temperature Curve of Relapsing Bronchopneumonia; Recovery. *History.*—Male, nineteen months old; delicate. Consolidation on sixth day in left lower lobe behind; two days later small area of consolidation in right lower lobe behind; many râles both sides; eighteenth day, signs of consolidation had disappeared, but many râles persisted. Accession of fever on nineteenth and twentieth days, accompanied by extension of disease as shown by new râles, but no evidences of consolidation during second attack. Slow resolution and convalescence.

very high; but casts, renal epithelium, and a large amount of albumin are rare.

The temperature chart shown in Fig. 52 is a good example of a very frequent course of primary pneumonia of moderate severity terminating in recovery. In cases of this type the constitutional symptoms are not grave, and follow very closely the temperature curve.

The next chart (Fig. 53) illustrates a more severe but not uncommon course of the disease in which the fever is prolonged. The usual duration of cases of this type is between three and four weeks. The irregular fluctuations of the temperature, rarely touching the normal line, are exceedingly characteristic of bronchopneumonia.

The chart shown in Fig. 54 is that of relapsing pneumonia. The first attack was fairly typical, with about the usual duration. Resolution had begun, and was apparently progressing favorably, when there was a return of the fever, accompanied by new signs in the chest, the second attack being shorter and milder than the first. Very often the temperature falls to normal without any signs of resolution, and after an interval varying from two to three days to a week there is a recurrence of the fever and other constitutional symptoms, the second attack frequently proving fatal.

A frequent course in fatal cases is shown in Fig. 55. The duration of the disease, instead of being five days as in this case, is often only three or four. The temperature at first fluctuates widely, then rises gradually until death.

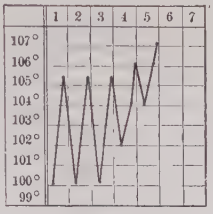


FIG. 55.—TEMPERATURE CURVE OF BRONCHOPNEUMONIA; FATAL. *History.*—Male, six months old; markedly rachitic; sudden onset. Signs first day were fine moist râles throughout the chest, marked prostration, and cyanosis; on third day, a small area of consolidation in upper lobe of left lung behind; increasing prostration, cyanosis, and death. *Autopsy.*—No pleurisy; consolidation at left apex behind, and posterior two-thirds of left lower lobe; consolidation of right apex posteriorly, lower lobe intensely congested.

Duration of the Fever.—The following figures give the duration of the fever in 231 cases. The majority were primary; none were secondary to diphtheria, and only a few complicated measles. Of the 169 cases that were fatal—

There died during the first six days	25.0	per cent.
" " between the seventh and twenty-first days	55.5	" "
" " " " twenty-first and sixtieth days	19.5	" "
	100.0	" "

Of 78 cases which recovered, the duration of the fever was—

Less than seven days	11.5	per cent.
From seven to twenty-one days	66.6	" "
" twenty-one to ninety days	21.9	" "
		" "
	100.0	" "

Physical Signs.—In considering the signs of bronchopneumonia, it is better to connect them with the different conditions in the lung than to group them in stages, as in lobar pneumonia.

(*a*) *Without Consolidation.*—It can not too often be repeated that bronchopneumonia may exist without signs of consolidation at any period during the course of the disease. When the attack is primary, the earliest signs are due to congestion of the lung associated with bronchitis of the fine tubes, which is usually localized, but which may be general. If the disease has followed bronchitis of the large tubes, its signs are added. Congestion of the lung gives feeble breathing over the affected area and occasionally slight dulness or diminished resonance. With this are found coarse sonorous, and finer sibilant râles, due to congestion and swelling of the mucous membrane of the larger and smaller bronchi respectively. These signs are soon replaced by very fine moist râles, which are usually localized in one of the lower lobes behind (Fig. 56). These localized fine râles are the first distinctive sign of bronchopneumonia. Soon a change in the respiratory murmur is heard in the affected area, which becomes feebler in intensity and higher in pitch. Elsewhere in the chest there may be coarse râles, due to bronchitis of the large tubes. In such cases the areas of pneumonia are so small and so scattered as to give in themselves no additional signs, and the case may go on to recovery without presenting anything more distinctive than the signs mentioned.

(*b*) *With Areas of Partial Consolidation.*—In the lung at this time there are small areas of consolidation, generally superficial and separated by healthy or congested lobules. Percussion in these cases may give negative results or there is slight dulness. The vocal fremitus is not usually altered. The fine moist râles may be heard over quite a large area, but at some point, usually near the spine, over one of the lower lobes, they are sharper, louder, higher pitched, and more metallic, and seem close under the ear (Fig. 57). Respiration is feebler here than elsewhere, and bronchovesicular in quality, approaching bronchial breathing more and more as the consolidation increases. The resonance of the voice and cry is exaggerated.

(*c*) *With Areas of Consolidation More or Less Complete.*—On percussion there is dulness, but surprisingly little in comparison with the other signs of consolidation present. It is due to the fact that the consolidated portion, though extensive, does not involve the lung to any great depth, and also that there are in the consolidated area many alveoli which still contain air (Plate VIII). On palpation there is usually a slight increase in the vocal fremitus. On auscultation, there are still present the evidences of bronchitis, usually only behind, but sometimes over the entire chest. Coarse and fine râles are intermingled. Over the consolidated parts are heard bronchial breathing and bronchial voice. At the center of these areas the bronchial

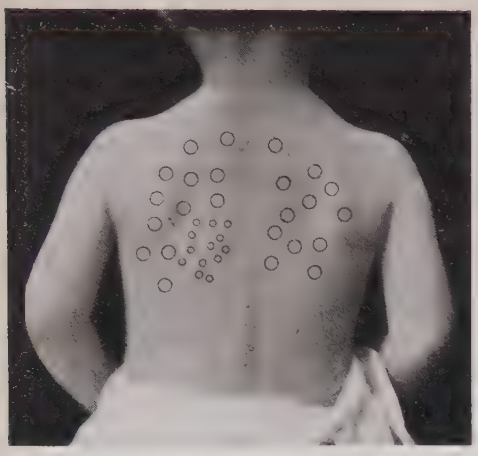


FIG. 56.—FIRST STAGE. Coarse râles over both lungs; localized fine (subcrepitant) râles at the left base. No change in breath sounds.

FIG. 57.—SECOND STAGE. Coarse and fine râles over both lungs behind; at left base an area of partial consolidation, with bronchovesicular breathing, exaggerated voice, and very sharp râles.

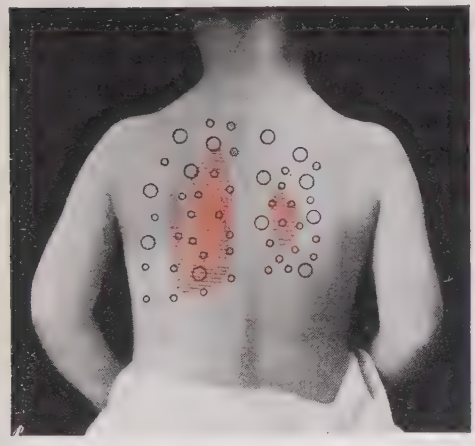


FIG. 58.—THIRD STAGE. A larger area of partial consolidation, and in the center a small area of complete consolidation, with bronchial breathing and voice and slight dulness. Signs over the right lung similar to what were previously present over the left.

FIG. 59.—FOURTH STAGE. Extensive disease of both sides; large area of complete consolidation on the left, with dulness, bronchial breathing and voice, and no râles; surrounding this, bronchovesicular breathing, with many râles. Signs in the right lung similar to those previously present over the left.

NOTE.—The large circles indicate coarse râles; the small ones finer râles; the red areas indicate consolidation partial or complete. The disease may stop at any one of these stages and resolution take place.

breathing is pure and râles are usually absent, but at the margin râles are present and the breathing approaches the bronchovesicular type (Fig. 58). The signs of consolidation are rarely sharply circumscribed as they are in lobar pneumonia, but shade off gradually. The consolidated area is at first small, usually in one of the lower lobes near the spine, but may gradually extend until nearly the whole of one or even both lungs behind are more or less completely solidified (Fig. 59). The signs are found as far forward as the axillary line, but usually stop there. Friction sounds may be heard over the consolidated areas, but very rarely except where signs of complete consolidation are present. It is often impossible to obtain any idea of the condition of an infant's lung during quiet, superficial respiration. Sometimes over a part which is completely consolidated there is heard only very feeble breathing, or the lung may be almost silent. If, however, the child is made to cry or to take a deep inspiration, both the bronchial breathing and râles are distinctly brought out. The intensity of the consolidation increases as the disease advances, and the signs become more and more like those of lobar pneumonia. During resolution there is first a disappearance of the signs of consolidation, which may be quite rapid, but friction sounds and râles of all kinds often persist for three or four weeks longer.

The following statistics are of some interest, as showing the frequency with which signs of consolidation were found, and the day when they were discovered. Their value is increased by the fact that the children were under observation in an institution at the time they were taken sick, and that in all the fatal cases—thirty-six in number—in which signs of consolidation were absent, the diagnosis of pneumonia was confirmed by autopsy:

Consolidation noted on or before the fourth day	47	cases
" " from the fifth to the seventh day	36	"
" " " the eighth to the twelfth day	12	"
" " after the twelfth day	9	"
No signs of consolidation	62	"
	166	"

In general, it must be borne in mind that in many cases signs of consolidation are never present, as the areas of pneumonia are small and widely scattered; that where there is consolidation it is usually incomplete, because there are small areas of healthy lung tissue between the hepatized portions; that the signs of consolidation usually shade off gradually; and that both sides are almost invariably involved, although one side usually to a greater degree than the other.

4. The Protracted Form—Persistent Bronchopneumonia.—This is seen in primary cases, especially among delicate children, and in the pneumonia complicating pertussis, influenza and measles, and is the form which often follows diphtheria. The onset and course of the disease for the first two or three weeks do not differ from an ordinary attack of moderate severity, but at the end of this period there is seen no tendency in the process to subside. The fever continues, although it may not be high, but by physical examination it is found that the areas of consolidation are gradually increasing day by day, until sometimes the greater part of both lungs behind are involved. The air vesicles become so distended with cells that the signs of consolidation are more complete than in ordinary bronchopneumonia. The physical signs present are

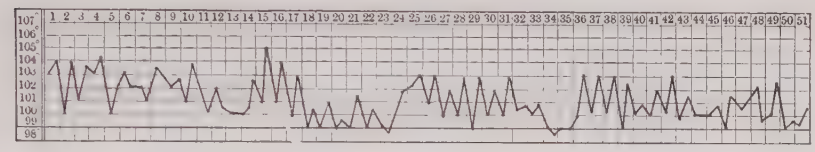


Fig. 60.—Temperature Curve of Persistent Bronchopneumonia, Terminating Fatally. *History.*—Male, two and a half years old; healthy; sudden onset; for two weeks the only signs were very fine moist râles throughout both lungs, front and back. The râles in front in great part gradually cleared up; those behind persisted, but it was not until the thirty-fourth day that positive signs of consolidation were discovered in the left lower lobe behind; these signs gradually extended, and, before death, were present over nearly the whole left lung behind and over the right lower lobe. There were also friction sounds over both lungs. *Autopsy.*—Old and recent pleurisy with general adhesions; left lower lobe completely solid, patches of consolidation in left upper lobe. Right lower lobe about one-half consolidated, with patches elsewhere. Bronchial glands large, but not cheesy. No evidence of tuberculosis upon either gross or microscopical examination (see Fig. 51).

marked dulness, sometimes almost flatness; there is bronchial breathing which is exaggerated in intensity until it resembles cavernous breathing, and it may be impossible to distinguish between them. However, the fact that it is heard over so large an area, that it shades off gradually, and that it is accompanied by friction sounds, usually make a distinction possible.

The temperature in these protracted cases for the first two or three weeks is from 100° to 105° F.; but after this time it is generally lower —from 100° to 102° or 103° F. The course is not at all regular, but marked by frequent exacerbations and remissions. The general symptoms are those of progressive asthenia. There is continued wasting, anemia, and steadily increasing prostration. The appetite is lost, often there is an aversion to food, and vomiting is easily excited if food or stimulants are forced. The stools show that even what food is taken is very imperfectly digested and assimilated. The skin becomes dry and loses its elasticity; bed-sores may form; fine punctate hemorrhages are

seen over the abdomen, sometimes over the chest and extremities. This condition is always a very bad symptom, and recovery from pneumonia is very seldom seen when it is present.

Death takes place from slow asthenia, usually after five or six weeks, but the attack may be prolonged for eight or ten weeks. The general symptoms, the temperature, and the wasting strongly suggest tuberculosis, and such is the diagnosis often made.

Although the majority of the cases in which the fever lasts over four weeks run the fatal course just described, such apparently hopeless cases occasionally recover. The temperature gradually falls lower and lower, until it remains at the normal point. For some time after this, often two or three weeks, little change can be seen either in the general symptoms or in the physical signs. Gradually the appetite returns, the child is brighter and begins to take an interest in his surroundings, the cough abates, and little by little the signs in the lungs clear up, and the child may recover completely. Convalescence, however, is always slow, and may be interrupted by relapses, it being many months before health is fully restored. Although the signs of consolidation disappear in a few weeks, râles are apt to persist for a much longer time. It is probable in such cases, even though all signs of disease disappear from the chest, that the lung does not become normal. Relapses and second attacks are always possible and indeed frequently occur. The area involved in the relapse always includes that part of the lung in which resolution was delayed. The general health may be so undermined that the child never regains his former vigor; yet in a surprising number of these cases recovery seems to be complete. Protracted cases of a mild type are sometimes seen, and, although the temperature persists for a number of weeks, it is never high. The course of the disease suggests tuberculosis.

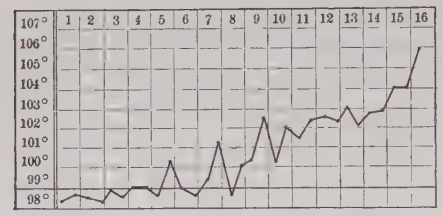


Fig. 61.—Temperature Curve of Fatal Bronchopneumonia, Complicating Pertussis. *History.*—Male, six months old; delicate; pertussis for three weeks. Early signs of bronchitis of large tubes only; on the eleventh day signs of consolidation in right upper lobe. Increasing prostration, cyanosis, and death. *Autopsy.*—Large areas of consolidation in right middle and upper lobes, small scattered spots throughout left lung.

5. Secondary Pneumonia.—(*a*) *Complicating Pertussis.*—It is not often that pneumonia develops during the first two weeks of this disease. The most frequent time is from the third to the fifth week, when the patient has become exhausted from the previous severity of the pertussis. In two-thirds of our cases the development of the pneumonia

was gradual, following bronchitis of the larger tubes. The temperature chart shown in Fig. 61 well illustrates this course.

When the onset is sudden, the symptoms do not differ essentially from those of primary pneumonia. The temperature of pertussis-pneumonia is usually not high, in a very large number of cases not rising above 103.5° F., and ranging most of the time from 101° to 103° F. These cases are very apt to be prolonged, the fever often lasting for three or four, and sometimes even for six weeks. The physical signs of consolidation may persist for a long time after the temperature has become normal, and yet the child may recover entirely. We have seen one case in which recovery apparently complete occurred after the signs of consolidation had persisted for six months, and another in which they had persisted for over eight months. Very often the signs continue during the entire attack of pertussis. Cerebral symptoms are common, especially toward the close of the disease. Of fifty-four fatal cases, twenty-five had convulsions, and in twenty-two this was the mode of death. Only one case which developed convulsions recovered.

(*b*) *Complicating Measles.*—In a small number of cases the pneumonia begins simultaneously with the invasion of measles, but generally not until the eruption appears. Instead of gradually falling to normal with the fading of the eruption, the temperature continues high. Any of the clinical types of primary pneumonia may occur in measles, the acute congestive variety, which is fatal in two or three days, being especially common. In its course and duration the pneumonia of measles resembles the severe form of primary pneumonia. The bronchopneumonia of scarlet fever differs in no way from that of measles.

(*c*) *Complicating Diphtheria.*—In many cases this does not give a distinct clinical picture of its own, its symptoms being mingled with those of diphtheritic bronchitis, with which it is frequently associated. In others the forms resemble those seen in measles. The majority of cases occur as a complication of diphtheria of the larynx, although it is not infrequent in the septic cases in which only the upper air passages are involved. Pneumonia after laryngitis may develop within two days from the beginning of laryngeal symptoms, and run a rapid course; or it may come as late as the second or third week. In a child wearing an intubation tube, the diagnosis of pneumonia presents difficulties, owing to the alteration in the respiratory sounds and the existence of the loud tracheal râles which obscure the usual auscultatory signs. Although pneumonia may be apparent by symptoms, its situation may be difficult to determine. The most important signs for diagnosis are the diminished respiratory murmur, localized râles, and dulness on percussion.

(*d*) *Complicating Influenza.*—Without doubt many cases usually regarded as primary are really secondary to influenza, particularly when

that disease is prevalent. While the pneumonia of influenza may differ in no essential points from the primary form, there are types which are quite characteristic. In one variety the cases are of short duration, frequently lasting but three or four days, but with high and often widely fluctuating temperature, the general symptoms being of only moderate severity. A second type is a prolonged pneumonia with exacerbations and remissions, which may last for two or three months with quite extraordinary fluctuations of temperature (Fig. 62). A third form is the recurrent type of pneumonia, of which a child may have several

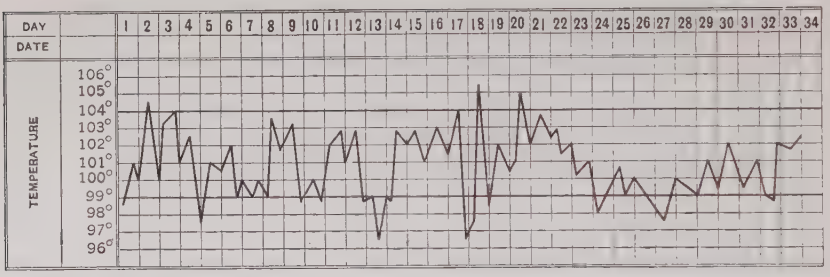


FIG. 62.—BRONCHOPNEUMONIA COMPLICATING INFLUENZA; DEATH. *History.*—Delicate infant, 7 months old, bronchitis and otitis four weeks before. Acute onset, early signs of consolidation in right lung; double paracentesis for otitis on 13th day; small area of consolidation in left lung on 16th day. Sputum cultures repeatedly showed B. influenzae. Signs in lungs not much changed; death from exhaustion. *Autopsy.*—Usual lesions of bronchopneumonia of moderate extent in both lungs. No other lesions of importance. Cultures from lungs showed the B. influenzae but no pneumococci.

distinct attacks in a single season, although in the interval neither signs nor symptoms entirely disappear. In a certain number of these cases a chronic form of pneumonia ultimately develops.

(*e*) *Complicating Ileocolitis.*—This is usually a somewhat subacute form of pneumonia which is scarcely recognizable except by the physical signs. It is seen in the protracted cases of ileocolitis and occurs late in its course. Very often pneumonia is not suspected during life, the constitutional symptoms being sufficiently explained by the intestinal lesions, although the autopsy discloses the fact that death was due in part to pneumonia.

Complications.—Most of those relating to the lungs have been described with the lesions. Pleurisy will be separately considered. Pulmonary emphysema is always present to a greater or less degree, but can not be made out by physical signs. In very rare instances subcutaneous emphysema has been seen. Abscess and gangrene can seldom be recognized by physical signs. Pneumothorax occurs even in infancy, but is very infrequent except as a result of puncture of the chest. Otitis is

exceedingly common, and one should be constantly on the lookout for it. It is recognized only by examination of the ear with a speculum.

Meningitis may complicate acute bronchopneumonia. It has occurred in about two per cent of our cases. It is in all respects similar to that occurring with lobar pneumonia. Meningeal hemorrhage we have seen only once, and it was the cause of death in a patient eleven months old, who a few days before was seized with convulsions, followed by a gradually increasing stupor, which continued until death. The hemorrhage covered the entire convexity of the brain. Endocarditis is extremely rare; it was not observed in any of our cases. Acute pericarditis is also rare unless there is an extensive pleurisy. When it occurs it is usually with pneumonia of the left side. Complications referable to the digestive tract are quite common. Herpetic stomatitis is frequent, and occasionally the ulcerative variety is seen. Thrush often occurs in the protracted cases among very young infants. Gastro-enteritis is not very common, considering the frequency of vomiting and diarrhea, these depending usually upon functional derangement. Nephritis is rare; it is usually of the acute exudative variety, and very seldom severe enough to affect the prognosis.

Old lesions of tuberculosis—cheesy nodules in the lungs and sometimes in the pleura—are not infrequently met with in patients dying of acute pneumonia of a non-tuberculous character.

Diagnosis.—An acute onset with continuous high fever, rapid respiration, and cough, should always lead one to suspect pneumonia. When to these symptoms are added prostration and a leucocytosis, the diagnosis of pneumonia is almost certain. Cases of the acute congestive type are the ones most frequently unrecognized, and in many of these cases a positive diagnosis is impossible during life. Many atypical cases of pneumonia are seen, particularly in young infants. An unusual temperature course is perhaps the symptom most likely to lead to a mistake. While this, as a rule, is high and remittent, sometimes it is not so, and it may be but little above normal. Rapid respiration is almost always present, but cough may be very slight, especially in infants. In very young infants, the diagnosis often rests upon the prostration, cyanosis, and rapid respiration, the other acute inflammatory symptoms being absent. Only the physical signs of the disease can positively settle the question of diagnosis.

When pneumonia follows bronchitis of the large tubes, whether the bronchitis is primary or complicates one of the infectious diseases, the extension of the disease to the lungs is usually marked by three symptoms—a steadily rising temperature, more frequent respirations, and increasing prostration. It may be twelve or twenty-four hours before the change is indicated by the physical signs.

At the outset, pneumonia can not be positively diagnosticated from severe bronchitis. Such a bronchitis often begins with severe pulmonary symptoms and a temperature of 103° or 104° F.; but this high temperature is of short duration, usually falling after twenty-four or forty-eight hours to 100° or 101° F. The prostration is much less and all the symptoms, possibly excepting the cough, less severe. The only physical signs are coarse râles, which are heard throughout the chest.

The same rules apply to bronchitis of the smaller tubes. The râles are heard both in front and behind, and usually over both sides. If with such râles the temperature continues to rise for three or four days in succession above 103° F., it may be assumed that pneumonia is present, provided there is no other disease which might explain the temperature. If the signs of bronchitis are limited to a single lung, or to one lung posteriorly, the existence of bronchopneumonia may be regarded as certain. Localized bronchitis, then, is always to be interpreted as bronchopneumonia, provided tuberculosis can be excluded.

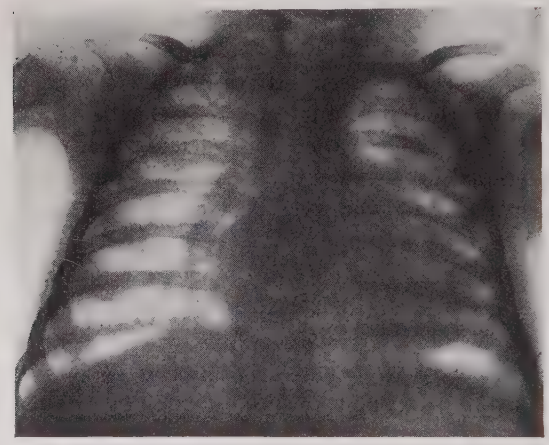


Fig. 63.—Bronchopneumonia. Infant 8 months old; areas of consolidation in both lungs, especially marked at the left apex and the root of the right lung. The only physical signs were scattered râles.

The differential diagnosis of bronchopneumonia from lobar pneumonia will be considered in connection with the latter disease. On account of the remittent temperature, bronchopneumonia may be confounded with malarial fever; or malaria may be suspected as a complication. An examination of the blood will remove the doubt.

Both the acute and the persistent forms of simple bronchopneumonia may be confounded with the tuberculous form; the points of distinction are considered in the chapter on Tuberculosis.

The X-ray is of value in detecting the presence of consolidation before this can be made out by physical signs. (See Fig. 63.) Small scattered areas of bronchopneumonia cannot be differentiated from tuberculosis. Large areas of consolidation do not differ in their appearance from those of lobar pneumonia.

Prognosis.—Bronchopneumonia is always a serious disease, and in an

infant dangerous to life. The prognosis depends upon the age, surroundings, and previous condition of the patient, upon the nature of the infection, whether the disease is primary or secondary, and, if the latter, upon the character of the primary disease. In private practice the mortality from bronchopneumonia is from ten to twenty per cent, depending upon the conditions mentioned. One whose knowledge of bronchopneumonia is derived from observations in private practice can, however, form but little idea of the frequency and severity of this disease in hospitals and asylums for infants and young children, particularly when it occurs with epidemics of measles, diphtheria, or pertussis. The statistics in the following table are taken from the records of two institutions, and fairly represent the results seen in such places in children under three years:

Forms of Pneumonia.	Cases.	Deaths.	Percentage Mortality.
Primary bronchopneumonia	194	96	49.4
Following bronchitis of the large tubes	29	19	65.5
Secondary to measles	89	56	62.9
" " pertussis	66	54	81.8
" " scarlet fever	7	7	100.0
" " diphtheria	47	47	100.0
" " ileocolitis	19	18	94.7
" " epidemic influenza	6	1	16.6
" " varicella	2	2	100.0
" " erysipelas	2	2	100.0
Totals	461	302	65.5

The mortality varies with the age of the patient, being highest during the first year, and diminishing steadily thereafter, as shown by the following table giving the result in 346 cases:

Age.	Cases.	Percentage Mortality.
During the first year	202	66
" " second year	102	55
" " third "	33	33
" " fourth "	6	16
" " fifth "	3	..

In this table are included no cases secondary to measles, scarlet fever, or diphtheria.

Probably the best of all guides to the nature and severity of the infection is the temperature. An excessively high temperature usually indicates a severe type of infection. Some idea of this may be gained

from these figures, giving the highest temperature and the mortality in two hundred and thirty-one cases, not including cases with measles or diphtheria:

Highest Temperature.	Cases.	Deaths.	Percentage Mortality.
106° F. or over	55	47	85.5
105° or 105.5° F.	94	56	60.0
104° or 104.5° F.	53	26	49.0
102° to 103.5° F.	22	13	60.0
99.5° to 101.5° F.	7	5	71.0

The high mortality of the cases with unusually low temperature is due to the fact that they nearly aways were seen in infants with very feeble vitality. The outlook in cases with a steadily high temperature—between 102.5° and 104° F.—is usually more favorable than in those with wide fluctuations, such as 100° to 105.5° F. As a rule, the danger from the disease increases steadily with every degree of temperature above 104.5° F.

An important factor in the prognosis is the previous condition of the patient. The association with rickets is unfavorable, both on account of the feeble muscular power of these children and their thoracic deformities. Marked and persistent tympanites is always an unfavorable symptom. As a rule, second attacks are more serious than the primary ones, especially if the interval between them is short.

In making the prognosis in any given case, the symptoms to be considered are the height and course of the temperature, the presence or absence of nervous symptoms, the condition of the organs of digestion, the presence of cyanosis and the extent of the disease as shown by the physical signs. We have not found the examination of the blood to aid greatly in prognosis. The leucocyte count varies widely and often without apparent reason. Blood cultures, however, are of some assistance. In our hospital cases which gave positive blood cultures, the mortality was 70 per cent, while in those which gave negative cultures it was 44 per cent.

Convulsions occurring early in the disease do not affect the prognosis; but of thirty-seven cases in which convulsions occurred at a late period all but one proved fatal.

So long as the nutrition of the patient can be well maintained, no protracted case is hopeless, no matter how extensive the local disease may be; but the existence of vomiting, diarrhea, or persistent tympanites makes the issue doubtful, even though the other symptoms are favorable.

Treatment.—The most important part of prophylaxis is to give careful and early attention to every attack of bronchitis in an infant, for every such attack should be regarded as a possible precursor of pneumonia. It is striking that one sees bronchopneumonia so seldom in private practice among the better classes, even though bronchitis is very frequent; while among hospital and dispensary patients, where bronchitis is very often neglected, bronchopneumonia is constantly seen. Cases of measles and diphtheria which are complicated by pneumonia should, if possible, be carefully isolated from others, and wards in which they are treated should be thoroughly disinfected before they are used for simple cases.

The hygienic treatment of bronchopneumonia is important, and usually it receives too little attention. It is much the same as that of cases of acute bronchitis already discussed. What was said in that connection regarding the necessity for fresh air and the caution as to very cold air, may be here repeated. The cold-air treatment is not admissible in very young or delicate infants, nor in cases of disseminated pneumonia (capillary bronchitis). The best results from this treatment are seen in the cases with extensive consolidation and with the minimum amount of bronchitis, and it is to be highly recommended in the pneumonia of the severe acute infections—diphtheria, measles, and scarlet fever. The dress and protection of the patient with the cold-air treatment are discussed under Lobar Pneumonia.

Older children with pneumonia should be kept in bed. Infants for a considerable part of the time may be held in the nurse's arms. A frequent change of position in all cases is essential; no child should be allowed to lie for hours directly on the back. The general rules previously laid down for feeding all sick children should be followed here. As a rule, medicine should not be administered in the food.

The same local treatment may be employed as in cases of bronchitis. Counter-irritation, best by means of the mustard paste, may be employed from three to six times daily. It is of the greatest value in the early stage of acute pulmonary congestion, and during attacks of cardiac or respiratory failure. Poultices should not be used.

Alcohol may be needed in pneumonia secondary to diphtheria, measles, or scarlet fever, also in many primary cases. Its use has been greatly abused in this disease. Although there is little doubt that it is at times of much benefit, there is considerable doubt as to its mode of action. The dose is to be regulated by the condition of the patient. Not over one-half ounce daily should be given to an infant of one year.

Of the circulatory stimulants, caffein, camphor, and digitalis may be used, and are recommended in the order named.

For a child of one year the following doses are suitable: Caffein, gr.

$\frac{1}{8}$ to gr. $\frac{1}{2}$ every three hours; camphor is especially valuable for quick effect; ♏ iij to v of a ten per cent solution in oil may be given hypodermically; digitalis, the fluid extract is generally to be preferred as more reliable than the tincture, ♏ $\frac{1}{2}$ may be given every four hours. For immediate effect in sudden heart or respiratory failure, nothing compares with epinephrin given intramuscularly—doses ♏ ij to ♏ v of a 1-1,000 solution; atropin, also used hypodermically, is sometimes useful —dose, gr. $\frac{1}{400}$. Oxygen may be given continuously, but always mixed with atmospheric air. It sometimes seems to benefit greatly cases with marked cyanosis; often it does no good. Gentle friction of the chest wall, without disturbing the patient, is sometimes useful in stimulating the respiratory muscles, especially in protracted cases.

It should be remembered that the normal range of temperature in bronchopneumonia is from 101° to 104.5° F. This temperature is not in itself exhausting, and the chances of recovery are not improved by reducing it so long as it remains within these limits. Too much can not be said in condemnation of the practice of giving the coal-tar products in full doses for the reduction of temperature. In small doses they are often useful to allay nervous irritability, restlessness, and promote sleep.

Antipyretic measures are indicated in cases of hyperpyrexia, which we may define as 105° F. or over, especially when extreme nervous symptoms exist. In these circumstances, the most certain, the most within our control, and hence the safest antipyretic, is cold. It may be used by the evaporation bath, the cold pack, sponging, cold compresses, or an ice-bag applied to the chest. (See chapter on General Therapeutics.)

Not all children bear cold well, and in its use and frequency of repetition one must be guided by its effect upon the child's general condition as well as upon the temperature. When with hyperpyrexia we have general cyanosis, cold surface, feeble pulse, shallow respiration, and stupor, cold is contraindicated and a hot mustard bath should be used.

Inhalations are of more value in relieving cough and in promoting bronchial secretion than any other means we possess. The same substances are to be used, and in the same way as mentioned in the article on Bronchitis.

The nervous symptoms,—restlessness, loss of sleep, etc.,—are often best controlled by cold or tepid sponging; in other cases by small doses of phenacetin—i.e., one grain every three hours to a child of six months. Opium is to be avoided unless there is severe pain, which is very rare; or when the incessant cough is not relieved by inhalations. Codein may be given in doses of gr. $\frac{1}{20}$ every three or four hours to a child of one year, or morphin in half this dose.

Sudden attacks of general collapse with cyanosis are frequent in severe cases of bronchopneumonia. They may come on at any period in the disease. When occurring in the early stage, if promptly and energetically treated, recovery may take place, but when they come on in the late stages they are usually fatal. They may be due to acute congestion or edema of the lung not previously involved, or to circulatory failure. The most efficient treatment is the use of dry cups or the hot mustard bath, the administration of epinephrin and caffein or camphor hypodermically, and to give oxygen continuously.

When the fever continues for five or six weeks, with no disposition on the part of the disease to subside, one should continue the sustaining treatment adopted in the earlier part of the disease—careful feeding and judicious stimulation, but most of all should these patients be given the benefit of the fresh-air treatment. Some apparently hopeless cases recover; but, unfortunately, in the majority the continuance of the pneumonic process is in itself evidence of the weakened vitality of the patient, and, though he may live a long time, usually such attacks prove fatal.

When the fever has disappeared, and there is only a persistence of the physical signs and the general cachexia, the cases are more hopeful. Here, a change of air is more important than all other means of treatment. If in the winter or spring the child can be removed to a warm, dry climate where he can be kept in the open air, or, in the summer, he can be taken to the mountains, immediate improvement is often seen, followed by rapid recovery. With the change of air a general tonic plan of treatment should be followed, cod-liver oil, arsenic, and iron being used, according to the indications in each particular case.

One should never declare one of these cases of protracted pneumonia to be hopeless, nor should he be too ready to assume that tuberculosis is present because the child is wasted and anemic, and the physical signs have persisted.

No specific treatment of pneumonia has yet been proposed which can be recommended for general use.

CHAPTER V

DISEASES OF THE LUNGS.—(Continued)

LOBAR PNEUMONIA

(Fibrinous Pneumonia; Croupous Pneumonia)

Etiology.—*Age.*—Lobar pneumonia may occur at any age. We have seen it in an infant of three months; but it is not until after the first year that it begins to be frequent. After the third year most of the cases of primary pneumonia are of this variety.

Of 500 cases the ages were as follows:

Age.	Cases.	Per cent.
During the first year	76	15
From the second to the sixth year	309	62
" " seventh to the eleventh year	104	21
" " twelfth to the fourteenth year	11	2
Totals	500	100

Season.—In 136 cases the seasonal occurrence was as follows:

Season.	Cases.	Per cent.
In the three winter months	48	35
" " " spring "	62	46
" " " summer "	6	4
" " " autumn "	20	15
Totals	136	100

Lobar pneumonia, in children therefore, as in adults, occurs most frequently during the spring months. March and April show the largest number of cases.

Previous Condition.—In our hospital cases, eighty-two per cent of the children were previously in good condition, and only eighteen per cent were delicate, rachitic, or syphilitic. This observation has been borne out by our experience in private practice, viz., that as a rule lobar pneumonia affects children who were previously healthy. Or to state the matter differently, if a strong child contracts pneumonia it is nearly always of the lobar variety.

Previous Disease.—Previous attacks of pneumonia are observed in but a small proportion of cases. It was noted only five times in 160 cases. In the vast majority of cases lobar pneumonia is a primary disease, although it occasionally occurs as a complication of pertussis, measles, typhoid or scarlet fever, and even diphtheria—chiefly, however, in children over three years old.

Epidemics of lobar pneumonia we have never witnessed, although on several occasions we have seen two children in a family attacked either simultaneously or in rapid succession. Exhaustion, fatigue, and exposure are to be ranked as associated exciting causes.

In addition to other causes, there is required for the production of the disease the presence and growth of the pneumococcus. Associated with it are often found the staphylococcus aureus and occasionally the bacillus of influenza. The bacillus of Friedländer is very seldom the exciting cause of pneumonia in children. It was found but once in blood cultures of 87 cases in the Babies' Hospital.

Lesions.—*The Seat of the Disease.*—In 950 cases in children under fourteen years, this was as follows:

Seat of Disease.	Personal Cases.	Collected Cases.	Totals.
Right lung, upper lobe only	39	137	176
" " middle " "	8	4	12
" " lower " "	26	142	168
" " more than one lobe	13	64	77
Totals, right lung	86	347	433
Left lung, upper lobe only	25	68	93
" " lower " "	49	214	263
" " more than one lobe	9	29	38
Totals, left lung	83	311	394
Both lungs, upper lobes	...	13	13
" " lower "	3	38	41
" " elsewhere	9	60	69
Totals, both lungs	12	111	123

The right lung was thus affected in 45.5 per cent; the left lung in 41.5 per cent; both lungs in 13 per cent. In the order of frequency, the disease involves, first, the left base; second, the right apex; third, the right base; fourth, the left apex. The disease affects, as a rule, a single lobe, and often only a circumscribed portion of a lobe.

The anatomical changes resemble those seen in the adult lung. There is an exudation into the alveoli and smaller bronchi of fibrin, serum, leucocytes, and red blood-cells (Fig. 47). There is usually in addition

an inflammation of the mucous membrane of the larger bronchi and of the pleura. The frequency and severity of the pleurisy is a peculiarity of the lesion in children.

In the first stage, that of *congestion,* the portion of lung involved is dark-colored, heavy, and edematous, and shows under the microscope a serous and cellular exudation into the air vesicles, with swelling of the epithelial cells lining the alveoli.

In the second stage, that of *red hepatization,* there is usually some exudation upon the pulmonary pleura, generally a thin layer of fibrin, giving it a dull look. The lung itself is of a uniform dark-red color. It is solid and cuts like liver. It looks as if it had been inflated to its utmost extent and then injected with a material which had solidified. The consolidated area is sharply defined. Under the microscope the air vesicles are seen to be distended with an exudation which is chiefly fibrin, but with some leucocytes, red blood-cells, and desquamated epithelial cells. The cells are chiefly leucocytes, and are usually more abundant than in the pneumonia of adults.

In the third stage, that of *gray hepatization,* the lung is more moist, and the inflammatory products are partly decolorized. This change takes place irregularly throughout the lung, giving it a mottled appearance.

The fourth stage, that of *resolution,* follows gray hepatization, and consists in the degeneration and liquefaction of the products of inflammation, which are ultimately carried away by the lymphatics in great part, only a small amount being pushed out into the bronchi and removed by coughing.

The duration of the stage of congestion is from a few hours to several days; that of the stage of red hepatization from two days to two or three weeks. This is the condition in which the lung is most often seen at autopsy. The stage of gray hepatization is commonly shorter. Resolution usually begins when the temperature falls to normal, but occasionally it may be delayed for several days. It is generally complete in about a week.

Variations in the Lesions.—(1) Instead of clearing up at the usual time, the lung may remain consolidated for several weeks, and then resolve. (2) The stage of gray hepatization may be followed by a great exudation of pus cells, which may everywhere infiltrate the affected lung; or these may be circumscribed so as to form a single large abscess or many small ones. (3) There may be small areas of gangrene. All these three conditions are rare in young children. (4) There may be excessive pleurisy, or pleuropneumonia. This is found at autopsy in about one-half the cases, and will be separately considered elsewhere.

The lesions in the other organs are for the most part due to the pneumococcus. There may be pericarditis, especially with pneumonia of the left side if complicated by excessive pleurisy. This is seen even in infants. The pericardial inflammation closely resembles that of the pleura. There is a very abundant exudation of fibrin and pus, coating both surfaces of the pericardium. Acute meningitis is rather rare. It is an acute purulent inflammation, with a very abundant exudation of greenish-yellow fibrin and pus, chiefly at the convexity. Less frequently peritonitis is present. Acute parotitis and acute arthritis are seen as rare complications of pneumonia. In most of the complicated cases the other lesions are second to those in the lungs; but they may begin simultaneously with, or even precede, the pneumonia. In severe and rapidly fatal cases with meningeal or peritoneal complications, a general pneumococcus septicemia is usually present.

The heart is generally found in diastole, with the cavities, especially those of the right side, distended with soft clots. There may be found ante-mortem thrombi, which may extend into the pulmonary artery or the aorta.

Symptoms.—(1) *The Typical Course.*—A child three or four years of age, after a few hours of slight indisposition, is suddenly taken with vomiting, followed by a rapid rise in temperature. He is dull and heavy, complains of headache and general weakness, refuses food, and is easily persuaded to remain in bed. He has the appearance of being quite ill, even after a few hours. Occasionally sharp pain in the side is complained of. The skin is dry; there are marked thirst, restlessness, and the other symptoms which accompany fever. The temperature is found to be 104° F., or even higher; the respirations 40 to 50 a minute; the pulse full, strong, and 120 to 130. On the second day the patient is no better. The temperature remains high; the tongue is coated; the anorexia continues; the pain is more severe; cough is present and may be quite frequent.

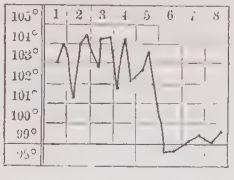


Fig. 64.—Typical Temperature Curve of Lobar Pneumonia. *History.*—Male, three years old; in fair condition; sudden onset; signs of consolidation—bronchial respiration and voice, and dulness—over left lower lobe behind, not distinct until the morning of the fifth day. On the seventh day the lung was resolving.

After the second or third day the patient is usually more comfortable, and sleeps better, but may be disturbed by the cough. At times there is restlessness, and at night there may even be slight delirium. The respiration continues rapid and the temperature high. These general symptoms show very little change until the sixth or seventh day, when, after a long sleep, which has been more natural than before, the patient wakes, decidedly improved as to all his symptoms.

There is less fever, and the temperature continues to fall rapidly until it touches the normal line, or it may even go below this. As the fever subsides the pulse drops to 90 or 100, and the respirations to 25 or 30 a minute. The appetite soon returns, and convalescence is usually rapid. In a week the patient is out of bed, and in a week or two more he is out of doors. This is the course seen in fully two-thirds of all the cases of lobar pneumonia at this age.

(2) *Pneumonia of Short Duration.*—Instead of running the usual course of from five to eight days, cases are seen in which the duration is only three or four days, although the physical signs indicate that the process in the lung passes through the usual stages. These differ from the ordinary type chiefly in their duration. They are always mild.

(3) *Abortive Pneumonia.*—This form of the disease is rarely seen in hospitals, but it is not infrequent in private practice where the physician is summoned at the earliest signs of illness. The onset is precisely like that of ordinary pneumonia, and may even be as severe as the average case. The physical examination of the chest gives all the signs of the first stage of the disease, but on the second or third day the physician is greatly surprised to find that the temperature has fallen to normal, and that all the physical signs have disappeared. The process in such cases does not seem to go beyond the first stage of congestion; there is no evidence of hepatization of the lung. The course is often such as to lead the physician to the opinion that he has made a mistake in his diagnosis. This type of pneumonia corresponds with abortive types of other infectious diseases so frequently met with in children. The temperature curve in such a case is shown in Fig. 67. The diagnosis of these cases is always attended with some uncertainty. There can be no doubt that many of the unexplained high temperatures of brief duration which are seen in children are from this cause. Exactly why it is that the disease sometimes terminates in this way can not always be explained. It may be because the resistance of the patient is greater than usual, or the virulence of the pneumococcus is less.

(4) *The Prolonged Course.*—Although usually lasting about a week, it is not rare for pneumonia to continue ten, twelve, or even fifteen days. This prolonged course is usually due to the fact that the disease spreads from one part of the lung to another, or even to the opposite lung, involving in succession two, three, or more lobes. This is sometimes known as "creeping" pneumonia; it is always severe and the outlook is generally unfavorable. A prolonged temperature with physical signs limited to a single lobe should always suggest complications, most frequently empyema, occasionally pericarditis.

(5) *Hyperacute Pneumonia.*—Pneumonia may very rarely be fatal in the first forty-eight hours. The onset is sudden, frequently with con-

vulsions. The prostration is extreme and in a few hours the child may be pulseless. Delirium or deep coma is the rule. There may be no cough and no symptoms or physical signs pointing to a pulmonary lesion. The respiration may be slow and very deep like the breathing in the air hunger of acidosis. The system seems overwhelmed by the intensity of the toxemia. Unless one has seen autopsies upon patients with this form of pneumonia it seems impossible to believe that the course could differ so from the type of disease usually observed. The diagnosis can only be suspected unless consolidation of the lung can be made out. This type of pneumonia is not found in infancy. In a few such cases a complicating acidosis has been shown to be present by laboratory tests.

(6) *Cerebral Pneumonia.*—This term was first applied by Rilliet and Barthez to cases of pneumonia in which the cerebral symptoms predominate. They will be considered later.

Onset.—Prodromal symptoms of more than a few hours' duration are quite rare. The onset of lobar pneumonia is almost invariably abrupt, with well-marked symptoms—vomiting, diarrhea, chill, or convulsions. Vomiting is altogether the most frequently seen. In summer particularly, there may be vomiting and diarrhea. A distinct chill is rare in a child under five years of age, and is not very common even in older children. Convulsions are not very infrequent, being seen in about five per cent of the cases. Their occurrence depends upon the suddenness of the invasion and the susceptibility of the patient.

Cough.—This is present in most of the cases throughout the disease, but often is not marked for the first day or two. It is seldom a distressing symptom. A disposition to suppress the cough on account of pain is very frequently noticed.

Expectoration.—This is rarely seen in early childhood, and practically never under five years of age. Children of ten or twelve may have the same expectoration as adults—white and viscid, or brownish-red early in the disease, yellow and abundant toward its close. This shows the presence of the pneumococcus in great numbers.

Pain.—Headache and general muscular pains in the back and extremities are frequent during the invasion. The characteristic pain, however, is pleuritic. It is not necessarily felt in the region of the affected lung, and often not in the chest at all. It is frequently referred to the loin, the epigastrium, or to any region to which the intercostal nerves are distributed. Pain in the right iliac fossa associated with extreme tenderness and some rigidity may lead to the suspicion of appendicitis when in reality the pain is referred from the inflamed pleura.

Prostration.—This is one of the characteristic features of pneumonia. The patient is generally willing to go to bed on the first day of the

attack, and shows little desire to leave it while the disease continues. Ambulatory cases are not common in children.

Respiration.—This is always accelerated, and generally out of proportion to the pulse. The normal ratio of the respiration to the pulse is one to four; in pneumonia, frequently one to two. The respiration is not labored and not quite panting, although this term is sometimes used to describe it. It is jerky. There is a short inspiration, then a momentary pause, followed by a quick expiration, which is accompanied by a short moan. This expiratory moan is very characteristic. The rapidity of respiration is usually in proportion to the amount of lung involved, but it is also modified by the temperature, as the respirations often drop from 60 to 30 in the course of a few hours at the crisis.

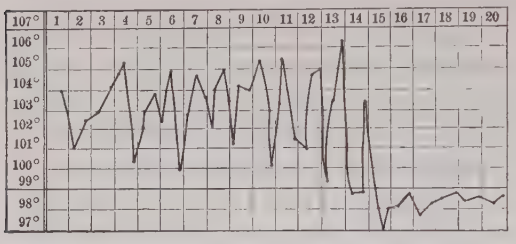


FIG. 65.—LOBAR PNEUMONIA WITH REMITTENT TEMPERATURE. *History.*—Female, eighteen months old; in fair condition; sudden onset; repeated examinations of chest made, but no abnormal signs until the ninth day, when there were very rude respiration and slight dulness at the right apex, in front; on the twelfth day all the signs of consolidation at the same point, no râles; four days after the crisis the lungs were clear.

Pulse.—In the early part of the disease this is frequent, full, and strong, from 120 to 150 a minute. Later it may be weak, small, compressible, and sometimes irregular. It is much more rapid in the child than in the adult, 160 and 180 being often seen in cases not especially severe. The pulse rate is of less importance than its character.

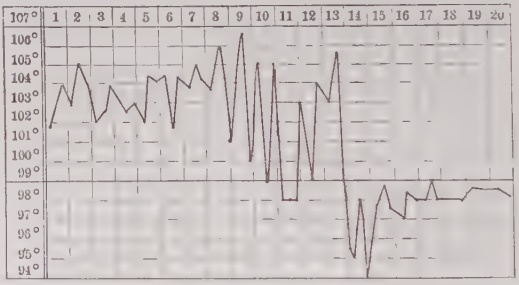


FIG. 66.—LOBAR PNEUMONIA WITH SUBNORMAL TEMPERATURE AFTER THE CRISIS. *History.*—Female, nineteen months old; fairly healthy; sudden onset; symptoms typical but physical signs delayed; consolidation in left mammary region on the eighth day; on the ninth in right lung middle lobe; on the eleventh day a pseudocritical drop followed after twenty-four hours of apyrexia by a further rise, which was accompanied by signs of extension of the disease in the right lung. Resolution rapid after crisis.

Temperature.—The typical temperature curve of lobar pneumonia (Fig. 64) is characterized by an abrupt rise usually to 104° or 105° F., and by daily fluctuations generally within the limits of two or three degrees until the crisis, at which time the temperature falls to normal,

usually in the course of twenty-four hours. After this time it does not go above the normal line. Such a curve is seen in the majority of cases over three years of age.

In children under three years of age it is not uncommon for the temperature to be of a more or less remittent type (Fig. 65).

These wide fluctuations often lead to great difficulty in diagnosis, particularly if the physical signs appear late, as they not infrequently do. It is probable that most of them are to be explained as mixed infections.

The chart shown in Fig. 66 illustrates three features which are often seen in pneumonia: (1) A temperature which early in the disease is steadily high and as the day of crisis approaches becomes remittent; (2) a secondary rise after being normal for twenty-four hours, which was due in this instance to an extension of the disease to a new part of the lung; (3) a fall to a point considerably below normal at the time of the crisis. In this case the temperature fell in the course of eighteen hours from 105° to 95° F., and later still lower; it was two days before it finally remained at the normal point. A fall to 96.5° or 97° F. at the time of crisis is not uncommon.

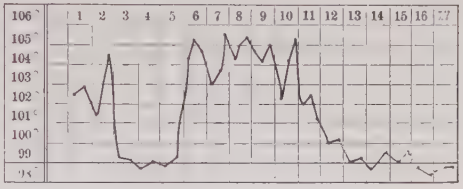


FIG. 67.—ABORTIVE PNEUMONIA IN LEFT LUNG, FOLLOWED BY TYPICAL PNEUMONIA IN RIGHT LUNG, TERMINATING BY LYSIS. *History.*—Male, seventeen months old, healthy; sudden onset; on the second day disseminated fine râles in both lungs behind, and over left lower lobe very feeble respiration, high-pitched—i. e., some bronchitis, with congestion (?) of left base. On the third, fourth, and fifth days, general symptoms gone and signs nearly disappeared. On the sixth day all symptoms of pneumonia, and on the seventh distinct consolidation of right base, rest of chest clear. Subsequent course typical, resolution rapid and complete.

In the foregoing cases the fever terminated by crisis. In Fig. 67 is shown one ending by lysis. This is a mode of termination much more frequent in young children than in those who are older. Thus, in 93 of our own cases, nearly all of which were in children under three years of age, the fever ended by crisis in 49, and by lysis in 44; while in 552 collected cases, the majority of which were in older children, 396 ended by crisis, and 126 by lysis.

The table on the following page shows the day of crisis in 567 cases of lobar pneumonia in children who recovered. From this it will be seen that the most frequent critical day is the seventh, and that in sixty-six per cent of the cases it was from the fifth to the eighth day. The causes of a post-critical rise in the temperature are chiefly two—extension of the disease to a new area, or the development of pleurisy, which is

apt to be purulent. Less frequently it is due to otitis, meningitis, pericarditis, or gastro-enteritis. In fatal cases the temperature is generally high until the end. In general, it may be said that the temperature is considerably higher in children than in adults; in the majority of cases it reaches 105° F., the usual range being from 102° to 105° F. In 15 of 137 cases, or eleven per cent, it reached 106° F. or over.

The Day of Crisis

Second day	3 cases.		Eleventh day	18 cases.	
Third "	22 "		Twelfth "	7 "	
Fourth "	43 "		Thirteenth "	8 "	
Fifth "	88 "		Fourteenth "	7 "	
Sixth "	83 "		Fifteenth "	1 case.	
Seventh "	132 "		Eighteenth "	3 cases.	
Eighth "	73 "		Twenty-first "	1 case.	
Ninth "	55 "		Twenty-sixth "	1 "	
Tenth "	22 "				

Gastro-enteric Symptoms.—These are more common in infants than in older children. At the onset there is frequently vomiting, sometimes also diarrhea. A continuance of the vomiting is rare, and is generally due to improper feeding or medication. It may be a very serious complication. Diarrhea is also rare, except at the onset and in summer cases. Great tympanites is a distressing symptom, and when present, it is a bad prognostic sign. Throughout the disease there are anorexia, coated tongue, and the usual symptoms of high fever.

Nervous Symptoms.—Cerebral symptoms are frequent and very often misleading. Pneumonia is often ushered in by convulsions, which may be repeated two or three times in the course of the first twenty-four hours. They are sometimes followed by drowsiness or stupor, sometimes by active delirium. Cerebral symptoms may predominate for several days. There may be opisthotonus, dilated or contracted pupils, irregular pulse, retracted abdomen, and, in fact, almost every symptom of meningitis. Lumbar puncture in these cases usually shows an excess of cerebrospinal fluid under high tension and it may contain a few pneumococci. Occasionally the decubitus *en chien de fusil*, or gun-hammer position, is assumed. These are often described as cases of *cerebral pneumonia*, and in many of them pneumonia is not suspected until the fourth or fifth day of the disease, sometimes not until the crisis occurs, when the rapid disappearance of all these nervous symptoms indicates their origin. Early convulsions are not generally followed by an especially severe type of the disease, only one of seven such cases proving fatal. On the other hand, cases with late convulsions are usually fatal, as they indicate either a very severe form of the disease or the development of a serious complication, usually meningitis.

Delirium is much more frequent than convulsions, and is seen in

nearly one-fourth of the cases. Generally it is slight and noticed only at night or when the temperature is very high. It is most pronounced at the height of the disease. Other nervous symptoms belonging to the typhoid state are occasionally seen, but only in the most severe forms of the disease.

It is impossible to establish any relation between the seat of the disease in the lungs and the occurrence of cerebral symptoms. They are more frequent in children under five years than in those who are older, and depend upon the suddenness of the invasion, the intensity of the infection, and the susceptibility of the child. Late in the disease they may indicate exhaustion, toxemia, or complicating meningitis. The usual nervous symptoms—restlessness, headache, sleeplessness, etc.—are nearly always proportionate to the height of the temperature.

Urine.—Throughout the febrile period of the disease the urine is scanty, high-colored, with a high specific gravity, usually loaded with urates and with marked diminution of the chlorids. A moderate acetone reaction is very common. In a small proportion of cases a trace of albumin may be found, and occasionally a few hyaline casts. Evidences of serious renal disease are seldom found in lobar pneumonia in early life.

Skin.—The face, in pneumonia, is usually flushed, sometimes on both sides and sometimes only on one; in other cases it is pale, but not indicative of pain. Cyanosis is rare except toward the close of the disease and is usually a sign of respiratory failure. Herpes of the lips or face is quite frequent.

Blood.—A marked polymorphonuclear leucocytosis is a characteristic feature of lobar pneumonia; the exceptions are in very mild cases or very severe infections with little or no reaction. The increase begins shortly after the onset and continues during the stage of exudation, generally reaching its maximum shortly before the crisis, when it declines rapidly. The usual number of white cells in an average case of pneumonia in a young child is from 25,000 to 40,000, but it is not rare for the count to run up to 50,000 or even 60,000. We have seen it over 100,000 several times. The absence of leucocytosis in a strong child who is acutely ill is always strong presumptive evidence against pneumonia. A well-marked leucocytosis is of much value in differentiating pneumonia from typhoid fever. Positive blood cultures were obtained in the Babies' Hospital in 14 per cent. of 108 cases studied. Otten found almost exactly the same proportion in a study of 70 cases. These observations indicate that positive cultures are much less frequent than in the pneumonia of adults.

Physical Signs.—The earliest signs in pneumonia are due to the

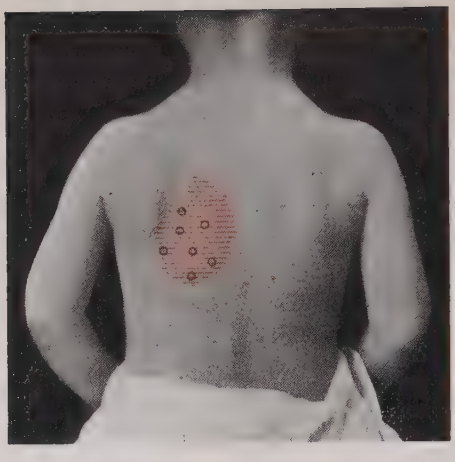


FIG. 68.—FIRST STAGE. Congestion of left lower lobe, with crepitant râles. Feeble breathing of a rude character, with slight dulness.

FIG. 69.—In the center of the area, a small spot of pure bronchial breathing and voice; surrounding this an occasional crepitant râle, with bronchovesicular breathing and slight dulness.

FIG. 70.—SECOND STAGE. Complete consolidation of left lower lobe. Pure bronchial breathing and bronchial voice; marked dulness; increased vocal fremitus, and at the lower part a few friction sounds.

NOTE.—During resolution the signs take the inverse order: those of Fig. 70 give place to those of Fig. 69, and these in turn to those of Fig. 68. In addition, many coarse râles may be heard.

acute congestion of the affected lung or lobe, in consequence of which less air enters this portion and more air the rest of the lungs. Percussion gives diminished resonance or slight dulness, often of a somewhat tympanitic character over the affected area, and exaggerated resonance over the remainder of this lung and over the opposite lung. Auscultation over the affected lobe gives feeble respiratory murmur, rather high in pitch; sometimes there may be so nearly an absence of all breath-sounds as to suggest fluid. The normal respiratory murmur over the healthy portions of the lungs is intensified. In children this exaggerated breathing is not infrequently mistaken for bronchial breathing,

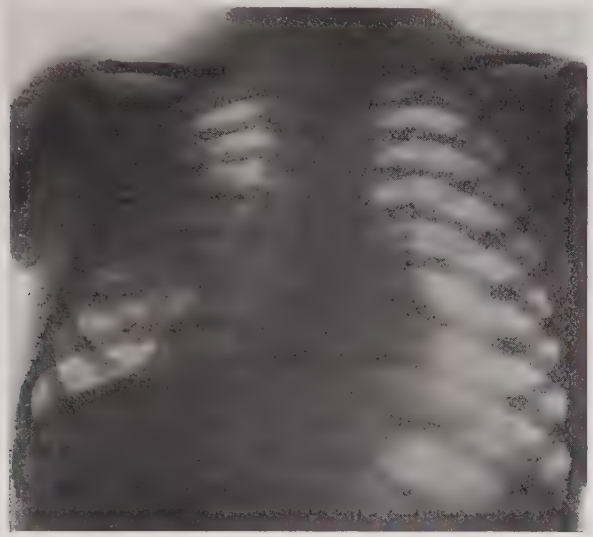


FIG. 71.—LOBAR PNEUMONIA. Child $2\frac{1}{2}$ years old. Lobar pneumonia of right upper and middle lobes, at the height of the disease with all the usual signs of consolidation.

and the physician may be led into the error of locating the pneumonia upon the wrong side. Exaggerated breathing differs little from normal breathing except in intensity. Bronchial breathing is higher in pitch, tubular in character, and is heard with nearly equal intensity both on expiration and inspiration. If the chest is frequently auscultated, crepitant or fine subcrepitant râles may usually be heard at some period at the end of full inspiration, but often they are present but for a few hours, and they may be missed altogether. (Figs. 68, 69, 70.)

A study of cases of lobar pneumonia by the X-ray shows that consolidation occurs early, and that it first affects the surface of the lung, gradually extending inward as the disease progresses (Fig. 71). Bronchial breathing is not usually obtained until the consolidation has reached the hilus of the lung. Feeble breathing and slight dulness occur earlier.

In the second stage, that of consolidation, no air enters the air vesicles of the affected portion of the lung. There is found here exaggerated vocal fremitus, and marked dulness, but very rarely flatness. Over the rest of this lung there is exaggerated, sometimes even tympanitic, resonance; this is especially frequent at the apex of the lung in front, when there is consolidation at the base behind. Under these conditions cracked-pot resonance may sometimes be obtained. Over the healthy lung there is exaggerated resonance. Over the consolidated portion there is bronchial breathing and bronchial voice, the area over which they are heard being sharply defined. Râles are usually absent, but there may be pleuritic friction sounds.

In the stage of resolution there is a gradual disappearance of the signs of consolidation. The pure bronchial is replaced by bronchovesicular breathing, the vesicular element gradually predominating. Moist râles of all varieties are heard. Usually the most persistent signs are slight dulness or diminished resonance, with a respiratory murmur which is feebler than normal and a little higher in pitch; sometimes there are also dry friction sounds. These signs may persist for two or three weeks.

Exceptional Physical Signs.—While in the majority of cases the signs of consolidation are distinct on or before the fourth day, in not a few they may be delayed much longer. Of eighty-two cases in which the day was noted on which consolidation was found, it was not until the fifth day or later in one-fourth the number. In six of them, although carefully and repeatedly examined, no consolidation was found until the seventh day or later and in one case not until the twelfth day. These cases of delayed or concealed physical signs have often been regarded as examples of central pneumonia. That pneumonia may exist only in the center of a lung for a number of days is extremely improbable. At autopsy we have very frequently seen superficial pneumonia but never central lobar pneumonia. X-ray studies have shown conclusively that with a superficial consolidation no bronchial breathing may be heard even though the consolidation may be fairly extensive. When the process extends toward and reaches the hilus of the lung bronchial breathing is readily heard. It is the superficial pneumonia, then, that escapes detection rather than the central. There are, however, two regions in which pneumonia may exist and yet not be accessible by our means of physical examination, viz., at the apex of the lung in the part covered by the shoulder, and along the posterior border of the lung where it lies against the vertebrae. It is quite common in cases with late physical signs that the first distinctive evidences of disease are found high in the axilla, or beneath the clavicle in front, and these regions should be closely watched in all doubtful cases.

Complications.—The occurrence of dry pleurisy over the consolidated portion of the lung is so constant that it can hardly be considered a complication. A slight serous exudation of two or three ounces is very common and often develops rapidly. In the most severe cases of pleurisy there is an excessive exudation of fibrin and pus. This has occurred in about eight per cent of our cases. This variety is known clinically as pleuropneumonia, and will be considered separately. Pericarditis is uncommon. It is seen more often in infants than in older children. It most frequently develops at the height of the pneumonia rather oftener when this affects the left lung than the right; it occurs in pleuropneumonia much more often than in the simple form. The pericarditis is usually of the fibrinopurulent type. It may sometimes be discovered by physical signs; but rarely gives rise to any new symptoms. Endocarditis is extremely rare, though now and then it occurs upon valves previously the seat of a chronic lesion. Meningitis is rare, and generally develops late in the disease. It is nearly always ushered in by repeated attacks of vomiting or convulsions. Its course is short and progressive. Peritonitis causes few new symptoms except abdominal distention, pain, and tenderness. Parotitis and arthritis are very rare and are easily recognized.

Course and Termination.—In the great majority of cases lobar pneumonia terminates either in perfect recovery or in death. When ending in recovery, resolution commonly begins immediately upon the cessation of the fever, and is complete in about a week. Delayed resolution is not common in children; chronic pneumonia and tuberculosis are rare sequelae, but empyema is very frequent. Its symptoms sometimes develop immediately after the pneumonia, the temperature continuing high; or there may be an interval of a few days before the development of the pleural symptoms. Some pleuritic adhesions probably remain in every case in which there has been much dry pleurisy, and when severe and extensive, these may be the cause of subsequent symptoms, like any other dry pleurisy.

Death from uncomplicated pneumonia may be due to exhaustion, or to circulatory failure, with or without failure of the respiration. The signs of circulatory failure sometimes develop quite rapidly in cases which are apparently doing well. The symptoms are: coldness of the hands and feet, then of the legs and arms; a rapid, compressible, and sometimes irregular pulse; muscular weakness and pallor, but usually no cyanosis. The symptoms of respiratory failure are: very rapid superficial respirations, sometimes 100 a minute; blueness of the lips and finger nails; often a leaden hue of the whole body; there are loud tracheal râles, and recession of all the soft parts of the chest on inspiration.

Death may occur early in the disease, when the pneumonia has spread rapidly, involving both lungs. In most of the uncomplicated fatal cases, death results from failure of the circulation at about the end of the first week. In the complicated cases death usually occurs in the second week; but we have known fatal meningitis to develop as late as the end of the fourth week.

Diagnosis.—The most characteristic clinical and pathological differences between broncho- and lobar pneumonia are shown in the following table:

BRONCHOPNEUMONIA	LOBAR PNEUMONIA
1. Often secondary.	1. Almost always primary.
2. Under two, chiefly under one year.	2. Most common between three and eight years.
3. Occurs more frequently in delicate and debilitated children.	3. More often in those previously healthy.
4. Bacteria—in primary cases, usually the pneumococcus; in secondary cases, usually mixed infection.	4. The pneumococcus, very often alone.
5. Products of inflammation chiefly cellular; process often diffuse.	5. Chiefly fibrin; process circumscribed.
6. Onset often gradual, sometimes insidious, especially when secondary.	6. Onset sudden, with well-marked symptoms.
7. No typical course; fever often lasts three or four weeks; rarely terminates by crisis.	7. Typical course; crisis, usually from fifth to eighth day.
8. Involves both lungs as a rule, most frequently lower lobes posteriorly.	8. Usually one lobe or a part of a lobe; left base most frequently, right apex next.
9. Signs of bronchitis mingled with those of consolidation; râles in other parts of the same lung, or in the opposite lung, throughout the disease.	9. Râles only early, and during resolution; frequently no signs in opposite lung.
10. Consolidation later—fourth to seventh day: there may be none; apt to be incomplete; shades off gradually.	10. Consolidation earlier; second or third day. Consolidation complete; area usually sharply defined.
11. Resolution slow, one week to two months; often incomplete; strong tendency to become chronic.	11. Resolution rapid, usually complete within a week.
12. Relapses and second attacks frequent.	12. Both are rare.
13. Sequelae: Empyema, chronic interstitial pneumonia, sometimes tuberculosis.	13. No sequelae except empyema.
14. Prognosis always serious from the age and the circumstances in which disease occurs.	14. Prognosis good; rarely fatal except from complications — empyema, meningitis, pericarditis.
15. Hospital mortality 50 per cent of primary cases, 65 per cent of all cases.	15. Mortality about 4 per cent of all cases.

In the majority of cases the symptoms are plain and the physical signs so typical that it is difficult to overlook pneumonia if any degree of care is used in the examination of the patient. The difficulties in diagnosis are due to the great variation in the general symptoms, and to the late appearance of the physical signs. The error usually made is to mistake pneumonia for some other disease, rather than to mistake some other disease for pneumonia. On account of its frequency in children, pneumonia should always be excluded before accepting any other explanation of a continuously high temperature. The rule should be followed, in all cases of acute illness, of making a thorough examination of the chest daily until the diagnosis is clear. If, to high temperature, rapid respiration and marked leucocytosis are added, one should always suspect pneumonia, no matter what the other symptoms may be. It not infrequently happens that the general symptoms are quite characteristic and yet the physical signs appear late. In such cases pneumonia should always be looked for high in the axilla or just beneath the clavicle, since it is particularly in the cases of apex pneumonia that this obscurity is likely to exist.

In their onset, scarlet fever, tonsillitis, and gastro-enteritis may all resemble pneumonia. Scarlet fever is recognized by the sore throat and the characteristic eruption on the second day; tonsillitis, by the local symptoms. In infancy, pneumonia often begins with vomiting and sometimes there is also diarrhea, which may lead one to mistake the disease for gastro-enteritis. The constitutional symptoms of influenza often closely resemble those of pneumonia; the diagnosis is frequently in doubt for several days until definite physical signs of pneumonia make their appearance. From all other general diseases, pneumonia is to be differentiated by the physical signs.

Pneumonia with marked cerebral symptoms sometimes resembles cerebrospinal meningitis. In both we may have the abrupt onset, convulsions, delirium or stupor, opisthotonus, prostration, and marked leucocytosis. The only positive means of differential diagnosis are by the physical signs in pneumonia, and the findings from lumbar puncture in cerebrospinal meningitis.

The question sometimes arises in pneumonia with cerebral symptoms, whether or not pneumococcus meningitis also exists. If the nervous symptoms are present from the beginning, there is probably no meningitis. If they develop suddenly during the course or toward the close of the disease, meningitis should be suspected. The only positive means of differentiation is by lumbar puncture.

Lobar pneumonia is to be differentiated from a pleuritic effusion. The most common mistake is to confound empyema with unresolved pneumonia. In pneumonia rarely if ever do the signs point to involve-

ment of an entire lung. There is increased vocal fremitus, dulness, bronchial voice and breathing, and occasional râles or friction sounds. In empyema the whole lung is often affected, there is displacement of the heart, flatness on percussion, diminished or absent vocal fremitus, and although bronchial voice and breathing are present, they are usually distant and feeble. There are no râles or friction sounds. In doubtful cases an exploratory puncture should always be made. Serous effusions give the same physical signs as empyema.

The X-ray may be of marked assistance in diagnosis. The shadow of consolidation in lobar pneumonia is usually clear and sharply circumscribed. It is often wedge shaped as shown in Fig. 71.

Prognosis.—There is probably no disease in which the patient appears so ill, and yet so often recovers completely, as lobar pneumonia in children over three years old. Of 1,295 collected cases, chiefly from hospital practice, there were but 39 deaths, a mortality of three per cent. In 187 cases of our own there were 21 deaths, a mortality of eleven per cent. In only one of the fatal cases was the child over two years old. The difference between the mortality among our cases and the general mortality given, is due to the fact that a large proportion of the first group were observed in children under two years, while of the collected cases, the vast majority were in older children. Combining the above figures, we have a total of 1,482 cases with 60 deaths, a mortality of four per cent. In nearly all our cases death was due either to complications or to very extensive disease, as when both lungs were involved, or nearly the whole of one lung. In only one case was an uncomplicated pneumonia of a single lobe fatal.

The prognosis depends upon the age of the patient, the intensity of the infection, as shown by the temperature, nervous symptoms and pulse, the presence or absence of complications, and the extent of the local disease. These factors are to be taken into consideration rather than any special symptoms. Early convulsions do not materially affect the prognosis. Late convulsions are always very unfavorable.

The occurrence of vomiting, diarrhea, or marked tympanites late in the disease is always unfavorable.

A temperature range between 102° and 105° F. is the rule, and within these limits the fever does not affect the prognosis. Even very high temperature does not increase the danger from the disease as much as might be expected. Of fifteen cases in which the temperature reached 106° F. or over, all but three recovered; while of six cases in which it was 106.5° or over, only one died. The highest recorded temperature in our cases—107.5° F.—was in a patient who recovered. A transient rise, even though the temperature may go very high, is seldom serious. Much more serious is a fever which remains steadily above 105° F., as

in most cases this accompanies either very extensive disease or pleuropneumonia. The continuance of the fever after the tenth day is a bad symptom; for, although the crisis may be postponed until the twelfth day and occur normally, such a prolonged temperature is an indication of a new focus of disease or the development of complications. In a severe attack, the extension of the disease to another lobe after the fifth day is unfavorable. If resolution does not begin soon after the temperature becomes normal, the development of empyema, or some other pulmonary complication, should be apprehended.

The results of blood cultures have some prognostic value. Of 108 hospital cases the mortality of 15 with positive cultures was 33 per cent; of 93 with negative cultures it was but 8 per cent.

Treatment.—The specific treatment of lobar pneumonia has not yet reached a point where it is to be advised with children. In considering the management of this disease several cardinal facts are to be kept in mind. It is a self-limited disease, having a strong tendency to recovery in the great majority of cases regardless of the treatment adopted. The fatal cases are almost always in children under two years of age; the rare deaths in older ones are usually due to complications. There is as yet no treatment which can be relied upon to abort an attack of pneumonia or shorten its course. It follows, therefore, that the indications are, so far as possible, to make the patient comfortable during his illness, to watch for complications, and to treat the individual symptoms as they arise.

In the majority of cases, hygienic treatment is all that is required. The patient should be kept in bed, no matter how mild the attack; he should be disturbed as little as possible. Most children with pneumonia get too much treatment. There seems to be a decided advantage not only in fresh air, but in cold air. Patients in cold rooms sleep better, cough less, and altogether seem more comfortable than when carefully housed to prevent their "taking cold." Wide-open windows are desirable even though the room temperature is constantly as low as 50° F. The patient should be properly protected by blankets, flannel wrapper, woolen stockings, and at times a hot-water bag at his feet. Food should be given at regular intervals, usually not oftener than every four hours. It should not be forced when the patient is suffering only from thirst, especially early in the attack, when the appetite is often completely lost. Water should be allowed freely at all times.

These measures, careful nursing, an occasional dose of codein (gr. $\frac{1}{10}$ to a child of three years) when the patient is very restless, fretful, or sleepless, an ice-cap to the head, and cold sponging when the temperature makes him uncomfortable, are usually all that is necessary, except to keep a sharp lookout for complications.

Special symptoms may require treatment. When not severe, the nervous symptoms may be controlled by codein alone or in combination with small doses of phenacetin or the bromids. Sometimes sponging with tepid water is better than drugs. Severe nervous symptoms, such as delirium, stupor, great restlessness with impending convulsions, when associated with high temperature, call for ice to the head, cold sponging, or the cold pack or bath. Pain, if moderate, may be relieved by counter-irritation, by a mustard paste, by dry cups, an ice-bag, or by a hot poultice; if severe, codein may be used in addition. The cough is rarely severe enough to require treatment. When it is so severe as to prevent sleep, small doses of Dover's powder or codein should be given. Antipyretic measures are not necessarily called for even if the temperature is very high. Some nervous children are less disturbed by the temperature than by the means used to reduce it. Under such conditions the temperature should be closely watched, but not necessarily interfered with unless other symptoms develop. The nervous symptoms are a better guide than the thermometer to the use of antipyretics. Cold we believe to be the safest and most certain antipyretic we possess. It may be used as a cold sponge bath, the cold pack or an ice-bag to the chest. There is no objection to the bath except the prejudice of the laity. While cold is applied to the trunk the extremities should be closely watched, and heat applied if necessary. The duration of the pack or bath, and the frequency of their use, will depend upon the individual case. In the majority of cases stimulants are not required. They are called for when the pulse is weak, compressible, and rapid, when the face is pale and the extremities are cold. The same stimulants are to be employed, and in the same way, as in bronchopneumonia. Circulatory and respiratory stimulants are usually required in larger quantity at the time of and just after the crisis; they are to be used as in bronchopneumonia.

PLEUROPNEUMONIA

Under this term are included cases of pneumonia with an excessive amount of pleurisy, the two processes uniting to produce a single clinical type of disease.

In nearly all cases of lobar pneumonia there is a certain amount of inflammation of the pulmonary pleura, and also in those cases of bronchopneumonia which are accompanied by any marked degree of consolidation. In both of these conditions the pleurisy is usually co-extensive with the consolidation. But in certain cases, in both forms of pneumonia, the amount of pleurisy is excessive, and this so modifies the symptoms and course of the disease as to require for them a separate consideration.

In some it appears that the inflammatory process begins almost simultaneously in the lung and in the pleura; while in others the pleurisy follows the pneumonia. These cases are almost invariably due to the pneumococcus, although in some there is a mixed infection.

In 398 hospital cases of pneumonia there were 27, or 6.8 per cent, which could be classed as pleuropneumonia, the diagnosis being confirmed either by autopsy or operation. Of 190 fatal cases, 12.5 per cent were cases of pleuropneumonia. Most of these hospital patients were under three years of age, and the disease is more frequent at this period than in older children.

Lesions.—Of these 27 cases, 17 were classed as bronchopneumonia and 10 as lobar pneumonia. The left lung was more frequently affected than the right in the proportion of three to two. In most of the cases the pleura covering the entire lung was involved, even though the pneumonia affected but a single lobe, or only a part of a lobe. In nearly half the cases both lungs were involved, but one to a very much less extent than the other. In a small number of cases the pleurisy was limited to the posterior surface of the lung.

In pleuropneumonia both the visceral and the parietal pleura are coated with a layer of yellowish-green fibrin, in thick, shaggy masses, causing adhesions of the lung to the chest wall, the diaphragm, and the pericardium (Plate IX). The exudation varies between one-eighth and one-half of an inch in thickness. It can often be stripped from the lung or scraped from the chest wall by the handful. In its meshes small pockets may form, which contain only a few drops, or sometimes a dram, of pus, or less frequently, serum. This is the condition in which the lung is usually found when death has occurred at the height of the disease. If the process has lasted longer, larger collections of pus may be present. The lung itself shows the usual changes of pneumonia, and if there has been any considerable accumulation of fluid, there are in addition the evidences of compression. The disproportion between the changes in the pleura and those in the lung may be striking. Frequently the pulmonary lesions are relatively insignificant.

With pleuropneumonia of the left side, the pericardium is frequently involved. The lesions closely resemble those of the pleura. Meningitis and peritonitis are by no means rare, and in most of the fatal cases a general pneumococcus septicemia is present. The organisms may be found in the blood in great numbers during life or post mortem.

An inflammation of the intensity described is very often fatal in the acute stage, if the patient is a child under two years old. Occasionally at this age, and very frequently in older children, we see the later stages of the process. The most frequent course is for more and more pus to be poured out from the inflamed pleura until the chest is filled, the case

becoming thus one of empyema. Sometimes the fluid is serous instead of purulent, but this is very rare in infancy. In other circumstances the exudation is partly absorbed, but the greater part becomes organized so as to form a thick jacket of fibrous tissue which binds the lobe or lung to the chest wall and interferes seriously with its subsequent full expansion. Chronic interstitial pneumonia may follow.

Symptoms.—There is little which distinguishes a case of pleuropneumonia except the severity of all the constitutional symptoms; the temperature is often higher, the prostration greater, and the patient in every way impresses one as being more seriously ill than with ordinary pneumonia. Sometimes the thoracic pain is more severe and more constant than is usual in pneumonia.

In the early stage pleuritic friction sounds are unusually prominent; after two or three days the signs of consolidation come out clearly in most cases, but still accompanied by loud friction sounds. After the fibrinous exudation is very abundant, the signs are often obscure and confusing, and there may be at no time well-defined signs of consolidation. There is usually a mingling of the signs of consolidation with those of effusion. There is marked dulness, and sometimes flatness. The vocal fremitus is apt to be diminished, and it may be absent. Bronchial voice and breathing are heard, but they are not distinct as in consolidation; they are, however, feeble and distant, as over fluid. There are usually coarse, moist râles but these may be absent. The signs may be found over one entire lung, or they may be limited to the posterior region, and even to a single lobe. They resemble those present over fluid, with one exception—viz., the heart is not displaced. If an exploratory puncture is made, nothing is found; occasionally the exploring needle happens to strike one of the small pockets of pus in the meshes of the fibrin, and a few drops of pus are withdrawn. If an incision is made under the supposition that the case is one of empyema, no more pus may be found, the surgeon coming upon the fibrinous masses as soon as the chest is opened. There is scarcely any condition in the chest giving signs more puzzling than those just enumerated. They are, however, easily explained by the pathological condition.

Prognosis.—The prognosis in pleuropneumonia is much worse than in simple pneumonia. In infants the outlook is very bad, the majority of the cases being fatal during the acute stage. Very young children may be overwhelmed with the extent and the intensity of the inflammation, and die in four or five days. In children over two years old the most frequent result is for the case to go on to empyema, which with proper treatment usually terminates in recovery. Where there is organization of the fibrin with the production of extensive adhesions, the ultimate result often is not so favorable as when empyema develops. Convalescence is

ACUTE PLEUROPNEUMONIA

The lungs have been separated in front and spread out to show the whole external surface as seen from behind. The left lung, with the exception of a narrow strip along its anterior border, is completely covered with a thick, ragged exudation of fibrin. The left lower lobe was hepatized; the right lower lobe deeply congested. From a child one year old.

usually slow, and the patients are liable to exacerbations of pleurisy; they may suffer for years from the partial crippling of one lung.

Treatment.—Cases of pleuropneumonia are to be managed like the ordinary cases of pneumonia of the severe type. In some, the excessive pain may call for more active counter-irritation and a freer use of opium than in other forms of pneumonia, and the greater prostration may require that stimulants be given earlier and in larger quantities.

HYPOSTATIC PNEUMONIA

This can not often be recognized clinically, but it is very frequently seen upon the post-mortem table. It represents an inflammatory process of a low grade and is seen to some degree in almost every case where an infant has died of chronic disease. It is particularly frequent in those who have died of marasmus. It invariably occupies a strip along the posterior border of both lungs, and usually of both the upper and lower lobes. This is from one to two inches wide, of a uniform dark red color, and is sharply outlined. The pleura is not involved, and the remainder of the lung may be normal, congested, or slightly emphysematous. On section, it is seen that the pneumonic area is quite superficial, rarely involving the lung to a greater depth than half an inch. Under the microscope there is found a distention of the small blood-vessels in the affected area, and the air vesicles are filled with many red blood-cells, epithelial cells, and a few leucocytes. Between the areas of consolidation are groups of air vesicles which are normal, congested, or collapsed. It is a lobular rather than a bronchopneumonia. The lesions in this form of pneumonia are probably the result of venous stasis, owing to the child's recumbent position.

At autopsy the condition may be confounded with atelectasis. Little significance is to be attached to the finding of hypostatic pneumonia at autopsy, and it alone should never be regarded as a sufficient cause of death, although it is perhaps the only lesion present. During life it may give rise to fine moist râles, which are heard along the spine, usually upon both sides; but there is seldom either dulness or bronchial breathing. The treatment is that of the primary disease.

CHRONIC BRONCHOPNEUMONIA—CHRONIC INTERSTITIAL PNEUMONIA—BRONCHIECTASIS

Chronic bronchopneumonia is an inflammation of the connective-tissue framework of the lung, involving the stroma, the alveolar septa, the walls of the bronchi, and the pleura. It is usually accompanied by

cylindrical dilatation of the bronchi—bronchiectasis. Chronic pneumonia may occur in the well nourished and apparently robust but is more common in the delicate. While seen at all ages its beginning is usually before the fifth year.

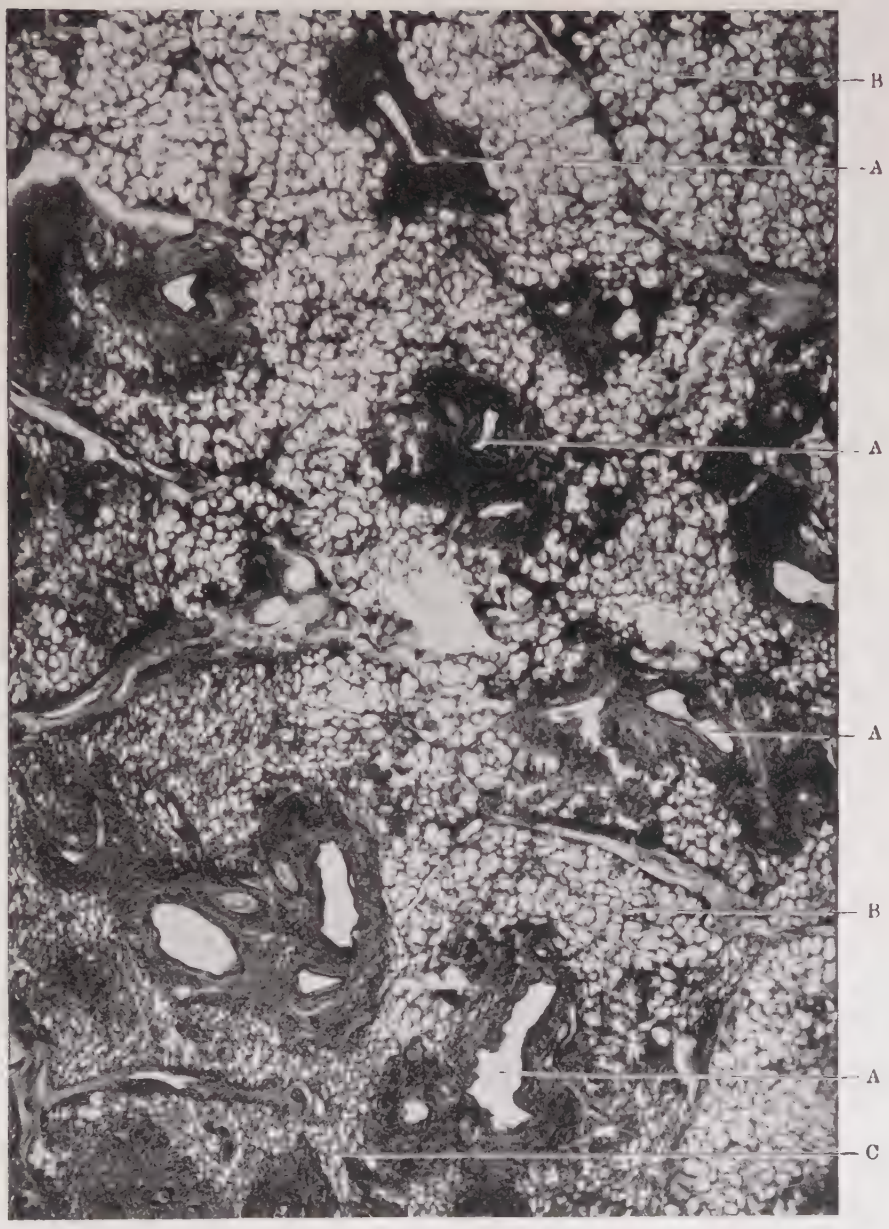
Etiology.—In children, as in adults, this process is most frequently associated with pulmonary tuberculosis; but in early life it is not an infrequent condition apart from tuberculosis. The non-tuberculous cases, as a rule, are preceded by an attack of acute bronchopneumonia, sometimes by several such attacks, separated by longer or shorter intervals. Foreign bodies may cause localized interstitial pneumonia of great severity. The organisms associated with chronic pneumonia may be the pneumococcus or the staphylococcus, but more frequently we believe it is the influenza bacillus either alone or in combination. It is hard to say why in one case complete resolution takes place in a diseased lung and in another there follows a chronic progressive lesion. It is probably dependent upon a balance between the individual resistance and the severity of the infecting organism.

Lesions.—The part of the lung affected may be an entire lobe, but usually it is a portion of one lobe, or there are areas in more than one lobe. There are dense connective-tissue adhesions binding the diseased part to the chest wall, to the diaphragm and to the pericardium, often so firmly that the lung is torn on removal. The affected lung is smaller than in health; it is hard, tough, and fibrous. Surrounding the fibrous portions are emphysematous areas. On section, the process is seen to be somewhat irregularly distributed through the lung, the lesion being usually most marked in the vicinity of the smaller bronchi, and sometimes seen only there, the intervening lung being nearly normal (Plate X). In some portions, where the process is most advanced, almost all trace of lung tissue may have disappeared, the part resembling a solid fibrous tumor, through which run the bronchial tubes, usually much dilated. In places this dilatation may be sufficient to form cavities of considerable size. The bronchial glands are often enlarged to the size of a hazelnut, and they may be tuberculous.

Upon examination with the microscope, the pleura is found greatly thickened, with bands of new fibrous tissue passing from it into the lung. The walls of the small bronchi are in most places thicker than normal, but elsewhere they have undergone cylindrical dilatation, and are filled with pus. The walls of the alveoli show a marked proliferation of the connective-tissue elements, and the alveoli are filled with organized inflammatory products, so that they are nearly or quite obliterated. The stroma is much increased in amount throughout the affected lung.

Symptoms.—In most cases there is a history of repeated attacks of acute bronchopneumonia, from which the child made a slow convales-

PLATE X



CHRONIC BRONCHOPNEUMONIA

In the greater part of the specimen the disease is limited to the vicinity of the small bronchi, A A A, each of which is surrounded by a zone of new connective tissue, the result of the inflammatory process, the intervening lung tissue, B B, being normal. In the lower left-hand portion, the disease is more diffuse: the air vesicles, C, between the areas of new connective tissue are greatly compressed, and in some places entirely obliterated.

(After Delafield.)

cence, remaining pale, anemic, and sometimes wasted for several months. Improvement then took place in the general symptoms, the appetite and strength returned, and in many cases the lost weight was nearly or quite regained. However, neither the pulmonary symptoms nor the physical signs entirely disappeared. There remained a dry, hard cough, which at times was severe. Pains in the chest were occasionally complained of, and perhaps shortness of breath on exertion was noticed.

Examination shows a persistence of the dulness on percussion, with a rude or bronchovesicular respiratory murmur of very feeble intensity. Little change may take place in these signs for months; then an acute attack of bronchitis or bronchopneumonia may occur. If the latter, the same lung is affected, and a fresh consolidation is added to the previous disease. This attack may not be very severe, but it drags on for several weeks, with slight fever and little or no change in the physical signs. Partial resolution may then take place, but the lung is left much more seriously crippled than before. Often there is a history of several such attacks, each one leaving the lung a little worse than it found it.

The characteristic physical signs of chronic bronchopneumonia are not usually present until the process has continued for many months. They may be found over part of a lobe, or over an entire lobe, or even the greater part of one lung. On inspection, there may be seen, in a well-marked case, retraction of the chest, which is especially noticeable when the disease is situated at the apex of the lung. The vocal fremitus is usually increased, but it may not be abnormal. There is marked dulness, often flatness, over the affected area, with exaggerated resonance over the rest of the lung. The area of flatness shades off gradually. The most striking thing on auscultation is the very feeble respiratory murmur; in many cases the lung is almost silent. More rarely there is marked bronchial voice and breathing. Râles and friction sounds are usually absent except during an acute exacerbation of the symptoms, when they may be heard as in any attack of bronchopneumonia. In recent cases there is no displacement of the heart; in those of long standing it may be drawn far to the affected side by contraction of the adhesions. There may be clubbing of the fingers in cases of long standing.

When these lesions are once present complete recovery is impossible, and there is always a tendency for them to increase rapidly or slowly, according to the child's vigor of constitution, his surroundings, and the frequency with which exacerbations occur. If the process is extensive the patient often succumbs to some intercurrent disease or to an acute attack of pneumonia; if limited in area, the process may be arrested and the patient recover, always, however, to be more or less embarrassed because of the crippling of a part of one lung. Not a small number of these children ultimately die of tuberculosis, and in such cases it is al-

ways a difficult matter to decide whether tuberculosis was present from the beginning, or whether it was due to subsequent infection.

The cases in which bronchiectasis is the most important condition are not common. The only characteristic additional symptom is a copious mucopurulent expectoration, which is usually very fetid. It may amount to several ounces a day, and is expelled after paroxysms of coughing, which usually occur in the morning. This may continue for months, or even years, and yet these patients are generally without fever, seldom lose weight, and may have the appearance of being in very good health. It is rare that the physical signs of a cavity are present.

Prognosis.—This depends on the extent of the disease, the patient's age and constitution, and on our ability to prevent by treatment, climatic and otherwise, the occurrence of acute exacerbations. Under the most favorable conditions, a few patients may recover completely so far as symptoms are concerned; but the majority remain at best delicate during childhood, or even throughout life.

Diagnosis.—The most important thing is to distinguish between the simple and the tuberculous cases, and this, by symptoms and physical signs, is in the majority impossible. If the family history is good, if the patient lives in the country, if his symptoms begin with a well-defined acute attack of pneumonia, if the seat of disease is the base posteriorly, and if the examination of the sputum is negative for tubercle bacilli the process is probably simple. If the family history is doubtful or is positively tuberculous, if the patient lives in the city, and especially if he is an inmate of an institution or if his home is in the tenements, if the initial symptoms are indefinite, if the disease is situated anteriorly, the process is probably tuberculous. The cutaneous tuberculin test aids much in diagnosis. With a negative reaction tuberculosis can be excluded almost with certainty; but a positive reaction does not prove that the pulmonary process is tuberculous, although it is strongly suggestive. The discovery of tubercle bacilli in the sputum is, of course, conclusive.

Foreign bodies in the lung may give symptoms of chronic bronchopneumonia; metallic and many solid substances may be detected by the X-ray.

Treatment.—Nothing has any essential influence upon the disease except change of climate. This should be the same as for tuberculous cases. The treatment of the patient has for its object the maintenance of the general nutrition at its highest point, by careful feeding, judicious exercise, and by most of the measures enumerated in the chapter on Malnutrition. Cod-liver oil may often be given with advantage especially during the winter. The cough may be treated as in cases of chronic bronchitis.

Cases of bronchiectasis may obtain considerable relief from inhalations of creosote. Operation is not to be recommended.

ABSCESS OF THE LUNG

Multiple small abscesses are not uncommon as a termination of acute bronchopneumonia, in which connection they have already been considered. Larger non-tuberculous abscesses of the lung are rare, very obscure in their symptoms, and apt to be mistaken for localized empyema, sometimes for interstitial pneumonia with bronchiectasis. Four such cases have come under our observation. One was discovered at autopsy, the other three were recognized during life and successfully treated by operation. Other examples in young children have been reported by Huber and by Hedges. The cause of these single abscesses is usually a previous attack of acute primary pneumonia, less frequently an inflammation excited by a foreign body in the lung.

An abscess due to a foreign body is usually accompanied by wasting, and a widely fluctuating temperature of a hectic type—symptoms suggestive of a rapidly advancing tuberculous process. If the abscess follows an ordinary pneumonia the course is generally less intense. The constitutional symptoms differ little from those of empyema. There is an irregular type of fever, sometimes quite high, but more often only from 99° to 101° or 102° F., a moderate cough, not much wasting, and generally not very marked prostration. A leucocytosis of 30,000 to 50,000 is usually present. The physical signs are somewhat confusing and are a combination of those present in effusion and consolidation. There is an area of flatness shading off into dulness. The vocal fremitus may be increased or it may be diminished. The respiratory murmur is very feeble or absent over the abscess, often it is bronchovesicular in character. Friction sounds and râles are usually present. The heart is slightly or not at all displaced. If an exploratory needle is introduced, pus may not be found even by repeated punctures; or it may be obtained at one time and not at another, although introduced in the same intercostal space, the difference in result being due to the direction in which the needle is passed into the lung. When pus is found, the diagnosis of a localized empyema is generally regarded as established, and it is not until the chest is opened that the mistake is discovered. The operator then comes upon the lung, which may or may not be adherent. If the abscess follows an acute pneumonia the pus may show a pure culture of the pneumococcus. If it is due to a foreign body, there is invariably a mixed infection, and the pus is apt to be fetid.

When not treated surgically, abscess of the lung may rupture into

the pleural cavity, producing a secondary empyema, or spontaneous evacuation may take place through a bronchus and recovery follow. When the cause is a foreign body, rapid recovery often follows its expulsion by coughing. If the diagnosis is made and proper surgical treatment is instituted, recovery occurs in probably the majority of cases.

The general plan of treatment should be the same as in empyema. In a small proportion of cases aspiration may suffice for a cure. However, incision is usually necessary. If the pleura is not adherent, adhesions should be excited by packing the thoracic wound with gauze, and after a few days a second operation may be done. The lung should be opened with a blunt instrument, following the line of the exploring needle, and a drainage-tube inserted as in empyema, the subsequent treatment being the same as for that disease.

GANGRENE OF THE LUNG

Pulmonary gangrene is rare in children, although probably more common than in adults. It is most frequently associated with pneumonia. It is usually circumscribed, and seldom diagnosticated during life.

Etiology.—All but one of our cases have been in children under three years old, the youngest an infant of four months. Gangrene occurs for the most part in children who are ill-conditioned, feeble, or cachectic, and often follows one of the infectious diseases, particularly measles. Of twelve cases which have come under our personal observation, eight complicated acute bronchopneumonia. Pulmonary gangrene has been present in about three per cent of our autopsies upon cases of pneumonia. The immediate cause of the necrotic process is interference with the circulation in a part of the lung, which is usually due to thrombosis or embolism of some of the branches of the pulmonary artery. To this there is added the entrance of putrefactive bacteria. In some cases pulmonary gangrene may begin as a septic thrombosis, this infection originating in some process in a distant part of the body.

Lesions.—The lower lobes are more frequently affected than the upper, and the surface of the lung rather than the central portions.

Two forms of gangrene may be seen: the diffuse form, which affects a whole lobe, or even a whole lung; and the circumscribed form, which occurs in a number of small scattered areas. The latter is the variety usually seen in children. In the diffuse form the lung is of a dirty-green or brown color, moist, and emits a gangrenous odor. In the circumscribed form, when occurring in pneumonia, the parts affected are of a gray or green color, usually wedge-shaped, with the base at the

surface of the lung. In the early stage they are not softened, and have no gangrenous odor; later, both these conditions may be present, and masses of necrotic lung tissue may be found in a cavity with ragged walls, partly filled with fetid pus. Careful dissection will reveal, in many cases, the presence of thrombi in the vessels leading to the gangrenous parts.

Symptoms.—There are but two distinctive symptoms of pulmonary gangrene: the fetid odor of the breath, and the expectoration of masses of necrotic lung tissue. In the cases associated with acute pneumonia, which include the majority of those seen, death nearly always takes place before there is any separation of the sloughs, and even before very active decomposition in the necrotic areas has occurred. Both the peculiar symptoms are therefore wanting, and the diagnosis is made only at the autopsy. This has been true of nearly all the cases which have come under our observation. But these patients, with two exceptions, were infants. In older children, particularly in cases secondary to the entrance of a foreign body, the characteristic symptoms are more frequently seen, and there may be a third symptom—hemorrhage. This is present in about one-fourth of the cases (Rilliet and Barthez), and may be fatal. The general symptoms associated with gangrene are those of profound asthenia, resembling the typhoid condition.

From what has been said, it will be evident that the diagnosis is very difficult. If the characteristic odor of the breath is present, conditions in the mouth from which it might arise must be excluded. Cavity formation in tuberculosis may also be a cause of a very foul breath. The cutaneous tuberculin test will aid greatly in the diagnosis. The physical signs differ in no respect from those of ordinary cases of pneumonia. The termination is almost always in death. This is due not only to the condition itself, but to the circumstances in which it is seen.

Treatment.—The general treatment should be supporting and stimulating, as in all severe cases of pneumonia. For the local process but little can be done, except the inhalation of antiseptics, of which creosote and turpentine are undoubtedly the best.

ACQUIRED ATELECTASIS—PULMONARY COLLAPSE

These terms are applied to a state of the lung resembling the fetal condition, but occurring in a lung which has once been expanded. It may be due to compression or to obstruction.

Collapse from Compression.—The principal cause of this form is pleuritic effusion. It may also be produced by pneumothorax, enlargement of the heart, pericardial effusion, deformities of the chest from

rickets or Pott's disease, and tumors of the mediastinum or the thoracic wall. In these conditions, on account of the external pressure, the air vesicles are not filled, although the bronchi are pervious. After collapse has existed for a considerable time, changes may take place in the lung which render expansion difficult or impossible. Unless, however, there are pleuritic adhesions, expansion often takes place readily after many weeks or even months. The symptoms and signs are those of the original disease.

Treatment is available chiefly in that form which follows pleuritic effusion, and will be considered in the chapter on Empyema.

Collapse from Obstruction.—This is due to two factors: blocking of either the large or small bronchial tubes, and feeble inspiratory force. The importance of collapse from obstruction in the acute diseases of the lung in infancy has undoubtedly been exaggerated. Whenever a large or small bronchus is completely obstructed by a foreign body, the portion of the lung to which the bronchus is distributed gradually becomes collapsed. If it is one of the primary bronchi which is occluded, a whole lung may be collapsed; if one of the lobar divisions, an entire lobe; if one of the smaller divisions, only a small area. The collapse does not take place immediately, but the contents of the air vesicles are gradually absorbed. The collapsed portion is slightly depressed below the surface of the lung. It is of a dark-red color, very vascular, and to the naked eye resembles a pneumonic area, which it may subsequently become.

Many writers explain the development of bronchopneumonia from bronchitis of the smaller tubes, through the intervention of pulmonary collapse, assuming that the obstruction of the small bronchi, from swelling of their walls and the accumulation of secretion, produces the same result as the plugging of a bronchus by a foreign body. In our own autopsies we have found little support for this theory. In acute bronchitis of the smaller tubes the lumen is narrowed, but seldom enough to prevent the entrance of air. The result is usually emphysema, not atelectasis. Such, at least, has been the condition we have most frequently found in autopsies in the earliest stage of bronchopneumonia following bronchitis of the fine tubes. There are very often groups of collapsed air vesicles surrounding pneumonic areas, but these are neither an essential nor a very important part of the lesion. Collapse of a large part of the lung, or even of a lobe, we have never seen, either in pertussis or in acute bronchitis.

There is seen in delicate or rachitic infants a form of collapse which comes on very gradually. It is accompanied by bronchitis affecting the tubes in the dependent part of the lung. It may resemble the congenital form of atelectasis. Under the microscope there is almost

invariably found, accompanying the collapse, lobular pneumonia and bronchitis of the tubes in the affected regions.

The symptoms of acquired atelectasis are much the same as in the persistent congenital form. The respiration is rapid, and there may be inspiratory dyspnea with deep recession of the chest walls, especially if there is rickets. There is also at times cyanosis of variable intensity. The temperature is not elevated, but frequently is subnormal. The physical signs are very uncertain. There is usually feeble respiratory murmur over the affected areas, occasionally accompanied by moist râles. The essential point of difference between these cases and those of congenital atelectasis is that in the former the patients are often strong at birth, crying and breathing well, giving no signs of anything wrong in the lungs until the general nutrition has suffered from some other cause.

The following is a fairly typical case: A female infant thirteen months old had been under observation for several months before death. During this period she suffered a great part of the time from mild bronchitis. The chest was extremely rachitic. The respiration was always accelerated, and on inspiration the lateral recession of the chest was at times extreme. There was occasionally seen slight cyanosis, and during the last few weeks it was constant. Death occurred quite suddenly. At autopsy there was found very marked vesicular emphysema of both lungs in front. Nearly the whole of both lower lobes were in a condition of collapse, and of a uniform grayish-purple color. The posterior portion of the upper lobes was similarly affected, but to a less degree. With moderate force all of the collapsed areas could be completely inflated. Bronchitis was present, but the pleura was normal.

The treatment of these cases is the same as that outlined in the chapter upon Congenital Atelectasis.

EMPHYSEMA

Pulmonary emphysema consists primarily in overdistention of the air vesicles. It may result in their rupture and the escape of air into the interlobular connective tissue of the lung. In infancy and childhood emphysema is usually associated with acute processes.

Etiology.—Cases of emphysema are divided into two groups which are due to quite different causes. In one group it is compensatory, and consists in overdistention of the air vesicles in certain parts of the lungs because the full expansion of other parts is prevented either because they are consolidated, as in pneumonia or tuberculosis, bound down by adhesions from old pleurisy, or subjected to external pressure, as from chest deformities due to Pott's disease or rickets. In these

conditions it is probable that the emphysema is produced during inspiration. It may also be produced by the artificial inflation of the lungs of the newly born.

In the second group of cases emphysema is produced by obstructive expiratory dyspnea or cough. It is seen in all forms of laryngeal stenosis, in acute bronchitis and bronchopneumonia, in asthma, pertussis, and occasionally it is produced by any condition which requires deep inspiration and holding the breath. In bronchitis the obstruction may be caused by a swelling of the mucous membrane or by an accumulation of secretion. In this group of cases air enters the lung, but as it can not readily escape, the air vesicles are distended, sometimes to such a degree that their resiliency is almost entirely lost.

Lesions.—The most common form in early life is acute vesicular emphysema, which occurs when the force distending the air cells is only moderate. In this form there is dilatation of the vesicles with very slight structural changes, there being usually rupture of a few alveolar septa only (Fig. 49). Although the dilatation may be quite marked, the emphysema is not permanent. The parts most affected are the upper lobes, particularly the anterior borders. In appearance the emphysematous lung is pale, sometimes almost white. The affected areas are prominent, and do not collapse upon opening the chest. With a lens, or even with the naked eye, the individual air vesicles can often be distinguished as minute pearly bodies, at times resembling miliary tubercles. When the disease is secondary to acute bronchitis or laryngeal stenosis it may affect nearly the whole of both lungs.

With a greater distending force rupture of many of the air vesicles results, and this may give rise to interstitial or interlobular emphysema. At times blebs are formed, varying in size from a pin's head to a cherry or even larger ones. These are usually seen at the anterior border or at the root of the lung on its inner surface. Again, the air finds its way between the lobules, dissecting them apart in all directions throughout the lung. Sometimes a large part of the surface of both lungs is seamed with irregular deep crevasses containing air, the largest being an inch or more in length and nearly one-fourth of an inch wide. The most severe cases occur in pertussis. On two or three occasions we have seen this form of emphysema, once to an extreme degree, when children had died from diseases unconnected with the respiratory tract, and when no history could be obtained which threw any light upon the etiology of the emphysema.

Localized emphysema not infrequently occurs in the subcutaneous tissue of the thoracic wall following exploratory puncture of the chest. This is seldom extensive and the air usually disappears in a few days by absorption without causing any symptoms. Sometimes from a rupture

of an emphysematous vesicle at the hilus of the lung there occurs emphysema of the mediastinum which may spread to the tissues of the neck and ultimately to almost the entire body. The patient gives the impression of having been artificially inflated (Fig. 72). Such widespread emphysema is usually associated with conditions which prove fatal, the emphysema adding much to the patient's discomfort but not increasing the danger of the original disease.

Fig. 72.—General Subcutaneous Emphysema, Child 14 Months Old. Following perforation of a caseous nodule at the root of the lung; pulmonary tuberculosis.

Symptoms.—Emphysema occurring in acute pulmonary diseases gives rise to no peculiar symptoms and to no physical signs except exaggerated resonance upon percussion. This masks dulness from consolidation and also that from the liver and spleen. If the patients recover from the original disease, the emphysema greatly diminishes or disappears completely in the course of a few weeks or months. Acute interlobular emphysema can not be diagnosticated during life, unless, as is sometimes the case, general subcutaneous emphysema is seen, which may come on quickly, last for several hours or days and then gradually disappear.

The treatment of emphysema is that of the disease with which it is associated.

CHAPTER VI

PLEURISY

All the common forms of inflammation of the pleura are seen in childhood. In the great majority of cases they are secondary to disease of the lung itself. Serous effusions are much less frequent than in adults, and under three years large ones are rare. Purulent effusion (empyema)

is, however, much more often seen than in adult life, and it is the most important variety of pleurisy with which the physician has to deal.

Whether inflammation of the pleura ever occurs as a strictly primary disease is still a mooted point. Cases are occasionally observed clinically in which both the serous and purulent forms of the disease appear to be primary, but these are extremely rare. Acute pleurisy may, however, follow inflammation of the lung so rapidly that it is not easy to determine that the lung was first affected. In infants, extension from the lung is almost the sole cause. It occurs both with lobar and bronchopneumonia, existing to some degree in nearly every case in which there is consolidation of the lung. Next in frequency to simple pneumonia as a cause of pleurisy are the tuberculous processes of the lung. Tuberculous pleurisy without tuberculosis of the lungs or the bronchial glands is of doubtful occurrence. Acute pleurisy is an occasional complication of the infectious diseases, particularly scarlet and typhoid fevers, measles, and influenza. In most of these cases also it is secondary to disease of the lung. Pleurisy in older children occasionally follows cold and exposure, although it is doubtful whether in any case this is the only cause. In them also it may occur as a complication of rheumatism.

The most important cause of acute pleurisy being extension from pneumonia, it follows that it is most frequent in the cold season, that it occurs more often in males than in females, and between the ages of one and five years. It may, however, be seen at all ages, and may even occur in intra-uterine life. The youngest case in which we have found extensive pleuritic adhesions as an evidence of previous inflammation was in an infant of three months. In this case firm connective tissue adhesions were found over the whole of both lungs.

DRY PLEURISY

In infants and young children this usually accompanies pneumonia or tuberculous processes in the lung. In older children it may be primary.

Lesions.—On account of the frequency with which this occurs in pneumonia we have an opportunity of observing it in all stages. In the mildest varieties it affects only the pulmonary pleura, and occurs over the pneumonic areas. The pleura is injected, has lost its luster, and appears dull or roughened. This is due to an exudation of fibrin upon its surface. If the process continues, more fibrin is poured out, and there are in addition swelling and a proliferation of the connective-tissue cells, and an exudation of leucocytes from the blood-vessels. The pleura is then

coated with a layer of fibrin of variable thickness, in which are entangled pus cells and new connective-tissue cells. The layer of fibrin varies from the thickness of tissue paper to that of an ordinary book cover. In recent cases it may easily be stripped off, while in older ones it becomes organized and is firmly adherent. The color of the exudate varies with the number of pus cells. It is gray, grayish-yellow, or yellowish-green, according as these cells are few or numerous. As a rule, dry pleurisy is localized, but the two opposing surfaces are affected. Part of the exudate is usually absorbed, but it is doubtful if complete recovery occurs, there being left behind some adhesions between the visceral and parietal layers.

In the dry form of tuberculous pleurisy there may be only an exudation of fibrin, or the pleura may be covered with gray tubercles and yellow tuberculous nodules. These are not only seen upon the surface of the pleura, but develop in the exudation. In this form, which is usually chronic, great thickening of the pleura may take place. Both the serous and purulent effusions occurring in conjunction with tuberculosis are likely to be sacculated because of the previous existence of adhesions.

After nearly every case of dry pleurisy there probably remains some slight thickening of the pleura. In certain cases there follows a chronic inflammation of the pleura with the production of new connective tissue, which results in thickening and adhesions which may be so extensive as to entirely obliterate the pleural cavity. Either one or both sides may be affected. It is usually accompanied by external pericarditis. This form is rare in childhood.

Symptoms.—As an independent clinical disease, acute dry pleurisy has no existence in infancy or early childhood. The cases which are occasionally so diagnosticated have in our experience invariably proved to be bronchopneumonia. In older children dry pleurisy may occur under the same conditions as in adults.

The symptoms are sharp, localized pain, increased by full inspiration, sometimes tenderness upon pressure, and a short, teasing cough. The pain is not always felt upon the affected side, and it may be referred to the abdomen. Upon physical examination, dry pleurisy is recognized by the presence of a pleuritic friction sound. This is usually of a dry rubbing character, generally localized, and heard both on inspiration and expiration. It is quite superficial, and not changed by coughing. This form of pleurisy, as a rule, runs a course of a few days or a week without constitutional symptoms. When dry pleurisy occurs as a complication of pneumonia it is recognized by the signs just mentioned; but it usually causes no new symptoms except pain.

Treatment.—The treatment consists in counter-irritation by mus-

tard or iodin, according to the severity of the inflammation, and in the use of opium. Severe pain can sometimes be relieved by firmly encircling the chest with a broad band of adhesive plaster.

PLEURISY WITH SEROUS EFFUSION

This form of pleurisy is not common in young children, and in infants except with acute pneumonia it is rare. In those somewhat older it is usually tuberculous in origin in which case it frequently acts like a primary disease. It occurs as a complication of pneumonia and may be seen in nephritis, acute rheumatism, scarlet fever, or any of the other acute infectious diseases. Bacteria are occasionally present in the exudation, even in cases which do not become purulent, but their number is usually small. The tubercle bacillus, the streptococcus and the pneumococcus are the forms most often seen.

Lesions.—The early changes are much the same as in dry pleurisy, but in addition serum is poured out from the blood-vessels, in some cases almost from the beginning of the inflammation. This may be small in amount, or it may fill the pleural cavity. The lesions are similar to those seen in adults, except that in children there is apt to be more fibrin. The process usually terminates in absorption of the serum, but, as in dry pleurisy, more or less extensive adhesions are left behind from the fibrinous exudation. In other cases there is at first a clear serum, often containing pneumococci, then it becomes somewhat turbid, and finally purulent. This is especially common in infants.

Symptoms.—The very small serous effusions which occur so frequently as a complication of pneumonia rarely cause new symptoms or a change in the physical signs. In the present connection only those cases will be discussed in which the amount of effusion is considerable. This form of pleurisy sometimes follows a well-defined attack of pneumonia. Other cases come on with acute febrile symptoms somewhat resembling those of pneumonia, but with all the symptoms less severe, except the pain. After an illness of only two or three days the chest may be found full of fluid. In a third group the disease comes on insidiously, with little or no fever, and often with no distinct pulmonary symptoms except shortness of breath. There is general weakness, sometimes loss of flesh, anemia, and moderate prostration; but usually the patients are not sick enough to go to bed. The symptoms of pleurisy with effusion vary greatly. When it occurs as a complication of some acute infectious disease, it is often latent, and the diagnosis is to be made only by the physical examination of the chest.

In cases in which the fluid does not become purulent, the usual course

of the disease is for the fluid to disappear gradually by absorption, the case going on to spontaneous recovery. Serious symptoms resulting from pressure upon the heart and lungs are not common, but may occur when the fluid accumulates rapidly; hence they are most likely to be seen early in the attack. There may be great dyspnea, sometimes orthopnea, cyanosis, weak pulse, and even attacks of syncope. Death may occur with these symptoms. In certain cases there is seen no tendency to spontaneous absorption, and the exudation may remain stationary for months. There may then be fever, usually slight but sometimes quite regular, with a decline in the general health, pallor and anemia, which may strongly suggest the existence of pus, although this is not present. Others are regarded as cases of tuberculosis.

Physical Signs.—The signs in the chest are essentially the same whether the fluid is serous or purulent. On inspection, there is diminished movement of the affected side, sometimes bulging of the intercostal spaces, and if the effusion is large, an increase in the measurement of the affected side of the chest. The apex beat of the heart will usually be considerably displaced if the effusion is upon the left side. It may be found at the epigastrium, at the right border of the sternum, or even in the right mammary line. In disease of the right side the displacement is less, and occurs only with a large effusion. It may then be found in or near the left axillary line. On palpation, the vocal fremitus is usually diminished or absent, but it may be but little changed. Percussion gives marked dulness or flatness. In a large effusion this is over the entire lung. There is also a sensation of increased resistance appreciable by the percussing finger. With a smaller effusion there is usually flatness over the lower part of the chest and dulness or tympanitic resonance above; sometimes dulness is found behind and tympanitic resonance at the apex in front. The line of flatness may change with the position of the patient. Grocco's sign is found in the majority of cases. This is a small triangular area of dulness posteriorly, with its base to the spine, on the side opposite to the effusion. The signs on auscultation are variable, and probably lead to more frequent mistakes in diagnosis than in any other pulmonary affection. Bronchial breathing and bronchial voice over the fluid are common in children. Absence of both voice and breathing is sometimes met with, but it is exceptional. The bronchial breathing over fluid usually differs from that over consolidation, in that it is feebler and distant; in some cases, however, it is indistinguishable from that heard over consolidation. Friction sounds may be heard above the level of the fluid, or when the fluid is subsiding, and there may be bronchial râles.

Diagnosis.—The most reliable signs for diagnosis are displacement of the heart, flatness on percussion, absence of râles and friction sounds,

and (usually distant) bronchial breathing. In an infant, flatness should always lead one to suspect fluid. If there is flatness over one entire lung, the existence of fluid is almost certain. Between serous and purulent effusions a positive diagnosis is possible only by the use of the exploring needle. This should be employed in every case, as it is important to know early whether or not we have a purulent effusion to deal with. The amount of fluid in serous pleurisy is generally less than in the purulent variety.

Pleurisy is further to be differentiated from pneumonia, and from tuberculosis. From pneumonia, the acute cases are distinguished by the lower temperature, the less severe prostration, lower leucocyte count and the fact that all the general symptoms are milder; but especially by the physical signs. The differential diagnosis by the physical signs between effusion and the various forms of consolidation is considered under the head of Empyema.

Prognosis.—In the acute cases complicating pneumonia, a serous pleurisy is very apt to become purulent. Other forms of pleurisy with effusion, as a rule, terminate in recovery by absorption. In cases coming on without definite cause there should always exist a suspicion of tuberculosis, and hence every patient should be closely watched for the development of the other signs of that disease.

Treatment.—In the great majority of cases, only symptomatic treatment is required during the acute period. The patient should be kept in bed, and pain relieved by opium, counter-irritation, or dry cups. After the fever has ceased the patient may be allowed to sit up, but all exertion should be carefully avoided if the effusion is large. Sudden death has occurred when this rule has been violated. The patient should in suitable weather be kept in the open air as much as possible. In the course of a few weeks the effusion usually subsides under simple tonic treatment. Absorption may sometimes be hastened by counter-irritation and diuretics; but convalescence is apt to be slow, and it may be several months before the health is entirely restored.

The removal of the fluid by operation is indicated in the acute form when it is accumulating so rapidly as to endanger life from the pressure upon the heart and lungs; also when there is no tendency to absorption after from two to three weeks of constitutional treatment. In such cases nothing is to be gained by waiting, and harm may be done to the lung by the delay. The usual method is by aspiration. In the acute stage enough should be removed to relieve the patient's symptoms, aspiration being repeated if necessary in twelve or twenty-four hours. In infants, particularly, there is great danger of wounding the lung when aspiration is repeated several times. This usually results in the production of pneumothorax which may mask the re-accumulation of the fluid. In

the subacute stage the removal of a portion of the fluid may be all that is required, spontaneous absorption of the remainder often taking place quite promptly.

EMPYEMA

Fully nine-tenths of the cases of empyema in children under five years either occur with or follow pneumonia, being usually the sequel of the form described as pleuropneumonia. In some of these cases, however, the pleurisy masks the pneumonia, so that the former appears to be the primary disease. Tuberculosis is a rare cause in early childhood, but becomes more frequent after the seventh year. Empyema may complicate scarlet fever, measles, or any of the other acute infectious diseases. It is met with in pyemia from all causes. It may occur in the newly born as the result of infection through the umbilical wound or the skin. It is seen with suppurative inflammations of the joints and with osteomyelitis. It may complicate suppurative processes in the abdomen, such as appendicitis or purulent peritonitis. Among the local causes may be mentioned traumatism, necrosis of a rib, and the rupture into the pleural cavity of abscesses originating in the mediastinum, in the thoracic wall, or below the diaphragm.

Since empyema is generally secondary to pneumonia, its causes are mainly those of that disease. Of 186 cases observed at the Babies' Hospital in which the nature of the organism was determined it was as follows, 83 per cent of these patients being under two years of age:

Pneumococcus	115	=	64.0 per cent.
Streptococcus	26	=	14.4 "
Staphylococcus	14	=	7.8 "
B. Influenzae	1	=	0.5 "
B. Tuberculosis	1	=	0.5 "
Mixed infections	23	=	12.8 "

Two-thirds of the mixed infections showed the pneumococcus. The predominance of the male sex is even more striking than in pneumonia. Of 204 consecutive cases in the same institution the proportion of males was 68.6 per cent.

Lesions.—Empyema is an inflammation with the production of serum, fibrin, and pus. In most of the cases—and the younger the child the more frequent its occurrence—it succeeds pleuropneumonia. There is first an exudation of fibrin with an excess of pus cells. As the process continues, more and more pus is poured out, with serum. At first the fluid collects in small pockets formed by the slight adhesions. As it accumulates these are broken down, and the pleural cavity may be filled with pus. If the original inflammation involved but a portion of

the pleura the empyema may be sacculated. This is often seen even in infants. Much has been written regarding inter-lobar empyema. This we have never seen either at autopsy or operation and we believe it to be a very rare condition in children. Localized empyema is, however, seen very often. It is usually posterior and over one lower lobe, but may be in any part of the chest. In very rare cases there may be several sacs containing pus, separated by septa. Such a condition we have never seen in empyema following pneumonia. The cases just described are those in which, in infants and young children, the pneumococcus is regularly found. The amount of fibrin is large, covers both surfaces of the pleura, and many large masses float in the fluid. The pus is usually thick, creamy, and odorless. In another group of cases the evidences of inflammation of the pleura are much less marked, and in some they may be slight. There is but little fibrin in the exudate, and adhesions are rare. In this form the streptococcus or the staphylococcus is the organism usually found. In these cases the inflammation may be purulent from the outset, and the pus is thinner than in the preceding variety. Empyema following pneumonia is occasionally preceded by a serious effusion which, although almost clear, is usually found to contain great numbers of bacteria, usually pneumococci.

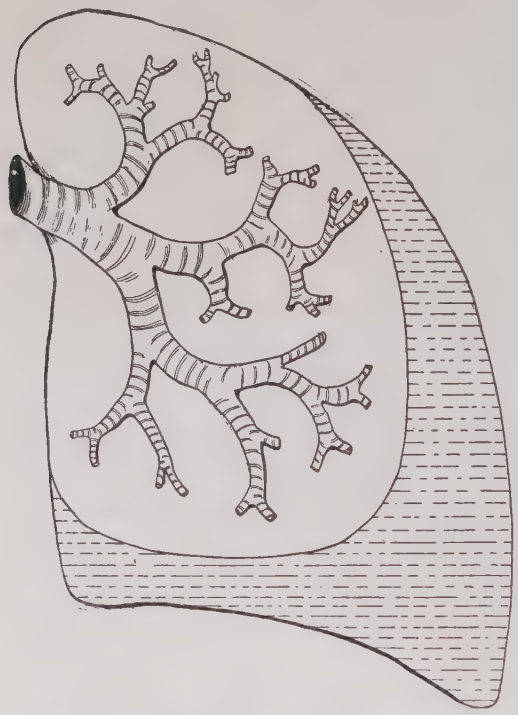


FIG. 73.—SECTION OF A LUNG. To illustrate the distribution of the fluid in the chest with a moderate effusion (diagrammatic).

Even when the fluid is moderate in quantity it is not all at the bottom of the chest, but is generally distributed over a considerable part of its surface, and its depth at the middle and upper part of the chest may be only half an inch, or even less. When the accumulation is larger, the lung does not float on the surface of the fluid, but the fluid surrounds the lung, which is compressed on all sides (Figs. 73, 74, 75). The

heart is displaced; the diaphragm and the abdominal viscera are somewhat depressed, and there may be bulging of the chest on the affected side. The amount of fluid in ordinary cases is from four to twenty ounces, although in neglected cases it may accumulate until it amounts to four or five pints. The effect upon the lung will depend upon the amount of fluid and the duration of the compression. When the quantity is small, or when the pressure is removed early, the lung in most cases readily expands, air being forced into it from the opposite lung, especially during the act of coughing. With the exception of adhesions, recovery may be complete. Although wide in extent, the adhesions are not usually strong enough to interfere seriously with the function of the lung. If the pressure is great and has been long continued, the adhesions over the lung may become so dense and firm that expansion is difficult, and can at best be only partial. In such cases recession of the chest wall occurs. In old cases expansion is still further interfered with by the changes taking place in the lung itself, usually a low grade of interstitial pneumonia.

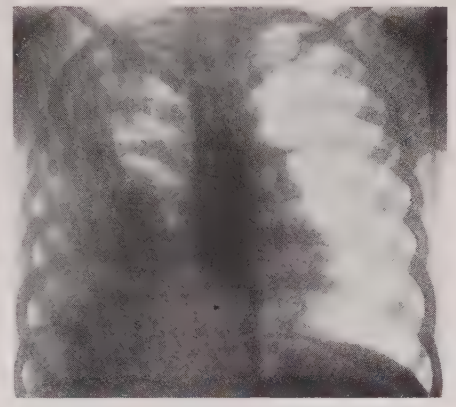


FIG. 74.—EMPYEMA, RIGHT SIDE; MODERATE EFFUSION.

FIG. 75.—EMPYEMA WITH LARGE EFFUSION.

In cases receiving no treatment, absorption of the pus is possible, but is not to be expected. It generally seeks an external outlet; the lung may be perforated and the pus be evacuated through the bronchi, or external rupture may occur, generally in the neighborhood of the nipple. In still other cases the pus may burrow along the spine, or through the diaphragm reaching the peritoneum.

Empyema is more often of the left than of the right side, the proportion being about three to two. It is double in about three per cent of all cases, but much oftener in infants. The most serious complication

in young children is pericarditis, usually with empyema of the left side; in older children a frequent complication is pulmonary tuberculosis.

Symptoms.—When it occurs as a sequel of pneumonia, the symptoms of empyema may follow those of the original disease without any intermission; or after the temperature has been normal or nearly so for sev-

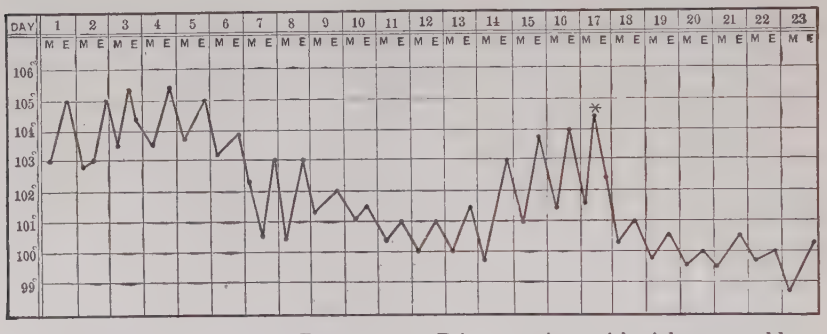


FIG. 76.—EMPYEMA FOLLOWING PNEUMONIA. Private patient, girl, eight years old; severe pneumonia terminating by lysis; development of empyema indicated by secondary temperature; operation on seventeenth day; recovery.

eral days it may rise again, sometimes quite suddenly, but more often gradually. With this accession of fever there are other symptoms pointing to an increase in the thoracic disease. (See Figs. 76 and 77.) After scarlet fever or other infectious diseases, the onset of empyema is often signalized by cough, rapid breathing, and the other usual symptoms of pulmonary disease. In the cases where empyema appears to be primary, the onset is acute, with high temperature and general and local symptoms resembling those of pneumonia. After such a beginning, the chest may be found full of pus by the third or fourth day. In older children empyema may come on with gradual, and even insidious symptoms, there being only slight fever, dyspnea, and cachexia. Marked leucocytosis, 25,000 to 40,000, is almost invariably present. The proportion of polymorphonuclear cells is usually from seventy-five to eighty-five per cent.

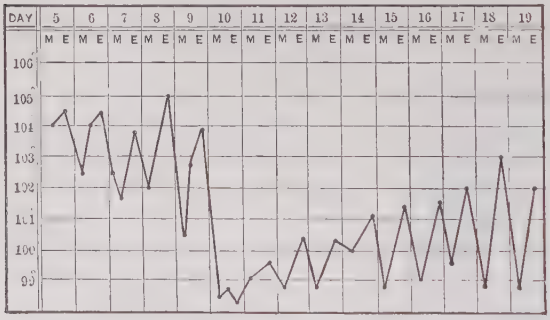


FIG. 77.—EMPYEMA FOLLOWING PNEUMONIA. Hospital patient, two years old; single-lobe pneumonia with crisis on ninth day; no resolution, but instead gradual development of signs of empyema closely following the temperature curve.

Of 88 patients with empyema in the Babies' Hospital, nearly all under three years old, positive blood cultures were obtained in 41 per cent. The pneumococcus was the organism usually found.

Whatever may have been the mode of onset, when the pus has been in the chest for some time the symptoms are fairly uniform. During the acute stage there are present pallor, anemia, and prostration. The respirations are always accelerated, being usually from forty to seventy a minute. Cough is present; there is dyspnea, sometimes marked, but more often it is scarcely noticeable. The temperature is exceedingly variable; usually it ranges from 101° to 103° F. A typical hectic temperature with sweating is in our experience very rare. The pulse is rapid but of fair strength. There is loss of flesh, sometimes even emaciation and anorexia; occasionally there is diarrhea. The stage of acute symptoms may last from two to four weeks. This may be succeeded by a subacute stage which may last for months. In this there is little or no fever; the patient seems convalescent so far as regaining strength and color are concerned; but cough, dyspnea, and rapid respiration continue. The chest shows no change in signs from those of the acute stage. In chronic cases the general symptoms closely resemble those of tuberculosis. There may be clubbing of the fingers, albuminuria, swelling of the feet, and often marked lateral curvature of the spine.

Diagnosis.—The physical signs do not differ essentially from those present in serous effusion. If there are signs of fluid in the chest and the patient is under three years of age, the fluid is likely to be purulent; and from the third to the seventh year, pus is much more often found than serum. A marked leucocytosis always makes pus more probable. In every case in which fluid is suspected the exploring needle should be used, because of the great importance of an early diagnosis. The skin should be surgically clean and the needle sterilized. Pus may not be found because the needle is too small, too short, or because it is introduced too far into the chest; for when the layer of pus is thin, the needle may be pushed through this into the lung.

The physical signs upon which most reliance is to be placed are, marked dulness or flatness on percussion, feeble breathing, and displacement of the heart. When in a young child these signs are present, whether general or localized, a needle should be inserted, and if pus is not found at the first trial, repeated punctures should be made until the presence or absence of fluid is definitely settled.

Empyema is most frequently confounded with unresolved pneumonia. The differential points are that in unresolved pneumonia the dulness is usually over a single lobe, râles or friction sounds are heard, and there is no displacement of the heart; empyema may give flatness over the whole lung, or over the lower half of the chest in front and behind, râles and

friction sounds are absent over this area, and the heart is usually displaced. In both conditions we may get bronchial breathing and voice. The confusion of acute pneumonia or tuberculosis with empyema, generally arises from placing too much reliance upon auscultation. In pleuropneumonia, with an excessive exudation of fibrin, the signs may be identical with those of empyema, except that the heart is not displaced. We have several times seen pulmonary tuberculosis, with caseation of an entire lobe, which gave signs that were identical with those of a sacculated empyema. It is by the exploring needle, and by that alone, that empyema is positively differentiated from these pulmonary conditions.

There are some other thoracic diseases from which the diagnosis may be even more difficult. A large pericardial effusion gives signs which are in some cases identical with those of empyema of the left side. Marked displacement of the heart to the right is always a strong point in favor of empyema; besides, such pericardial effusions are extremely rare in young children. A pulmonary abscess of considerable size—also a rare condition—gives signs identical with those of localized empyema, and is only distinguished from it by autopsy or operation. Abscesses from broken-down tuberculous glands may give signs resembling those of localized empyema, and like an empyema may point between the ribs in the upper part of the chest. The constitutional symptoms of empyema may at times resemble typhoid fever or malaria; but it is distinguished from them by the physical signs and by the examination of the blood.

Prognosis.—The outcome of a case of empyema depends chiefly upon the age and general condition of the patient, the exciting cause, the duration of the symptoms, the presence or absence of serious complications, and the treatment. The mortality in young children is high, particularly in the first year. Of 204 consecutive cases admitted to the Babies' Hospital, the death rate was as follows:

First year	74	cases;	mortality	74	per cent
Second "	93	"	"	59	" "
Over two years	37	"	"	13	" "

It is often difficult to understand why the cases in infancy do so badly; many of these children on admission are in excellent condition and do well for a week or more after operation. Then the temperature rises, the patients lose ground rapidly and die of exhaustion during the third or fourth week. Their inability to expand the compressed lung properly seems an important factor, as this condition is almost invariably found at autopsy. Very seldom is there trouble with drainage. Em-

pyema in children over three years old seen reasonably early and receiving proper treatment, almost invariably terminates in recovery, unless the disease is double or serious complications exist. The best results are seen in the cases that follow pneumonia. Pneumococcus and staphylococcus cases have a better outlook than those due to the streptococcus or to mixed infections. Tuberculosis before the seventh year is an exceedingly infrequent cause, and gangrene of the lung and general pyemia are both rare causes in early life. It is these three conditions that make the prognosis of the disease in adults so serious. Great delay in operation makes the prognosis worse, because the more difficult the expansion of the lung the more tedious is the disease, and the greater the likelihood of a sinus remaining. With proper early treatment these patients not only recover, but in most cases the recovery is surprisingly complete. Retraction of the chest and its resulting lateral curvature of the spine are rare, and seen only in neglected cases. In very many patients, in which a reasonably early operation was done, it is impossible, after the lapse of two or three years, to detect any difference whatever in the physical signs of the two sides of the chest. There are few serious diseases the treatment of which is more satisfactory than that of acute empyema in older children.

Spontaneous recovery in empyema may take place by absorption; but this is so rare that it is not to be expected. The pus may be evacuated spontaneously through a bronchus, rupture having taken place through the visceral pleura. When this occurs, a large amount of pus may be coughed up in a few hours, usually followed by immediate, but not always lasting, improvement. This is the most favorable of the natural terminations. External opening may take place, usually in the region of the nipple. There is an area of redness, then a fluctuating tumor, and finally the pointing of an abscess. The discharge may continue for months, or even for years. External opening rarely occurs until the disease has lasted several months. Of 19 cases of empyema in children collected by Schmidt, in which a spontaneous discharge of pus occurred either externally or through a bronchus, there were 17 deaths and 2 recoveries. Empyema may burrow behind the diaphragm into the abdominal cavity, appearing as a psoas abscess; it may burrow posteriorly into the lumbar region; it may rupture into the esophagus, or through the diaphragm into the peritoneal cavity. All these conditions, however, are very rare. The chances of spontaneous cure in empyema are small. Of 32 cases, reported by Rilliet and Barthez, which received no surgical treatment, 21 proved fatal. The statistics of empyema before the general adoption of surgical treatment are appalling. Patients were either worn out by the protracted suppuration, or died from amyloid degeneration, pneumonia, or tuberculosis.

Treatment.—The medical treatment relates to the patient only; the disease is always to be treated surgically. Like any other acute abscess, empyema requires free incision and drainage with proper aseptic precautions.

Aspiration as a means of cure is now seldom used. Unquestionably it sometimes suffices to cure empyema, most frequently when it is localized, and when the cause is the staphylococcus. How often this occurs is shown by the following statistics: Of 139 cases which we collected that were treated by aspiration, 25 were cured, 8 of these by a single aspiration; 13 died, and the remaining 101 were afterward subjected to other treatment. The objections to aspiration are, that it is not possible to remove all the pus; that it affords no opportunity for the removal of the large fibrinous masses; besides, there is the danger, especially with repeated aspirations, of puncturing the lung and producing pneumothorax. Simple aspiration, therefore, is to be advised in children only for temporary relief when the amount of fluid is large and the patient's condition such as to make it desirable to defer any more serious operation. It is to be advised also in the case of double empyema until sufficient adhesions have formed upon the first side operated upon to make opening of the other pleural cavity safe. Aspiration, followed by the injection of formalin and glycerin, is not, from our experience, to be recommended.

Incision and Drainage.—In most cases it is preferable to delay incision until the period of most acute inflammation has subsided, as shown by lower temperature and stationary physical signs. This is usually seen two or three weeks after the pleural invasion. Such delay is not admissible if either the local condition or the temperature points to a steady increase in the disease; nor when the general symptoms indicate increasing prostration or sepsis. The dangers attendant upon general anesthesia are considerable, and in most cases it is better not to employ it. We have known of six deaths on the table during operation, and in several other cases have seen very alarming symptoms occur. Chloroform is more to be feared than ether. It is well, when possible, to employ local anesthesia. The most favorable point for incision is the posterior axillary line in the seventh intercostal space upon the right side, the eighth upon the left. In a case of localized empyema, the lowest point at which pus can be obtained by puncture should be chosen. The incision is made in the middle of the intercostal space. No matter what has been found by puncture on previous occasions, the exploring needle should always be used at the time of operation and at the site of the incision before the latter is made. The incision should be only large enough to allow the introduction of two tubes side by side into the pleural cavity. The hemorrhage is very rarely sufficient to require a

ligature. It is undesirable to attempt to empty the chest at the time of operation. A better plan is to insert the tubes at once and apply the dressings, allowing the pus to escape slowly. The drainage tubes should be of rubber, fenestrated, one-fourth to three-eighths of an inch in diameter and about three inches long. To secure them from slipping into the cavity, the outer end should be transfixed by a large safety-pin before introduction.

Both the original operation and the subsequent dressings should be done with strict aseptic precautions on account of the danger of secondary infection, the occurrence of which adds to the severity and prolongs the course of the disease. After the third or fourth day the second tube may be omitted and the remaining one gradually shortened. Often, by the end of the fourth week, and sometimes before, the tube may be dispensed with altogether. The time of redressing and the removal of the tube is determined by the amount of discharge and the temperature.

Simple incision with drainage is in infants to be preferred to rib resection. It requires less time, no general anesthetic, and is altogether a much less severe operation. Our experience is that following it pulmonary expansion takes place with more facility than when a large opening is made in the chest, and that in the great majority of cases it secures all the room required for drainage. There are, however, some disadvantages. The smaller opening may not give adequate room for the removal of large masses of fibrin. In old cases, particularly, it not infrequently happens that after the chest has been emptied the ribs become so closely approximated that but little space is left, and the drainage tubes are pinched. Furthermore, the contact of the tubes may lead to erosion and superficial necrosis of the adjacent ribs, sometimes to exostoses.

Incision with Rib Resection.—This is the operation to be preferred with children over three or four years of age. It is sometimes needed as a secondary operation in cases which cannot be properly drained by the simple incision owing to approximation of the ribs. The removal of an inch of rib is usually all that is necessary. This allows the insertion of the finger into the chest, the removal of masses of fibrin and the breaking down of adhesions if any are present, and it secures free drainage. The extensive manipulation which is sometimes practiced in these cases with older patients is not admissible with young children.

Siphon Drainage.—This method of treatment introduced many years ago by Bulau and recently revised and improved by Kenyon has much to commend it for young infants. The opening made into the chest is a very small one admitting only a single large drainage tube. The wound is tightly strapped about the tube so as to exclude air. The

thoracic tube is connected by a glass tube with several feet of rubber tubing and this with the wash bottle which contains a sterile salt solution. This bottle is suspended beneath the patient's bed or placed upon the floor. The character and the amount of discharge can thus readily be seen. As the tube often need not be changed for several days the child is spared the fatigue and distress of frequent dressings. The exclusion of air diminishes the danger of secondary infection and favors the expansion of the lung. The bottle is emptied once or twice a day, the air being meanwhile excluded by clamping the tube. The chief objection to this method of treatment is interference with drainage by the blocking of the tube. Such an occurrence is at once recognized by inspection of the fluid in the wash bottle. A fibrin plug can sometimes be removed by suction, or by raising the bottle and allowing some of the sterile solution to flow into the chest, afterwards siphoning it out; but in many cases the tube must be removed to clear it. Even if this is carefully done by keeping a tight pad on the wound, air cannot be excluded from the chest nor secondary infection of the pleura entirely avoided. When repeated blocking of the tube occurs the treatment may have to be discontinued. The tube can usually be worn for ten or twelve days, after which it loosens owing to ulceration about it and an air-tight wound can no longer be maintained. The short tube with the dressing of gauze and cotton is substituted. An extensive trial of siphon drainage leads us to recommend its use in many cases of empyema in infants.

FIG. 78.—DEFORMITY AFTER AN OLD EMPYEMA OF THE LEFT SIDE FOR WHICH ESTLANDER'S OPERATION WAS PERFORMED. Portions of five ribs were removed. (From a photograph seven years after operation.)

Washing out the pleural cavity is indicated in cases in which

the pus is foul. A single washing for the purpose of removing fibrin is the routine practice of some surgeons. For this a warm, sterilized salt solution should be used. Personally, we have seldom found this necessary. Repeated irrigations should not be employed. The usual duration of the discharge in cases treated by simple incision is from three to six weeks, the average being about five weeks.

A persistence of temperature or a fresh rise after operation most frequently indicates defective drainage, generally due to blocking of the tube; but this is not always the case. It may be due to pneumonia, either a continuance of the old process or the lighting up of a new one, to abscess of the lung, to empyema of the opposite side, to pericarditis, or to some cause outside the chest, very frequently otitis. The mistake is often made of allowing the tube to remain for too long a time, so that a sinus is kept open which would otherwise close.

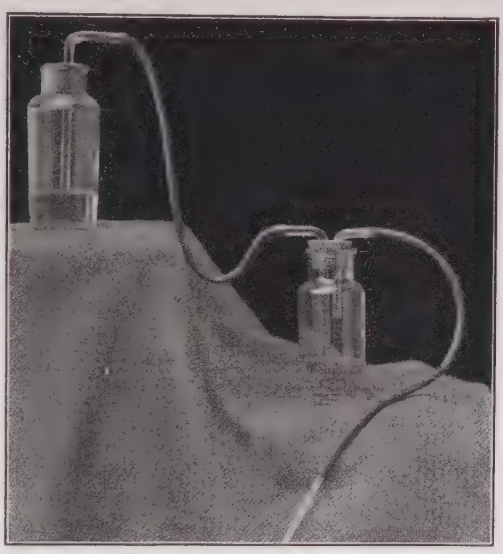


Fig. 79.—James's Apparatus for Expanding the Lung after Empyema.

In chronic cases, or those which have been long neglected, some further operative treatment is often necessary. The lung is so bound down by firm adhesions that further expansion is impossible, and even after the chest has receded to its utmost, so that the ribs are in contact, there still remains a cavity which cannot close. For such cases the only hope is an operation by which portions of several ribs are removed, thus allowing a greater collapse of the chest wall. This is known as "thoraplasty," or "Estlander's operation." The operation is of itself a serious one, and only to be advised as a last resort in inveterate cases. Such an operation is, of course, always followed by very great deformity (Fig. 78).

Methods of Inducing Expansion of the Lung.—In most of the cases, particularly the recent ones, complete expansion of the lung takes place without any difficulty, the chief agent being the cough. In some cases this may be insufficient. The apparatus, devised by James, shown in the accompanying cut (Fig. 79), serves at the same time as a toy for the child's amusement and as a most efficient means of inducing forced

expiration. One bottle is placed a few inches higher than the other, and the child blows a colored fluid from the lower into the higher bottle, allowing it to siphon back. Blowing soap bubbles often answers the same purpose.

SECTION V

DISEASES OF THE CIRCULATORY SYSTEM

CHAPTER I

PECULIARITIES OF THE HEART AND CIRCULATION IN EARLY LIFE

The Fetal Circulation.—During the latter part of fetal life the circulation may be briefly described as follows: The purified blood comes from the placenta through the umbilical vein. Entering the body, it divides at the under surface of the liver into two branches, the smaller one, the ductus venosus, communicating directly with the inferior vena cava; the larger branch joining the portal vein, so that its blood traverses the liver, and then enters the inferior vena cava through the hepatic vein. From the inferior vena cava the blood enters the right auricle, like that returned from the head and upper extremities by the superior vena cava. A part of the blood now passes directly into the left auricle through the foramen ovale; the remainder, through the tricuspid orifice into the right ventricle. As the requirements of the pulmonary circulation are not great, only a small part of the blood is sent through the pulmonary artery to the lungs; the greater portion passes from the pulmonary artery through the ductus arteriosus into the aorta, joining here the blood from the left ventricle. The blood thus finds its way from the right heart to the left, only in small part by way of the lungs, the greater part passing directly from the right auricle to the left, or from the right ventricle into the aorta through the ductus arteriosus. From the aorta, the blood reaches the placenta through the umbilical arteries, which are a continuation of the hypogastric arteries, which in turn are given off from the internal iliacs.

Changes in the Circulation at Birth.—With the ligation of the umbilical cord, the circulation through the umbilical vein and arteries and the ductus venosus ceases. With the establishment of respiration and the consequent increased demands made by the pulmonary circulation, the blood ceases almost at once to pass through the ductus arteriosus, and very soon through the foramen ovale. The umbilical vessels during the first few days of life are filled with small thrombi, which become organized. By the end of the first week, these vessels, as well as the

ductus venosus, are usually closed at their extremities, although they may remain patulous throughout the greater part of their extent for several weeks. They subsequently atrophy to the condition of small fibrous cords. For some weeks before birth the circulation through the foramen ovale is slight, it being gradually obstructed by the growth of a septum which nearly fills the space at birth. After the first week of extra-uterine life very little, if any, blood passes through it, although complete closure of the foramen often does not take place until the middle of the first year. In fully one-fourth of the autopsies we have made upon infants under six months old, there have been found minute openings at the margin of the foramen ovale, but they are usually oblique, and closed by the valvular curtain so as effectually to obstruct the current of blood. The ductus arteriosus is first closed by a clot, which becomes organized and blends with the products of a proliferating arteritis. It is rarely found open after the tenth day, and by the twentieth it is almost invariably obliterated.

Size and Growth of the Heart.—The relative size of the heart is slightly greater in infancy than in later life, it being smallest at about the seventh year. The average weight at the different periods of life is as follows:

The growth of the heart is rapid during the first three years, and nearly proportionate to that of the body. It is slowest from the third to the tenth year, and most rapid from the eleventh to the fifteenth year. At birth, the thickness of the right ventricle is very nearly the same as that of the left, the ratio being 6 : 7. The left ventricle, however, grows very much more rapidly than the right, so that at the end of the second year the ratio is 1 : 2, which is nearly that of the rest of childhood.

The Pulse.—The pulse in early life is not only more frequent, but it is very much more variable than in adults. The following is the average pulse-rate in healthy children during sleep or perfect quiet:

Six to twelve months	105 to 115 per minute.
Two to six years	90 " 105 " "
Seven to ten years	80 " 90 " "
Eleven to fourteen years	75 " 85 " "

The pulse is a little more frequent in females than in males, and more frequent when sitting than when lying down. Muscular exercise or excitement increases the pulse-rate by from twenty to fifty beats. Very trivial causes disturb not only the frequency but the force of the pulse. The pulse in young infants may be irregular even in health and during sleep. When rapid, it is frequently irregular without special significance. No dicrotism is seen in the pulse wave of early infancy.

The circulation is much more active in infancy than in later childhood; thus, according to Vierordt, the entire round of the circulation is

accomplished in the newly born in twelve seconds; at three years, in fifteen seconds; in the adult, in twenty-two seconds.

Age.	Ounces.	Grams.	Ratio to Body Weight.
Birth	0.50	14	
1 year	1.25	35	1 to 225
2 years	1.87	53	
3 "	2.25	64	
7 "	2.80	80	1 to 280
14 "	5.84	166	1 to 222
Adult	8.50	241	1 to 226

The figures in infancy are from one hundred and fifty-five observations made in the New York Infant Asylum; the others are taken from Sahli.

Position of the Apex Beat.—In the infant the heart is placed somewhat higher, and occupies a position a little nearer the horizontal than in the adult. This is partly due to the higher position of the diaphragm. The apex beat is therefore higher and farther to the left than in adult life. According to the observations of Wassilewski and Starck, whose combined examinations with reference to this point were made upon over 2,100 children, the apex beat is, as a rule, outside the mammary line until the fourth year; if it is less than one-third of an inch beyond the nipple, it can not be considered abnormal. From the fourth to the ninth year, the apex beat is in or near the mammary line. After the thirteenth year, under normal conditions, it is invariably within that line. During the first year the apex beat is usually found in the fourth intercostal space; from the first to the seventh year, it is found with about equal frequency in the fourth and the fifth spaces; after the seventh it is usually, and after the thirteenth year it is always, when normal, in the fifth space. The position of the apex beat may be considerably modified by severe deformities of the chest resulting from rickets, Pott's disease, or lateral curvature of the spine.

Examination of the Heart.—*Inspection.*—Bulging of the precordium is a frequent and important sign of cardiac disease during childhood. The cardiac impulse is generally weaker than in the adult, and often it is difficult to locate the apex beat owing to the thick layer of adipose tissue covering the chest.

Palpation.—This is usually a much more satisfactory method than is inspection for determining the position of the apex beat. For this purpose the child should be in the sitting posture, with the body inclined slightly forward. Great displacement of the apex beat is always significant, and should lead one to suspect pleuritic effusion; lesser degrees of displacement to the left indicate hypertrophy, especially of the left ventricle; epigastric pulsation indicates hypertrophy of the right ventricle.

Percussion.—This may be done either by the finger or the percussion hammer. A light blow should be used, on account of the thinness and elasticity of the chest walls. In percussing the heart, changes in the percussion note are generally better appreciated if one proceeds from the lung toward the heart rather than in the opposite direction. The outline of the area of "relative" or deep cardiac dulness, especially in small children, is proportionately larger than in the adult. This may lead to the mistaken opinion that the heart is enlarged, when it is really of normal size. The upper boundary of this area is at the second interspace or the upper border of the third costal cartilage, at the left margin of the sternum; from this point the line of dulness extends in a curved direction outward and downward, the extreme left limit being at or slightly beyond the mammary line at the fourth interspace. On the right side the line of dulness extends downward from the second interspace in a slightly curved direction along the parasternal line. The lower border is indeterminable on account of the liver.

The area of "absolute" or superficial cardiac dulness, or that part of the heart uncovered by the lung, resembles in shape the same area in the adult, but it is relatively larger.

Auscultation.—This is of little value unless the child is quiet. For an accurate diagnosis the stethoscope is indispensable, but auscultation should always be practiced with the naked ear as well. The rhythm and rapidity of the child's heart action are much more easily disturbed than are the adult's, and such disturbances are consequently much less significant. The rapidity of the heart in infancy is ordinarily so great as to make it difficult to determine the exact period in the cardiac cycle at which a murmur occurs. Normally, the loudest sound is the first sound at the apex; the weakest sound is the second sound at the aortic orifice. The pulmonary second sound is regularly louder than the second aortic up to the fourteenth year and in some children almost to adult life.

In consequence of the small size and the thin walls of the chest, all sounds, both normal and pathological, appear relatively louder than in the adult, and the area of diffusion is therefore much greater. Thus it is a frequent occurrence for murmurs to be heard all over the chest both in front and behind.

Reduplication of the heart sounds, in consequence of the valves of the two sides not closing exactly together, is not uncommon in children, and may be due simply to excitement. During the first four years of life nearly all the abnormal murmurs heard are systolic. Accidental murmurs are very common. They may be due to anemia and to many other conditions; although not so common as in older patients, they are by no means rare in infants.

In older children, especially when lying on the left side, there is

often heard a sound in the early part of diastole, the so-called "third heart sound." This is only heard in the region of the apex and always follows the second sound by an interval longer than occurs in true reduplication. The sound has the character of a dull, distant thud. It is never blowing. The sound probably results from the sudden tension of the auriculoventricular valves produced by the rapid entrance of blood into the ventricle. It should be recognized that this sound is not an abnormality. Failure to do so may cause errors in diagnosis.

CHAPTER II

CONGENITAL ANOMALIES OF THE HEART

Etiology.—Of the causes of congenital cardiac disease little is definitely known. It occurs more often in first-born children than later ones; 16 of 50 cases being in first children (Still). It is often associated with other forms of imperfect development, notably of the brain, as in Mongolian idiocy. An attempt has been made to connect cardiac malformations with syphilis. A syphilitic family history is very seldom found; but Warthin has brought forward additional reason for suspecting syphilis since he has found that a positive Wassermann test is given by some of the children with congenital cardiac disease, but in our experience this has not been found with sufficient frequency to establish a causal relation. There has not been adduced any evidence to show that rheumatism plays a part.

Lesions.—The congenital anomalies of the heart may be grouped under three general heads:

1. Malformations resulting from imperfect development of certain parts of the heart, most frequently one of the septa. Either the ventricular or the auricular septum may be affected, or that dividing the pulmonary artery from the aorta. Such failure in development perpetuates conditions which are normal in the early months of fetal life. There may also be atresia of any one of the orifices, absence of one or more of the valvular leaflets, or of any one of the large vessels.

2. The results of fetal endocarditis. The effects of this condition vary according to the time of its occurrence. It is almost invariably of the right side, most frequently affecting the pulmonic valves. Valvular disease in fetal life leads not only to hypertrophy and dilatation, but also interferes with the normal development of the heart by preventing the closure of the auricular or ventricular septum or the ductus arteriosus, these being kept open by way of compensation. It is not clear how

important a part fetal endocarditis plays in the production of congenital lesions, certainly not so important a one as it was formerly believed.

3. Persistence of fetal conditions, such as the foramen ovale or ductus arteriosus. This is usually by way of compensating for some malformation the result of imperfect development or of fetal endocarditis. Very rarely a fetal condition may persist when no sufficient reason for it can be found.

In the following table are given the lesions found in two hundred and forty-two cases collected from medical literature:

Frequency of the different lesions in 242 autopsies upon cases of congenital cardiac anomaly

Defect in the ventricular septum	149	cases;	the	only	lesion	in	5	cases.
Defect in the auricular septum, or patent foramen ovale	126	"	"	"	"	"	9	"
Pulmonic stenosis or atresia	108	"	"	"	"	"	6	"
Patent ductus arteriosus	68	"	"	"	"	"	3	"
Abnormalities in the origin of the great vessels	45	"	"	"	"	"	0	"
Pulmonic insufficiency	17	"	"	"	"	"	0	"
Tricuspid insufficiency	6	"	"	"	"	"	0	"
Tricuspid stenosis or atresia	3	"	"	"	"	"	0	"
Mitral insufficiency	1	"	"	"	"	"	0	"
Mitral stenosis or atresia	6	"	"	"	"	"	0	"
Aortic insufficiency	1	"	"	"	"	"	0	"
Aortic stenosis or atresia	6	"	"	"	"	"	0	"
Transposition of the heart	2	"	"	"	"	"	0	"
Ectocardia	1	"	"	"	"	"	0	"

The most frequent associated lesions

Pulmonic stenosis with defect in the ventricular septum	92	cases,	the	only	lesions	in	20	cases.
Pulmonic stenosis, with defect in the auricular septum	52	"	"	"	"	"	8	"
Defects in both septa	82	"	"	"	"	"	17	"
Pulmonic stenosis with defects in both septa	36	"	"	"	"	"	21	"

From this table it will be seen that, in the great majority of cases, several lesions are present, the most frequent combinations being pulmonic stenosis with defective ventricular septum; pulmonic stenosis with defective auricular septum; the three lesions associated; or the first two with a patent ductus arteriosus. Stenosis of the isthmus of the aorta, although not noted in this series, is not a very uncommon lesion; the obstruction is in the arch of the aorta beyond the point where the large vessels are given off.

Defect in the Ventricular Septum.—This is the most frequent lesion in congenital cardiac disease, and in half the cases is associated with pulmonic stenosis. The defect is generally at the upper part of the

septum (Fig. 80). It is usually from one-fourth to one-half an inch in diameter, but not infrequently there is a large defect; or the septum may be entirely absent, the heart then consisting of but three cavities—two auricles and one ventricle. If the auricular septum also is wanting, as may be the case, the heart has but two cavities. Frequently there are also abnormalities in the origin of the great vessels. The pulmonary artery and the aorta may be given off from a common ventricle, or the aorta may arise partly from one ventricle and partly from the other. If pulmonic stenosis or atresia is present, the opening in the ventricular septum is conservative, affording a channel for the passage of blood from the right to the left side of the heart.

FIG. 80.—CONGENITAL CARDIAC DISEASE. The left ventricle is shown with a defect in the ventricular septum, the opening being just beneath the aortic valve. (From a patient dying in the Babies' Hospital.)

Patent Foramen Ovale, or Defect in the Auricular Septum.—Although this is one of the most common congenital malformations, it is not one of the most important. It rarely occurs alone, but is frequently found with pulmonic stenosis or a defect in the ventricular septum. Small oblique openings in the auricular septum—usually at the foramen ovale—are not infrequently met with in autopsies upon young infants, but they are of no importance. In pathological conditions the opening is from one-fourth to one inch in diameter, and there may be more than one opening. A defect in this septum is frequently secondary to pulmonic stenosis.

Patent Ductus Arteriosus.—As a solitary lesion this is rare, but it is frequently associated with pulmonic stenosis, usually with a defect in one or both septa. It is then one of the channels by which the blood may find its way to the lungs when the pulmonary orifice is obstructed. It is not a malformation, but simply the persistence of a fetal condition usually necessitated by other changes in the heart. But the direction of the blood current is the opposite of that which exists in fetal life.

Pulmonic Stenosis.—This is one of the most frequent and most important lesions. It was formerly looked upon as being often due to fetal endocarditis, but is now believed in most cases to be due to a failure

of development of the infundibulum of the right ventricle. It is often a primary lesion, and when marked it is always accompanied by other changes, most frequently by a defect in one or both septa or by a patent ductus arteriosus. This is important, as being more constantly associated with cyanosis than is any other congenital lesion. Most of the children who live beyond six or seven years with cyanosis have this lesion, always accompanied by others of a compensatory character. The amount of obstruction varies from a slight narrowing of the orifice to complete atresia. The seat of obstruction may be at the pulmonic orifice, in the conus arteriosus, or in the pulmonary artery just beyond the valves. If there is atresia, the pulmonary artery is very small, and may be rudimentary.

Pulmonic Insufficiency.—This lesion is relatively rare. It is usually the result of fetal endocarditis, but there may be absence of the pulmonary valve. It is most frequently associated with a defect in the ventricular septum.

Tricuspid, mitral, and aortic disease are very infrequent and usually seen in cases with multiple defects. Atresia or stenosis is much more common than insufficiency.

Abnormalities in the Origin of the Large Vessels.—These are quite frequent; but, as will be seen from the table, they are always associated with other lesions. Three forms are seen: (1) Transposition of the large vessels—the pulmonary artery is given off from the left, and the aorta from the right ventricle. (2) Both arteries arise from a common trunk. This is usually due to an incomplete development of the lower part of the septum dividing the two arteries. Usually the pulmonary artery appears to be a branch of the aorta. This condition is frequently associated with other abnormalities, often with so large a defect in the ventricular septum that there is really but one ventricle. (3) The aorta has an abnormal origin, arising from the right ventricle, or partly from both ventricles. This also is associated with a large defect in the ventricular septum. When described as arising from both ventricles, the aorta is usually given off directly above the line of the septum.

An abnormality in the number of valvular segments is quite frequent, but seldom impairs the valve's function. In rare cases a valve is rudimentary, and it may be absent, generally at the pulmonic or tricuspid orifice. Absence of the right auricle and absence of the pericardium have been recorded; also opening of the pulmonary veins into the right auricle, and a single pulmonary artery. In one case in the series there was ectocardia, this being associated with a congenital fissure of the sternum. We have seen two very remarkable instances of congenital cardiac displacement; the heart was in both situated in the abdominal cavity. The pulsations could be plainly seen and felt just above the

umbilicus. In each case there was a congenital defect of the abdominal walls and also an opening in the diaphragm.

Transposition of the heart, or true dextrocardia, was recorded but twice in this series of cases. The transposition of the heart alone is a very rare condition. It is not so unusual to find transposition of all of the thoracic and abdominal viscera. In the last two years we have seen three such cases.

Secondary Lesions.—In congenital malformations the right heart is usually found hypertrophied, since there are present one or more of the fetal conditions in which the greater part of the work is thrown upon the right ventricle. Such hypertrophy is in most cases accompanied by some dilatation. Changes in the muscular wall of the left heart alone are exceedingly rare. In four cases there was evidence of malignant endocarditis which was the cause of death, all but one of these patients being adults.

Symptoms.—The symptoms of congenital cardiac disease are usually manifested soon after birth. Of 128 cases in which the time of the first symptoms was noted, they were congenital, or appeared during the first month, in 85; after one month and during the first year, in 18; from one to sixteen years, in 15; while in 10 no symptoms were observed until after puberty. Congenital cardiac disease is one of the causes, but not a frequent one, of death during the first days of life.

The most striking objective symptom is cyanosis. This is present in most of the severe cases; but, considering all varieties, cyanosis is more often absent than present, and it may be absent even with serious lesions. It may be slight and noticed only upon exertion, as upon coughing or crying, or it may be intense and constant, giving the skin a dark, leaden color, and the mucous membrane of the mouth a raspberry hue. The view that cyanosis depends upon an admixture of arterial and venous blood is generally discredited. In the great majority of the cases at least, the explanation is a deficient oxygenation of the blood in the lungs, owing to some interference with the pulmonary circulation. In sixty-three per cent of the cases with cyanosis in the series, there was found pulmonic stenosis or atresia, or a small pulmonary artery. Cyanosis is of much value in diagnosis, as in acquired cardiac disease it is rarely persistent. The degree of cyanosis and its constancy are of some importance in determining the gravity of the lesion, although cyanosis alone is not to be depended upon.

Another frequent symptom is the enlargement of the terminal phalanges known as clubbed or "drum-stick" fingers (Fig. 81) and toes. This almost invariably accompanies cyanosis, and is generally proportionate to it. The enlargement, which usually involves all the phalanges, is probably due to venous obstruction. Occasionally there are seen dysp-

nea, edema of the face or lower extremities, dropsy of the serous cavities, and hemorrhages, particularly hemoptysis and epistaxis.

There is generally marked dyspnea on exertion in the cases in which cyanosis is present; but in most of those without cyanosis there is no dyspnea, and, in fact, no objective or subjective symptoms, even though the murmur may be very loud. The majority of the cyanotic children are undersized and develop slowly; in them the problem of nutrition is a difficult one.

In cases accompanied by cyanosis, or with obstruction to the pulmo-

FIG. 81.—CLUBBING OF THE FINGERS IN CONGENITAL HEART DISEASE. (From a boy five years old.)

nary circulation, a polycythemia is present. The increase in the number of red cells is generally proportionate to the cyanosis; the average of fifteen cases studied in the Vanderbilt Clinic by Wile was 7,495,000; the highest was 12,480,000. The hemoglobin is usually correspondingly increased. In the series mentioned the average was 107 per cent, the highest being 130. The number of white cells is changed very slightly, if at all; the average in these cases was 10,200, which disproves the theory of blood concentration. The best explanation of the polycythemia seems to be that it is compensatory, and that the blood hypertrophies like other tissues. The blood-forming organs are stimulated to greater activity by the demands of the tissues for oxygen. The quantity of blood remains the same, but the number of red cells and the hemoglobin, and

consequently the oxygen-carrying power, are very greatly increased. This in part compensates for the smaller amount of blood that can traverse the lungs and there become oxygenated.

Diagnosis.—The most important diagnostic features are cyanosis, the presence of a loud murmur, and signs of enlargement of the right heart.

Murmurs are present in fully nine-tenths of the cases. They are almost always systolic in time, are heard loudest to the left of the sternum, usually at the base or over the body of the heart, rarely at the apex. They are in most cases widely diffused, often being audible all over the chest. The point of maximum intensity is important for diagnosis. In the great majority of cases only a single murmur is heard. A systolic murmur is usually due to pulmonic stenosis, deficient ventricular septum or aortic stenosis, very rarely to mitral or tricuspid regurgitation. Since these conditions are very often associated, it may be difficult to tell upon which one the murmur depends.

A patent ductus arteriosus usually gives a prolonged, continuous murmur with systolic intensification, which is loudest in the second or third left interspace. From the presence of a continuous murmur in a child, a diagnosis of patent ductus arteriosus can be made. The associated lesion is almost always pulmonary stenosis. In a young child, a loud murmur at the base of the heart with cyanosis almost always means congenital disease. A thrill is often present but it is not important for a diagnosis.

Enlargement of the right heart, chiefly from ventricular hypertrophy, is present in most of the cases.

A diagnosis of the precise nature of the malformation is very difficult, and in the great majority of cases only a probable diagnosis is possible. Nearly all the cases are complex, and the variety of combinations is very great. A study of the histories and autopsies of the cases in this series reveals many apparently contradictory facts. Loud murmurs are sometimes heard which are difficult to explain by the lesions, and murmurs may be absent when there is every reason from the post-mortem findings for expecting their presence. With reference to the other conditions, we can not do better than give the more frequent clinical symptoms with the results of the autopsies in the series of cases which have been presented.

A Systolic Murmur at the Base with Cyanosis.—This was the most common combination met with, and was present in about one-third of the entire number. In over eighty per cent of the cases with these symptoms, pulmonic stenosis was found. The remainder were complicated cases of quite a wide variety. Pulmonic stenosis was usually associated with a defect in one of the cardiac septa, or a patent ductus arteriosus.

A Systolic Murmur without Cyanosis.—In this series of autopsies this was not a frequent combination, being noted but six times. It is usually dependent upon a defect in the ventricular septum without pulmonic stenosis. Clinically, however, this is more often seen, in fact it is one of the most common types. The murmur is generally loudest at the left margin of the sternum at the third space. There is a striking absence of all other symptoms. We have watched a number of such patients grow to maturity and go through serious attacks of illness without showing any symptoms referable to the heart.

A Systolic Murmur at the Apex with Cyanosis.—Of the six cases with this combination, all were examples of complex malformation, the most frequent lesions being a defect in the auricular septum, transposition of the great vessels, and patent ductus arteriosus.

Cyanosis without murmurs was noted fourteen times. It usually indicates either pulmonic atresia or the transposition or irregular origin of the great vessels, but is sometimes seen when lesions such as usually give murmurs are found at autopsy.

Diastolic murmurs were heard in but two cases, and depended upon pulmonic insufficiency. Diastolic murmurs are also heard when an acute endocarditis causing aortic or pulmonary insufficiency supervenes upon a congenital lesion. We have seen one such case and several have been reported.

Absence of both cyanosis and murmurs was recorded in five cases. The lesions found were: atresia of the aorta, both arteries arising from the right ventricle, or defective septa.

The only cases, therefore, in which a fairly certain anatomical diagnosis can be made are those of pulmonic stenosis, deficient ventricular septum, and patent ductus arteriosus.

Diagnosis of Congenital from Acquired Disease.—Congenital disease may be suspected if the patient is under two years of age; if there is no history of previous rheumatism; if the murmur is atypical in its location, character, or transmission; if there is a very loud murmur at the base or over the body of the heart, and if there is evidence of enlargement of the right heart. If cyanosis and clubbing of the fingers are present the diagnosis is almost certain.

Especially difficult are the cases without cyanosis seen in older children. But absence of hypertrophy of the left ventricle, continued absence of subjective symptoms, even with a very loud murmur, and a lesion which does not increase—all point strongly to a congenital malformation.

Diagnosis of Congenital from Accidental Murmurs.—This is often a more difficult matter than to decide between congenital and acquired disease. From a murmur alone one should be very cautious in making a

diagnosis of cardiac malformation in a very anemic infant. Anemic murmurs are systolic, usually basic, unaccompanied by enlargement of the heart, usually heard in the carotids, often in the subclavian arteries, but are seldom so loud as those due to malformations. In some cases it may be necessary to watch the progress of the case before deciding the question.

It is not uncommon in children to find at about the level of the nipple at the left border of the sternum a soft systolic murmur best heard in the recumbent position, which, as it usually disappears, must be considered functional. It may be mistaken for a congenital murmur, but is not so loud.

Prognosis.—Of 225 cases, 60 per cent were fatal before the end of the fifth year, and nearly one-half of these during the first two months; while sixteen per cent of the patients lived over sixteen years, and eight per cent over thirty years. The prognosis in cases without cyanosis is good: in many children the lesion has apparently little effect on the health or development. The prognosis is much worse in cases with cyanosis, and generally it is bad in proportion to the degree of cyanosis. The loudness of the murmur has no prognostic importance.

In the cases fatal soon after birth the usual lesions are large defects in the septa, transposition of the great vessels, or pulmonic atresia. In five of twenty-three cases dying thus early, the heart had but two cavities. Lesions which are compatible with the longest life are minor septum defects, and pulmonic stenosis which can be compensated for by patency of the ductus arteriosus and in other ways. Many exceptional instances are recorded in which patients have lived a long time in spite of extreme deformities. One child with transposition of the pulmonary artery and aorta lived two and a half years. Tiedemann's patient lived eleven years with a heart consisting of three cavities—two auricles and one ventricle—and with constant cyanosis. In three cases reported by Rokitansky, the patients lived over forty years with rudimentary auricular septa; cyanosis is not mentioned as being present. Gelpke's patient had cyanosis, and lived twenty-seven years with rudimentary auricular and ventricular septa, and with no tricuspid opening. Patients with serious congenital cardiac lesions are especially susceptible to pulmonary disease of all kinds and occasionally develop malignant endocarditis. Almost any acute illness may prove fatal.

Treatment.—These patients are prone to develop at times attacks resembling angina pectoris, which are best relieved by nitroglycerin or by the use of morphin hypodermically. No treatment is of the slightest avail in diminishing the amount of deformity or promoting the closure of any of the abnormal openings. All cases are to be treated symptomatically.

CHAPTER III

PERICARDITIS

Inflammation of the pericardium is uncommon in infancy and early childhood, only two cases being seen in 726 consecutive autopsies at the New York Infant Asylum. But in later childhood pericarditis is more frequent and more serious than the same disease in adults.

Pericarditis is almost invariably a secondary disease, following (1) empyema or pleuropneumonia; (2) acute rheumatism; (3) acute infectious diseases, especially scarlet fever; (4) pyemia; (5) tuberculosis; (6) local conditions. The relative importance of these causes differs with the age of the child. In infancy and early childhood nearly all the cases complicate disease of the lung or pleura, more frequently of the left side. After the fourth year rheumatism takes the first place as an etiological factor. Pericarditis is then generally associated with endocarditis, and may precede or follow the articular manifestations of rheumatism. Following scarlet fever, pericarditis often occurs in connection with nephritis or multiple joint inflammations. In typhoid fever also it is usually associated with pneumonia or joint lesions. Pyemia may be a cause in the newly born, or it may occur in connection with disease of the bones or joints in older children; in both it is usually associated with similar lesions of other serous membranes. Tuberculous pericarditis is more frequent after the third year, and is generally secondary to pulmonary tuberculosis. Among the local causes may be mentioned traumatism, ulceration of a foreign body from the esophagus into the pericardium, disease of the sternum, ribs, or vertebrae, and abscesses resulting from cheesy bronchial lymph nodes.

Lesions.—Pericardial transudations, or an increase in the normal pericardial fluid, are met with in many conditions in which there is a very marked degree of anemia, general dropsy, or a weak heart, particularly of the right side. Generally from one to two ounces of clear serum are found in the pericardial sac.

Pneumococcus pericarditis is always acute and closely resembles in its lesions the inflammation of the pleura due to the same cause. In the milder cases there is seen only a fibrinous exudate. In the more common and more severe forms the visceral and parietal pericardium is covered with a thick coating of fibrin and pus (compare pleuropneumonia), or more pus cells and serum may be poured out and the sac contain fluid pus. The amount is usually small, one-half to one ounce, but it may be as much as a pint. When the inflammation is excited by other pyogenic organisms, the staphylococcus or the streptococcus, the lesions are similar to those just described.

In rheumatic pericarditis the inflammation may be a plastic one with a fibrino-cellular exudate (dry pericarditis) or sero-fibrinous (pericarditis with effusion). The inflammation generally begins at the base of the heart and affects both the visceral and parietal layers. The quantity of fluid present is usually small, not exceeding two or three ounces; exceptionally as much as a pint may be present. It may be clear or slightly turbid. More important than the pericarditis are the associated changes in the heart muscle. These are present in every severe case. To the myocarditis and consequent dilatation the most serious symptoms of pericarditis are due.

Purulent pericarditis may be set up by a foreign body ulcerating into the sac, by the rupture of a mediastinal abscess, or by general pyemia. In these circumstances the process may be purulent from the outset. Any of the pyogenic bacteria may be found.

External or mediastinal pericarditis is always associated with mediastinal pleurisy, and results in more or less extensive adhesions of the pericardial and pleural surfaces, with an increase in the connective tissue of the mediastinum. This is often a tuberculous process. When severe, it may cause compression of the large blood-vessels, but seldom in any other way produces symptoms. With this form there is usually inflammation of the internal layer of the pericardium as well. Only inflammation of the internal layer is ordinarily considered as pericarditis, the other form being preferably classed as mediastinitis.

Pericarditis with an effusion of blood is very rare in children. It may occur from the rupture of organized adhesions or in certain blood states such as purpura, and very rarely in tuberculosis.

With acute tuberculosis there is usually only a deposit of miliary tubercles, or there may be a small serous or sero-sanguinolent effusion. In chronic cases there may be a tuberculous inflammation with the formation of caseous nodules, new connective tissue, and extensive adhesions. This generally occurs in connection with pulmonary tuberculosis—sometimes with tuberculous peritonitis.

In any form of pericarditis complete recovery, so far as pathological conditions are concerned, is rare—if, indeed, it ever occurs. After a rheumatic pericarditis adhesions remain, which may be slight, but are often complete, causing entire obliteration of the pericardial sac. Such adhesions are followed by secondary changes. The growth and development of the heart are interfered with, and there may be sufficient pressure upon the coronary vessels to lead to degeneration of the muscular walls and chronic dilatation of the heart.

Symptoms.—A pericardial transudation, or dropsy of the pericardium, is very rarely large enough to make a diagnosis possible.

External pericarditis is seldom recognized during life, there being no

symptoms except those of the pleurisy with which it is associated. Occasionally there may be heard, particularly if the inflammation is anterior, a pleuritic friction sound which is increased with the systole of the heart. The pulse may be weak during inspiration, and there may be an increased area of cardiac dulness. If the inflammation is chiefly posterior, it causes only the symptoms of mediastinitis, which is recognized principally by its pressure effects upon the great vessels. It may produce edema of the face or of the lower extremities, ascites, enlargement of the liver and spleen, but rarely albuminuria. It is usually progressive, and lasts from a few months to two or three years, according to its cause.

Pericarditis in infancy is usually overlooked, not only on account of its rarity, but also from the obscurity of its symptoms. When pericarditis develops at the height of an attack of pneumonia, as it usually does, there may be no new symptoms, or at most only increased prostration with perhaps a more rapid or slightly irregular pulse. On auscultation, if practiced early, one may hear pericardial friction sounds; but these are masked by the pulmonary signs and in infants seldom made out. The most striking sign is that the cardiac sounds formerly distinct are now feeble and distant, at times almost inaudible. Later there may be increased dulness from pericardial effusion, or from dilatation. The physician should be on the watch for it in infants with pleuropneumonia, especially of the left side.

Rheumatic pericarditis affecting as it generally does older children is easier of recognition. Localized pain and tenderness are usually present and also a certain amount of embarrassment of the heart's action, manifested by precordial distress, palpitation, or a tumultuous heart action with a rapid and at times an irregular pulse. There is often vomiting, dyspnea, and a teasing, dry cough; there may be orthopnea and some cyanosis. Sometimes there is delirium.

The earliest physical sign of pericarditis is a friction sound which can generally be heard all over the precordium, though sometimes only over a small area at the base. The sound is usually a double, to-and-fro sound, synchronous with the movement of the heart. In character, the sound is rough, scratching or grating, not at all blowing in character, and, while it may be heard widely over the heart, is not transmitted. With the accumulation of the fluid, the friction sound may only be heard over a restricted area, but almost always persists at the base even though fluid may be present in large amount. It differs thus radically from the friction sound in pleurisy with effusion. Very early there is an increase in cardiac dulness which is often considerable. This may be due to effusion or to cardiac dilatation, which is apt to occur in all severe cases of pericarditis. With early and rapidly developing dulness it is safe to assume that some dilatation is present. The dulness

can be made out both to the left and to the right of the heart. On the right side it is usually first noted in the fifth right intercostal space with an obliteration of the normal acute cardiohepatic angle, an obtuse angle resulting. The dulness usually does not extend more than an inch or two beyond the right border of the sternum and a similar distance beyond the left mammary line, but with very extensive effusion there may be dulness to the right of the right mammary line, and as far as the left anterior axillary line. (Figs. 82, 83.)

The area of dulness with small effusions is triangular or pear-shaped with the base below. With large effusions it is almost circular, in which

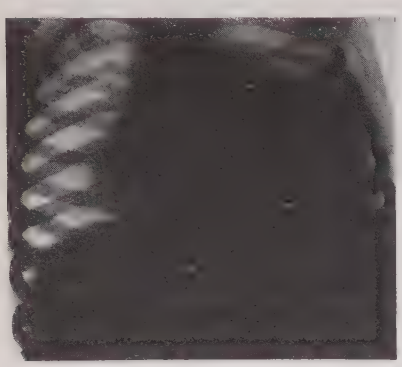


Fig. 82.—Pericarditis with Effusion. Anterior view, showing moderate distention of the pericardium, especially to the left of the middle line; right border at A. Boy eight years old.

Fig. 83.—Pericarditis with Effusion. Same patient as Fig. 82, but taken four days later. Great distention of the pericardium; right border at B. Complete recovery by absorption.

case the cardiohepatic angle again becomes acute. There also may be dulness to the left of the vertebral column behind. When there is considerable effusion, the apex beat is feeble and may be displaced upward. It may be impossible to locate it. The cardiac sounds are diminished in intensity and may be almost inaudible. Of assistance in diagnosis is sometimes the disproportion between the cardiac sounds and the force of the pulse—the latter may be nearly normal when the cardiac sounds can barely be heard. As the result of pressure upon the lung from large accumulations of fluid, bronchial breathing may be heard posteriorly, at and inside the spine of the scapula.

In cases terminating fatally the progress of the disease is quite rapid, the entire duration being seldom longer than three or four weeks, and it may be much less. Pneumonia often develops toward the close. When ending in recovery improvement is very slow and it may be two or three months before the patient is out of bed, and a much longer time before even a moderate degree of health is established.

Prognosis.—Acute pericarditis due to the pneumococcus in infancy

almost invariably ends fatally and in older children this is the usual termination. Occasionally at the later age resolution may take place before pus forms, or the pyopericardium which ensues is successfully opened and drained. Purulent pericarditis from other causes is usually fatal. In rheumatic pericarditis the outlook for life is better, but this with its associated myocarditis is without doubt the gravest manifestation of rheumatism in early life. No complication is more to be dreaded, both on account of immediate and remote dangers. Of forty-eight cases of acute pericarditis reported by Still in which this supervened during endocarditis, forty proved fatal in the course of a few weeks. In patients who do not die from the disease the remote consequences by reason of adhesions and subsequent dilatation are very serious.

Diagnosis.—Pericarditis is recognized by knowing when to look for it—in infants with pneumonia, in older children with rheumatism. The difficulties of diagnosis of dry pericarditis are very much greater in young children owing to the very rapid action of the heart. Dry pericarditis is recognized by the friction sounds, which are best heard over the base and are to be differentiated from endocardial murmurs. Pericarditis with effusion is to be differentiated from dilatation of the heart and from pleuritic effusion. From dilatation, the diagnosis is very difficult in childhood, but the recognition of small effusions is not essential, since the important condition is the accompanying dilatation. Large effusions may be mistaken for a sacculated empyema of the left side; in the latter, however, the heart is generally crowded to the right. When empyema and pericarditis coexist, it may be impossible to recognize the condition. The diagnosis between serous and purulent effusions can be made only by aspiration.

Treatment.—In an attack of acute pericarditis the patient should be kept in bed, absolutely quiet, and an ice-bag used over the heart. A layer of thin flannel should be placed beneath the bag. During the acute stage it should be applied constantly with perhaps a few hours' omission during the night. To be effective much attention to detail is necessary. Some children will not tolerate ice and for them dry heat may be substituted. It often mitigates the pain. Counter-irritation by mustard from time to time is useful, but blisters should not be employed in children. Leeching is much used in England, not so much in this country as its merits warrant. Four or five leeches are applied over the sternum or liver. The especial indications for the use of leeches are cyanosis, marked dyspnea, and dilatation as shown by increase in the cardiac dulness. A rapid increase in dulness is to be regarded as mainly due to dilatation rather than effusion. Opium is, in our opinion, of more value than any other drug. It has a steadying influence upon the excited heart, it relieves pain and also quiets the distressing cough. The

form of administration is immaterial. The patient should be kept moderately under its influence throughout the active stage of the attack. Digitalis is sometimes useful, but must be used with caution. Alcohol is not indicated. Strychnin and caffein may be used when symptoms of heart failure are present; but very little is to be expected from any drug. For the concurrent rheumatism anti-rheumatic remedies should usually be continued. Serous effusions usually subside under general treatment. With very large serous effusions aspiration may relieve distressing symptoms, after which the rest of the fluid may undergo absorption. If the exploring needle shows the fluid to be purulent, incision and drainage should be practiced as in empyema. The results of aspiration for pyopericardium are exceedingly unfavorable. Of eighteen cases collected by Keating, only four recovered. In puncturing the pericardium the point usually selected is a little to the left of the border of the sternum in the fifth intercostal space, the needle being directed upward and outward. In cases which do not end fatally a prolonged period of rest in bed is imperative on account of the dilatation.

CHRONIC PERICARDITIS WITH ADHESIONS

This is not a very uncommon condition. It is usually general, but may be localized. The youngest case which has come under our observation was in a child sixteen months old, who died from acute bronchopneumonia. The adhesions were old and general, the pericardial sac being completely obliterated. Chronic adhesive pericarditis may follow single or repeated attacks of acute rheumatic pericarditis; it may be tuberculous. The pericardium may become very greatly thickened and its cavity obliterated; it may be adherent externally to the pleura, diaphragm, and chest wall. Other changes are usually present in the heart. It is often the seat of chronic myocarditis; the cavities are usually greatly dilated, and the heart walls much hypertrophied. Valvular lesions may be present.

Partial adhesions cause no symptoms by which they can be recognized, and even general adhesions sufficient to obliterate the pericardial sac may be found at autopsy when not suspected during life. This is one of the conditions in which, after it has led to considerable dilatation of the heart, sudden death sometimes occurs. Usually there is pallor, slight cyanosis, localized edema of the chest and abdominal walls, and dyspnea upon slight exertion. The liver and spleen are often enlarged and there may be ascites. These symptoms often lead to errors in diagnosis.

The heart is almost invariably much enlarged, chiefly from dilatation.

On inspection, there may be bulging of the chest wall, with a diffused and often feeble or absent apex beat. The characteristic signs are a systolic retraction of the chest at or near the apex of the heart, sometimes at the tip of the sternum. This is due to the external pericardial adhesions, and is often better appreciated by palpation than by inspection. It is followed by a rapid rebound, associated with diastolic collapse of the jugular veins. A *pulsus paradoxicus* may also be present. Percussion shows an increase in the cardiac dulness in all directions. The position of the apex and the percussion outline of the heart do not change with the posture of the patient, and the cardiac dulness is but little affected by full inspiration. A systolic murmur is often present. The diagnosis of adherent pericardium always presents difficulties, but it can be made with tolerable certainty in a considerable proportion of cases. On account of the enlargement of the heart and the frequency of murmurs, it is usually mistaken for valvular disease. The prognosis is very bad. The lesion is a permanent one, and tends to increase. The treatment is symptomatic.

CHAPTER IV

ENDOCARDITIS AND VALVULAR DISEASE OF THE HEART

Endocarditis may occur even in fetal life. At this period it usually affects the right side of the heart, and is one of the important causes of congenital malformations. In infancy, acute endocarditis is exceedingly rare, but a single instance being found in over two thousand autopsies upon children under three years of age of which we have records. From the third to the fifth year it is less rare, and after five years is quite common.

The following table gives the age and sex in a series of cases of valvular disease:

Age.	1 year.	2 years.	3 years	4 years	5 years.	6 years.	7 years.	8 years.	9 years	10 years	11 years.	12 years	13 years	14 years	Totals.
Males.....	..	1	2	2	4	6	4	9	8	6	5	7	6	1	55, or 38%
Females.....	..	1	3	5	7	9	10	3	11	12	14	4	2	3	90, " 62%
Total.....	..	2	5	7	11	15	14	12	19	18	19	11	8	4	145

The proportion as to sex is very nearly the same as in our cases of rheumatism. Sturges, in 100 cases of chronic endocarditis, gives fifty-six per cent females and forty-four per cent males.

Endocarditis is usually spoken of as secondary to rheumatism; it is rather to be regarded as a manifestation, often the first, of that disease. Of 117 cases in our series, ninety-three, or eighty per cent, gave a history of previous rheumatism. Of the 31 cases which at the first examination gave no history of rheumatism, 8 subsequently developed articular symptoms, and 2 chorea; so that nearly ninety per cent of this series of cases presented conclusive evidence of a rheumatic diathesis. Thirty per cent had chorea previously, or developed it while under observation. The proportion of rheumatic cases corresponds very closely with Cheadle's observations. In a series of 150 cases of valvular disease, Still found distinct evidences of rheumatism in 142.

Endocarditis may occur alone or with other manifestations of rheumatism. While frequently associated with acute articular rheumatism, in a much larger number it is seen with articular symptoms which are so slight as to be overlooked entirely or passed over as unimportant. It may occur with or follow chorea, tonsillitis, or torticollis, with or without articular symptoms. The proportion of rheumatic cases in which endocarditis occurs is much larger in children than in adults. In rare instances endocarditis is seen in the course of almost any of the infectious diseases, most frequently with scarlet fever, being often associated with pericarditis; but even in these conditions it is possible that it is sometimes rheumatic. The bacteriology of rheumatic endocarditis has not yet been determined with certainty.

Lesions.—In the great majority of cases endocarditis affects the mitral valve, and often only this. In 150 autopsies upon children dying of cardiac disease, Poynton found the mitral valve involved in 149, but in 76 of these the changes were not marked; in only 9 was there marked mitral stenosis. The aortic valve was affected in 51, but in only 9 was it seriously involved. Very striking was the frequency of pericarditis. Pericardial adhesions were present in 113 cases, and in 77 the adhesions were complete, i. e., the pericardial cavity was obliterated. These findings agree substantially with the observations of other English authorities, but in America the pericardial lesions are certainly not so prominent.

The pathological changes of acute endocarditis do not differ essentially in early life from those seen in adults. There is first an accumulation of bacteria upon the endocardium of the valves. These produce necrosis, which is followed by a clot formation, consisting chiefly of blood platelets and fibrin, in the meshes of which are leukocytes and a few red cells. The next change is a growth of new connective tissue cells and blood-vessels, which may be slight and superficial, but the rheumatic lesion usually extends deeply with an extensive proliferation of connective tissue which after a time undergoes contraction.

In the mildest forms of endocarditis it is possible for complete recovery to take place. In other cases there is left only a slight valvular thickening, not enough to interfere in any important way with function. In most patients, however, more marked changes are left. The valvular segments are swollen, adherent, somewhat shortened and consequently insufficient. Other changes in the heart usually accompany acute endocarditis. Dilatation is almost invariably present and is an important factor in producing insufficiency. In cases ending fatally there is very little hypertrophy; but if recovery occurs, hypertrophy develops and the lesion is compensated for in this way. A certain amount of myocarditis probably occurs in every severe case. It is most marked when pericarditis is also present. Emboli in children are rare. Subsequent attacks are exceedingly common and each one leaves the heart more seriously crippled.

Chronic inflammation may follow the first attack or more often occur after repeated attacks. The changes resulting from chronic endocarditis are practically identical with those seen in adult life and need not be described here. Emphasis, however, should be laid upon the fact that the younger the child the more rapid the progress of the disease.

Symptoms.—When endocarditis occurs as a primary disease, or when it is the only manifestation of rheumatism, it may begin abruptly with rather severe general symptoms—a temperature of 101° to 104° F., prostration, exaggerated heart action, restlessness, and sometimes dyspnea. More frequently, however, it begins much less acutely with only general malaise and slight fever, which often is not recognized without the thermometer. If the heart is not watched the diagnosis is not made and there may be no suspicion of the nature of the primary attack until some time afterward, when the existence of valvular disease is discovered. If, however, the heart is carefully and frequently examined there is heard, usually on the third or fourth day of the illness, a soft, blowing, systolic murmur at the apex.

Endocarditis occurring with rheumatism is by no means limited to those attacks with well-defined articular symptoms. It is very common and often severe when the articular symptoms are no more than stiffness, pain on motion, and slight swelling of the feet or ankles. There is no relation between the severity of these symptoms and the seriousness of the cardiac lesion. Occurring during chorea or after tonsillitis there may be nothing to call attention to the heart except sometimes an increased rapidity or irregularity of the pulse and possibly increased prostration; but frequently the cardiac condition is not suspected until the heart is examined.

Most of the cases of acute endocarditis seen in this country are of this mild type. Attacks of such severity as to produce death in the acute

stage are relatively rare here, in marked contrast with the observations of English writers.

The usual duration of acute endocarditis is from two to four weeks, the general symptoms slowly subsiding and, if the case progresses favorably, the cardiac symptoms improve, but there is usually left behind a somewhat damaged heart because of valvular disease. In cases progressing unfavorably a fatal termination may come in the course of from two to six weeks owing usually to one of three causes or a combination of these: (1) The rapid development of dilatation accompanied by the usual signs of cardiac insufficiency; (2) pulmonary complications, generally pneumonia; (3) the supervention of acute pericarditis.

Course of Chronic Valvular Disease.—Chronic valvular disease follows one or more attacks of acute endocarditis, and may exist for months and sometimes for years, before it is recognized. Its course is usually divided into two periods, the first being that in which compensation is present, and the second after compensation has failed. The duration of the stage of compensation is indefinite. The only subjective symptom that is of much diagnostic value is shortness of breath on exertion. Occasionally other symptoms are present, such as precordial pain, attacks of palpitation, headache, epistaxis, anemia, loss of weight, and cough. These are rarely constant, but come on when the patient's general condition for any reason is below normal. As a rule, there is in young subjects a tendency to an increase in the disease, although this is often slow, and may be interrupted by long periods in which the process appears to be stationary. At such times the patients either have no symptoms, or suffer only from a slight amount of inconvenience on marked exertion.

Failure in compensation is generally brought about by one of the following causes: The most frequent is an intercurrent attack of rheumatism with a fresh endocarditis, which in a short time leads to a very great increase in the heart's disability. It may be due to additional work thrown upon the heart from excessive muscular exertion, or to the strain of a prolonged attack of some acute illness, especially one that is liable to produce changes in the heart muscle, such as typhoid, diphtheria, or scarlet fever. It is sometimes the increased work which is thrown upon the heart especially at the time of puberty, owing to the rapid growth of the body. It may result from any cause which seriously affects the patient's general nutrition, particularly when this is associated with marked anemia.

The symptoms indicating failure of compensation are marked dyspnea or orthopnea and cough, sometimes accompanied by profuse expectoration, which may be bloody, and in rare cases there may be larger pulmonary hemorrhages. With these may be associated other signs of

pulmonary congestion and even pulmonary edema. The obstruction to the systemic venous circulation leads to dropsy, which usually begins in the feet, sometimes in the face. There may be general anasarca and dropsy of the serous cavities, especially the peritoneum and pleura; also enlargement and functional disturbances of the liver, enlargement of the spleen, dyspeptic symptoms, and chronic congestion of the kidney, with scanty urine and albuminuria. There may be dilatation of the superficial veins and cyanosis; and there may be cerebral symptoms, such as headache, dizziness, and fainting attacks. The pulse is small and soft, and the heart's action rapid and irregular; the cardiac sounds are feeble and often indistinguishable, and it may be impossible to decide what murmurs, if any, are present.

It is rare to see all the symptoms of chronic progressive cardiac failure in children under seven years, but toward the time of puberty they are common enough. The symptoms may increase in severity until death occurs, or they may be severe for a time and then nearly disappear, to return again after a longer or shorter interval.[1] Death may be due to sudden cardiac paralysis, to intercurrent nephritis, pneumonia, embolism, inflammation of the serous membranes, or to edema of the lungs.

Physical Signs.—Mitral murmurs are altogether the most common

[1] The course and termination of these cases of chronic valvular disease is well illustrated by the following history of a little girl who was under observation for nine years: When first seen she was seven years old, and gave a history of cardiac symptoms for one year. There was then present a loud mitral regurgitant murmur, with considerable hypertrophy. There was general dropsy, and all the symptoms pointed toward acute dilatation. Under treatment, the dropsy and other symptoms disappeared, and she went on comfortably for over a year. In her eighth and ninth years there were frequent attacks of subacute rheumatism, during which time the heart lesion steadily increased in severity. At twelve years there was an eruption of subcutaneous tendinous nodules, which remained for over two years. During this year there was heard for the first time a presystolic mitral murmur, accompanied by a very marked thrill, mitral stenosis having been gradually brought about by the slowly progressing endocarditis. This murmur gradually increased in intensity from that time, while the mitral regurgitant murmur became less distinct. The apex beat was then in the sixth space, two and a half inches to the left of the nipple. From the twelfth to the fifteenth year she grew very little in height or weight, and showed no signs of maturity, the cardiac symptoms being nearly stationary. In the fifteenth year she developed a marked enlargement of the liver and spleen with general dropsy and all the symptoms of cardiac insufficiency, these being the first symptoms of this character since she was seven years old. There was now heard for the first time an aortic regurgitant murmur in addition to the others formerly present. The symptoms disappeared under treatment in the course of a few months, but six months later returned with greater severity and were accompanied by albuminuria, the patient dying from heart failure in a few weeks. During the last exacerbation there was heard a double aortic as well as a double mitral murmur.

At autopsy the heart weighed fifteen ounces. There was a very great hypertrophy, especially of the right ventricle, which was as thick as the left. All the cavities were much dilated. The most important valvular lesion was mitral stenosis, the orifice not admitting the end of the little finger. The valves were the seat of calcareous deposits. The curtains of the aortic valve were thickened and adherent; there was also thickening of the pulmonic and tricuspid valves.

both in acute and chronic disease. Of 141 cases of valvular disease, in children under fourteen years, observed clinically, mitral murmurs were present in 135; in 131 the murmur of mitral insufficiency was heard, and in 99 this alone. In mitral insufficiency there is regurgitation of blood from the left ventricle into the left auricle during systole. There is heard a systolic murmur, synchronous with the apex impulse and with the first sound of the heart, which may wholly or in part replace the first sound. It is loudest at the apex, transmitted to the left, and is usually heard at the inferior angle of the left scapula. In acute endocarditis the murmur is at first very soft and usually increases in intensity for several days. It may be represented by the syllables "whoo-ta" pronounced in a whisper. After attaining its maximum the murmur changes but little for some time. It may then diminish and eventually disappear entirely; but usually a murmur of moderate intensity remains. The only other important sign of acute endocarditis is enlargement of the heart which is almost entirely from dilatation. If the acute inflammation supervenes upon an old lesion, the previous murmur becomes louder and harsher. In chronic endocarditis the murmur is similar to that of acute endocarditis but generally louder and more widely diffused, and may be audible all over the chest. It is accompanied by an accentuation of the pulmonic second sound and by signs of hypertrophy, especially of the right heart. When both these signs are wanting, the existence of mitral insufficiency is somewhat doubtful, as a similar murmur may be functional or accidental. In the early stages of the disease and during compensation, the signs of hypertrophy predominate; in the later stages or with broken compensation, those of dilatation.

Mitral stenosis is relatively uncommon. It occurs after repeated attacks of rheumatism, with a slowly progressing endocarditis. It is usually associated with mitral regurgitation. With this lesion there is obstruction to the flow of blood from the left auricle into the left ventricle. It is mainly compensated for by hypertrophy of the right ventricle, but to a certain degree also by hypertrophy of the left auricle. The characteristic murmur of fully developed mitral stenosis is presystolic, prolonged, rough in character, and terminates abruptly with a sharp first sound of the heart. It is loudest at or just above the apex, but is audible over only a circumscribed area. Quite as constant and important for diagnosis is the presence of a "purring thrill," which is very distinct upon palpation, and terminates sharply as the apex strikes the chest wall. This murmur is not common in children and is heard only in cases in which cardiac disease has lasted several years.

With milder grades of mitral stenosis, or earlier in the course of the disease, there may be heard, shortly after the second sound, a murmur softer in quality and of short duration. It is usually audible above and

to the inner side of the apex beat. In point of time this is often spoken of as the early diastolic murmur of mitral stenosis. It may be represented by the whispered syllables "whoo-ta-whoo," in which the first syllable is the mitral systolic murmur, which is somewhat prolonged; the second syllable is the second cardiac sound; the last is the early diastolic murmur, which is much shorter than the systolic murmur. The pulse of mitral stenosis is usually small.

Aortic lesions in children are much less common than mitral lesions, with which they are usually associated; they are seen in rather older patients. Aortic insufficiency is much more frequent than aortic stenosis. We have never seen it as the only lesion. It causes a regurgitation of blood from the aorta into the left ventricle during diastole. It is compensated for by dilatation and hypertrophy of the left ventricle. The signs of aortic insufficiency are a prolonged diastolic murmur, with or taking the place of the second sound of the heart, generally loudest at the left border of the sternum in the third space, and transmitted downward to the apex of the heart or the ensiform cartilage. This is invariably accompanied by signs of hypertrophy and dilatation of the left ventricle, which are usually marked. With great hypertrophy there is often bulging of the precordial area which may produce striking thoracic deformity. A characteristic symptom is the intense throbbing of the carotids, with the sudden distention followed by a complete collapse of their walls, and the "water-hammer" pulse of Corrigan. A capillary pulse is often seen.

Aortic stenosis, unless congenital, is very rare in early life, and almost never occurs as the only lesion. Aortic stenosis is compensated for by hypertrophy of the left ventricle. It causes a systolic murmur, which is usually loudest at the right border of the sternum in the second space, and is transmitted upward, being distinct in the carotids. The second sound is generally weak and may be replaced by a diastolic murmur. A systolic thrill over the aortic area is usually present. Without the signs of hypertrophy of the left ventricle, a positive diagnosis should not be made.

Tricuspid insufficiency is usually secondary to disease of the left side of the heart, occurring in its late stages. It most frequently follows mitral insufficiency, when it is usually due to dilatation of the right ventricle without changes in the valves. It may be secondary to certain diseases of the lungs, such as emphysema, chronic interstitial pneumonia, or chronic pleurisy, and it may be due to congenital malformation. Tricuspid insufficiency gives a systolic murmur, loudest over the lower part of the sternum, but heard usually over a small area. It is associated with signs of dilatation of the right ventricle. The jugular veins stand out prominently, and often show systolic pulsation, especially upon the right

side. There may be also systolic pulsation of the liver. The symptoms associated with tricuspid regurgitation are due to general systemic venous obstruction.

Tricuspid stenosis, pulmonic stenosis, and pulmonic insufficiency are practically unknown in childhood except as congenital lesions.

Prognosis.—The danger to life in acute endocarditis is not great unless it is accompanied by pericarditis; but when both are present the outlook is serious. Of 115 fatal cases reported by Poynton, thirty-five proved fatal in the primary attack. It is difficult during the active stage to foretell how serious will be the resulting damage to the heart. It is only by watching the progress of a case that one can decide. As a rule the younger the child the worse the prognosis.

Complete recovery from valvular disease is possible only when the lesions are very slight. Not many children die from chronic cardiac disease before reaching the age of eight or ten years. Up to about the time of puberty many children do very well; then they begin to lose ground, and may fail rapidly. But more often it is a fresh endocarditis accompanying an intercurrent attack of rheumatism which marks the beginning of a downward course. The proportion of children who have serious cardiac lesions before the age of six years and reach adult life in good condition is very small.

There are several features of cardiac disease in children, in consequence of which serious lesions tend to progress more rapidly than in adults. The muscular walls are less resistant, and hence dilatation occurs much more readily in childhood than in adult life. The heart must provide not only for constant needs, but for the growth of the body. If the patient's general nutrition is poor during the period of most rapid growth, this tells quickly and seriously upon the heart, and dilatation makes rapid progress. The demands made upon the heart at puberty are especially severe, by reason of the rapid growth of the body and the frequency with which anemia and malnutrition are seen at this time. There is always present the danger of rapid advances in the disease from intercurrent attacks of rheumatism, from which children are more likely to suffer than are older subjects. Extensive pericardial adhesions are frequent, and seriously handicap the heart, greatly increasing the tendency to dilatation. The effect upon the heart of poor food, unhygienic surroundings, and general malnutrition is much more marked than in adults.

These unfavorable conditions are in part offset by others in which the child has an advantage over the adult. Disease of the coronary arteries is very rare, and the valvular lesion which is most frequently met with—mitral insufficiency—is that which admits of the most complete compensation.

In making a prognosis in any given case, the amount of hypertrophy or dilatation which exists, and the presence or absence of pericardial adhesions are more important than the location or the special character of the murmur. The presence of valvular disease in childhood increases the danger from every acute disease, especially pertussis, diphtheria, pneumonia, and scarlet fever. The chances of recurring attacks of rheumatism must also be taken into account.

Probably the most important factor in the prognosis of chronic cardiac disease in childhood is the care and attention which the patient receives. While as a rule, if properly treated, these children do well among the well-to-do, they do very badly among the poor where suitable protection and treatment is impossible.

Diagnosis.—Valvular disease is to be distinguished particularly from conditions in which there are heard functional or accidental murmurs. According to our experience the latter are very common even in young children. Mistakes usually arise from attaching too much importance to the presence of murmurs, and too little to the changes in the walls and cavities of the heart, with which valvular disease is almost invariably associated. It is not always possible to decide whether a murmur is organic or functional until the patient has been for some time under observation and treatment, particularly when anemia is present. The diagnostic points, so far as the murmurs are concerned, are mentioned in connection with accidental murmurs.

Treatment.—The first and altogether the most important indication for every case of recent endocarditis is to secure for the heart as complete rest as possible, not only during the period of active inflammation, but for several succeeding weeks. The reason for this is that some dilatation is always present and this very readily increases. With children, proper rest can be secured only by keeping them in bed and in a recumbent position. The duration of the period of rest after mild attacks of endocarditis should be at least six weeks, and after severe attacks, three months. In these young patients changes in the walls of the heart take place very rapidly and the gravest consequences are liable to follow a neglect of these precautions. In old cases rest is indicated during every acute exacerbation; also whenever there is much dilatation and little hypertrophy, and whenever the signs of failing compensation are present. In these older cases rest is often impossible in the recumbent position; if secured at all, it must be obtained with the child in the sitting posture or at least propped up with pillows. Whether much can be accomplished by the administration of anti-rheumatic remedies after endocarditis has developed is very doubtful. Salicylates or aspirin and alkalis may be used unless they disturb the stomach. A child who is the subject of a chronic valvular disease should be constantly under a physician's observa-

tion. Irreparable harm often results from ignorant disregard of the simplest and most important rules of life for these patients.

Several distinct conditions may be present which call for quite different management. The essential points may be stated in a few words: For all recent cases and during all exacerbations, rest, complete and prolonged; for deformed valves with good heart walls and perfect compensation, fresh air, moderate exercise, and general tonics; for feeble heart walls, failing compensation and dilatation, rest and cardiac tonics.

During the stage of compensation, treatment directed especially to the heart is rarely necessary. The main purpose should be to maintain the patient's general nutrition at the highest possible point during the period of active growth. At the very least the patient should be carefully examined three or four times each year, in order that the physician may note the progress of the disease, and be able to direct the child's education, occupation, exercise, and surroundings so as to meet, as far as possible, the changing conditions. To this end, diet, sleep, study, and exercise should receive the most careful attention. If malnutrition and anemia are allowed to go on unchecked until they become severe, the cardiac disease may make rapid strides, and as much harm be done in a few months as otherwise might not occur in years. The question of exercise and recreation is always a difficult one to settle. Often too little latitude is given, and the heart, like the voluntary muscles, loses its tone. Every form of exercise requiring a prolonged severe strain should be forbidden, particularly swimming and competitive games, like ball and tennis, and others requiring much running; but skating, rowing, horseback exercise, regulated gymnastics, and cycling on the level—all in moderation—may be allowed not only without harm, but with positive benefit; but any of these, used immoderately, may be productive of great injury. All exercise should be taken with regularity and system, the amount being carefully measured by the child's condition, and increased freedom allowed only after watching the effect. If the patient is a boy who must earn his own living, the physician should see to it that the occupation chosen is not one likely to make special demands upon the heart or to expose him unduly to conditions likely to induce rheumatism.

Special watchfulness is required at the time of puberty to prevent overpressure in schools, and the development of anemia. The first symptoms of these conditions should be treated energetically, and if the heart seems to be overtaxed the child should be put to bed. Those who are specially liable to rheumatic attacks should, if possible, spend the winter and spring months in a warm, dry climate.

In the stage of failing compensation, the same general conditions are present as in adults, and they are to be managed in pretty much the same

way. When such symptoms are first seen, prolonged rest in bed should be insisted upon as the thing most likely to restore the normal conditions. Digitalis and strophanthus are useful in children with about the same indications as in adults, viz., dilatation, dropsy, low arterial tension, and weak pulse. They may be used in doses of from five to ten drops of the tincture every four to six hours for a child of ten years. If there is much dilatation of the right side of the heart the same treatment is indicated as described in pericarditis. One should be cautious about using digitalis for an irregular and overacting heart, opium being decidedly preferable under these conditions. An overloaded venous circulation may be relieved by diuretics, by saline purgatives, or even by venesection. Iron and tonics generally are indicated, particularly strychnin and cod-liver oil.

MALIGNANT ENDOCARDITIS

Malignant or ulcerative endocarditis is rare in childhood. Among the youngest cases reported are one by Bond in an infant of two and a half months, and one by Harris in a boy four years old. In Bond's case the mitral valve was affected. The infection was with the B. pyocyaneus. In Harris' case the right side of the heart was affected and the lesion was secondary to a congenital malformation. We have seen endocarditis of the mitral valve in an infant of six months following a septic arthritis of the knee. Of the cases reported in early life, most have been in children over ten years of age. Malignant endocarditis is never primary. It may be seen in any infectious disease or septic process. In seventy-five per cent of the cases it is ingrafted upon a previous valvular disease. In the series of collected cases of congenital malformations of the heart, there were four deaths from malignant endocarditis, all but one, however, occurring in adult life. The bacteria most frequently concerned are the staphylococcus or streptococcus, next the pneumococcus, and rarely the gonococcus, the influenza or the pyocyaneus bacillus.

Malignant endocarditis presents a great variety of symptoms, often making the diagnosis extremely difficult. There is generally a remittent type of fever, sometimes repeated rigors, sweating, low delirium, stupor or coma, and extreme prostration. There is often a fine petechial eruption. Usually there is a cardiac murmur, the location of which depends upon the seat of the disease; it is most frequently the murmur of mitral regurgitation. It is sometimes faint, and may be absent. From the emboli there may result hemiplegia, rapid swelling of the spleen, bloody urine or pneumonia. The disease lasts from three weeks to three months, death being the almost invariable termination. The most characteristic

features of malignant endocarditis are the development of pyemic or typhoid symptoms with a petechial eruption, in a patient who has previously had valvular disease. Blood cultures in most cases give positive results, though not always early in the disease.

The treatment is symptomatic. The use of vaccines has not met expectations; in the most acute cases no benefit has followed their administration, and even in the more prolonged types it is very doubtful if they have any value.

MYOCARDITIS

Disease of the muscular wall of the heart is rare in children, and of comparatively little importance, except in connection with acute endo- and pericarditis and the acute infectious diseases. It is almost invariably secondary to some infectious process. Aside from the rheumatic conditions already considered the diseases which furnish most of the cases are scarlet fever and diphtheria. The most important local cause is pericarditis with adhesions.

Lesions.—In extra-uterine life, myocarditis as a rule affects chiefly the wall of the left ventricle, the papillary muscles, or the septum, but the entire organ is involved. The heart is of a grayish or yellowish-red, sometimes mottled color, very soft, friable, and flabby, and there is frequently dilatation of the cavities.

Two varieties of myocarditis are described, the parenchymatous and the interstitial. In the parenchymatous form there is a degeneration of the muscle fiber which, according to Romberg, is most frequently albuminous, next fatty, and least frequently hyaline. There is a loss of the transverse striations, and there may be complete disintegration of the fibers. This process may be circumscribed, but it is usually diffuse. In the interstitial form the lesion usually occurs in small, circumscribed areas. There is an infiltration of round cells, chiefly mononuclear, between the muscular fibers of the heart. The process, when acute, may result in absorption or in the production of small abscesses. In chronic cases it may lead to the formation of areas of dense connective tissue resembling cicatrices, in the heart wall. Either the interstitial or the parenchymatous form may occur alone, but in most of the acute cases they are combined. In addition, there is usually some degree of mural endocarditis and inflammation of the pericardium next to the heart wall. Dilatation frequently follows. Cardiac aneurism and even rupture have been known to occur in a child of six years (Hadden's case).

Symptoms.—In many cases in which advanced lesions have been found at autopsy there have been no symptoms appreciated during life. Careful examination of the heart, however, will usually show an altera-

tion in the first cardiac sound, the muscular quality diminishing and the valvular quality increasing. This may go on even to a total disappearance of the muscular quality and only a flapping valvular sound may remain. The first and the second sounds are then almost alike. In such severe cases diastole is relatively short and the rhythm is much like that of fetal life. A systolic murmur due to dilatation of the auriculoventricular ring, or to imperfect action of the papillary muscles, may be heard at the apex. The heart is usually slightly dilated, but may be excessively so. Its action is generally increased in rapidity and may be irregular; a slow heart, 50 to 70, with feeble, valvular sounds is less frequent but very characteristic. The apex beat is diminished in intensity and the pulse is soft and weak. The blood pressure is low, frequently 60 mm. or even less. Other symptoms may be present that are dependent upon feeble heart action—pallor, dyspnea, slight cyanosis, and attacks of syncope. Less frequently there may be dropsy of the feet or the serous cavities, and scanty urine sometimes containing albumin. Death may occur suddenly from cardiac paralysis or gradually from circulatory failure. Recovery may take place after alarming symptoms have been present, these slowly abating. It may be many weeks before the normal cardiac sounds are heard.

Treatment.—This is mainly symptomatic. After severe attacks of those infectious diseases in which myocarditis is liable to occur, and at any time when it is suspected, patients should be kept recumbent for several weeks, and special care exercised to prevent any sudden exertion, as death has resulted from so slight a thing as suddenly sitting up in bed. Once definite symptoms have developed, absolute rest is imperative. Much more is to be expected from complete rest than from drugs, which as often employed may do positive harm. Digitalis should be used with caution, and never in large doses. In some cases with symptoms indicating imminent heart failure rather striking benefit has followed the use of morphin hypodermically.

ACCIDENTAL MURMURS

Under this term are included those murmurs that do not depend upon organic change in the heart or are not functional in the sense that actual regurgitation takes place through a dilated orifice.

In early life such murmurs are exceedingly common. Our own observations confirm those published by Hamill and others, that murmurs may be heard on careful examination in nearly fifty per cent of all children. Their existence is often a cause of much needless anxiety and of many unnecessary restrictions of a child's activities. They are

almost invariably systolic in time; they are usually of moderate intensity, soft and blowing in character, and are not transmitted. They are unaccompanied by changes in the size of the heart or by symptoms referable to its function. They are apt to be inconstant in occurrence, and often change in character or disappear altogether by changing the posture of the child.

The exact method of their production is still a matter of doubt. In certain instances they are apparently dependent upon changes in the blood occurring in anemia. In several of our patients, infants with grave anemia, quite loud murmurs have disappeared after transfusion. In other cases there can be no doubt that the murmurs are produced in the lungs, air being forced through the bronchi by the movements of the contracting heart. The term cardiopulmonary is applicable to murmurs of this origin. This murmur is not loud, is never heard to the right of the sternum and disappears when the breath is held. It is usually loudest over the pulmonary artery, intensified by excitement or exertion, and often disappears when changing from a standing to a supine position.

Atonic murmurs are probably due to lack of tone in the cardiac muscle leading to a real but temporary insufficiency, usually at the mitral orifice. These murmurs correspond in most cases to a slight mitral regurgitant murmur. They are heard in the course of a number of acute febrile diseases—notably scarlet and typhoid fevers; also in many pale, delicate, nervous children, especially between the ages of eight and fourteen years.

Anemic murmurs are usually accompanied by a venous hum, but not by an accentuated pulmonic second sound. Other causes of accidental murmurs such as a functional stenosis of the pulmonary artery and infundibulum, functional mitral insufficiency and eddy currents within the ventricles are not so well substantiated by clinical or experimental proof.

Probably the most frequent of all accidental murmurs is the soft systolic murmur which is heard over the body of the heart near the left border of the sternum at about the nipple level; it is increased by placing the child on his back and in many patients is heard only in this position. This murmur is usually intensified by overaction of the heart whether due to excitement, exertion or fever. It is accompanied by no symptoms referable to the heart or circulation and it may be met with in children who are in perfect health. This murmur is more often heard in infants and young children, but may be present for many years. It is often confused with murmurs due to cardiac malformation, but it is not loud as are they, and is heard only over a localized area.

The differentiation from murmurs due to organic cardiac disease

may be difficult and only possible by continuous observation for some time; but in any infant or child one should hesitate to make a diagnosis of congenital or acquired organic disease from the mere presence of a soft systolic murmur.

FUNCTIONAL DISTURBANCES OF THE HEART

Disturbances of the heart's action unconnected with organic disease are quite common in children, especially from the seventh or eighth year up to puberty. Common causes are disorders of digestion, the excessive use of tea, coffee or tobacco, especially in the form of cigarette smoking, anemia, over pressure in schools or other conditions leading to nervous exhaustion. The exciting cause is sometimes a great emotional disturbance such as fright or excitement, or it may follow any serious acute illness. As a rule there are more subjective symptoms with functional than with organic disease unless the latter is advanced. Functional disturbance may take the form of attacks of palpitation, tachycardia, bradycardia or arrhythmia.

With attacks of *palpitation* there may be a sense of oppression in the precordium; there may be some dyspnea or even orthopnea; the pulse is generally rapid, often slightly irregular. There is strong pulsation of the carotids and sometimes headache or vertigo. There may be cold extremities and general perspiration. The duration of the attack is from a few minutes to several hours. The treatment is that of the general nervous condition upon which the palpitation depends.

Tachycardia (rapid heart) occurs in certain susceptible children from slight cause, most frequently when the general health is below par, in conditions of anemia, and in nervous children—particularly girls about the time of puberty.

In the same patient the symptoms may occur at intervals for years. The pulse at such times may be from 120 to 160 per minute or even more rapid than this. The condition may persist for days or weeks at a time, then subsiding, but the symptoms recurring at variable intervals. In some children a very rapid pulse must be considered an idiosyncrasy.

In a patient with an attack of tachycardia position makes little difference with the heart rate. Sometimes it is even more rapid when the child is recumbent. It is, however, almost invariably much lower during sleep and at such time may even be quite normal. The rhythm of the heart is not disturbed. It is important not to confound this condition with Graves' disease. The treatment is to be addressed to the nervous condition present, to which as a rule the cardiac symptom is secondary.

In certain children there is seen a more rare but severe form of this condition known as *paroxysmal tachycardia.* It has been observed in children as young as three years. There develops abruptly and without assignable cause an extraordinary heart rate which may be 200 to 250 per minute. Such attacks may last from a few minutes to several weeks, both beginning and ending abruptly. After an attack the pulse may for a time be abnormally slow. In prolonged cases some cardiac dilatation often occurs, and a systolic murmur may develop. Serious consequences may follow, such as swelling of the liver, dropsy, etc. The cause and mechanism of such an abnormal cardiac stimulus are as yet obscure. Curiously, attacks may often be cut short by vomiting. The indications for treatment are nervous sedatives, complete rest and prolonged treatment with digitalis in full doses.

Bradycardia (slow heart) is a much less frequent condition than tachycardia. It is seen in a variety of pathological conditions not involving the heart, such as jaundice, certain poisons, etc. Its persistence in young children is always a suspicious symptom suggesting cerebral disease, though in some children an abnormally slow pulse is an idiosyncrasy. Existing by itself, no importance is to be attached to it as a sign of cardiac disease.

Arrhythmia.—Like all other nervous adjustments the heart-regulating mechanism does not work with the same uniformity in children as in older subjects. In consequence of this, disturbances of cardiac rhythm are more frequently seen and occur from slighter causes in early life than later. Cardiac irregularity is exceedingly common in children, and is often seen in those who are apparently in excellent health. Of 321 unselected children studied by Friberger only 37 per cent had a fairly regular pulse, while over 12 per cent had a very irregular pulse. A certain degree of cardiac irregularity up to the time of puberty is so common that it must almost be regarded as the rule. Only exceptionally does it indicate disease of the heart; particularly is this the case when it occurs with slow heart action. The higher forms of irregularity are usually seen in younger children. In general, it is more often observed in girls, but is not affected by general development nor by cardiac weakness. It is slightly more frequent in nervous subjects.

Sinus arrhythmia, reflex arrhythmia or, as it is sometimes called, vagus irregularity, is the characteristic type of arrhythmia in early life. The alteration in cardiac rhythm is brought about by stimuli which arise outside of the heart and reach it by one of the cardiac nerves. The point of origin of the impulse is probably the sinus region. Reflex stimuli are constantly reaching the heart. The regulation of beats is usually so perfect, however, that they do not influence its rate. If the resistance to outside stimuli is less than normal these stimuli may reflexly affect

the rate. It has been shown by Einthoven and others that stimuli pass up the vagus nerves with each respiration. The nervous control of the various functions of the body is imperfectly developed in children and sinus arrhythmia is in them a frequent finding. It bears, in many instances, a close relationship to the respiration. The irregularity is generally but not always shown in the pulse at the wrist. It is best determined by auscultation. The irregularity is often rhythmical, varying with the respiration. With inspiration, the action of the heart becomes rapid and with expiration slow. Other varieties are, irregular pauses or a sudden retardation of frequency at irregular intervals.

There are no subjective symptoms and the patient is seldom conscious of the condition. The arrhythmia is present during sleep, often most marked at that time and associated with irregular respiration. A diagnosis of sinus arrhythmia is made chiefly by its association with respiration; it disappears with rapid respiration or when the breath is held. It is also characteristic of this condition that it is seen only with a slow heart action, disappearing at once when the heart's action from any cause becomes rapid.

This form of irregularity is not in itself significant. It is not a symptom of cardiac disease, nor does it affect the patient's health or his development. It may safely be ignored altogether. In certain children, however, it may be a constant phenomenon and may persist for many years. Sinus arrhythmia may be met with as a temporary condition after any severe acute illness; it may be seen in children of the neurasthenic type associated with other evidences of nervous instability. In many cases, even of the most pronounced type, no adequate cause can be discovered.

Another type of irregularity is due to the production of *extra systoles*. These are occasional, irregular beats caused by single abnormal stimuli, arising within the heart, either in the auricle or ventricle. Extra systoles are seldom observed in young children,—more frequently in those over eight or ten years old. The extra systoles may be followed by a long pause the following normal beat being omitted, or two beats may occur very close together. Extra systoles are usually not sufficiently forcible to open the aortic valves. For this reason they cannot often be felt at the wrist but may, in thin subjects, be recognized by palpation and they can be determined by auscultation or tracings. There may be no subjective symptoms or there may be complaint of precordial anxiety and unrest when the extra systoles occur. This form of irregularity is seldom a constant phenomenon, but with susceptible persons it comes and goes from apparently slight causes. It is most marked when the pulse is slow and may disappear when it becomes rapid, sometimes also on assuming a recumbent position.

Extra systoles are usually not associated with other signs or symptoms of cardiac disease in children and under such circumstances may be practically ignored. The condition is not a serious one. Its causes may be disorders of the stomach, an extremely sensitive nervous system, or convalescence from an acute febrile disease, especially pneumonia. The treatment should be addressed to the general condition, not to the heart. Exercise need not be restricted.

DISEASES OF THE BLOOD-VESSELS

Coarctation of the Arch of the Aorta.—This is a rare congenital lesion in which there is a partial or complete occlusion of the aorta at or near the junction with the ductus arteriosus. The ductus may remain patent and the systemic circulation be carried on almost exclusively by means of the blood which passes by way of the ductus from the pulmonary artery to the aorta below the constriction. Coarctation of the arch may be the only lesion or there may be associated lesions with death in the first few months. When the stenosis is beyond the opening of the ductus arteriosus a very complete collateral circulation develops chiefly by means of the superior intercostals and mammary arteries above, and the aortic intercostals and superficial and deep epigastric arteries below. In consequence of this there may be no symptoms of the condition. Instances are on record where persons with this lesion have lived to advanced age, but often they are stunted in growth, poorly nourished, and complain of dyspnea.

The physical signs are at times very characteristic. The collateral circulation may show superficially over the thorax and upper abdomen. A marked disproportion in intensity between the radial pulse and the femoral pulse may be present. There is frequently marked pulsation and a thrill in the suprasternal notch owing to dilatation of the arch of the aorta. A loud systolic murmur may be heard in the second or third spaces on the left side, well out from the sternum. Death may be due to intercurrent disease, to failing circulation and sometimes to rupture of the heart or of the arch of the aorta.

Abnormally Small Arteries (*Arterial hypoplasia*).—This condition is not a common one. The only thing which is abnormal in the circulatory system may be that the aorta, and sometimes all the large vessels are only two-thirds or three-fourths their usual caliber, or even less. This may interfere seriously with the growth and development of the body, especially of the genital organs, although this result is not a constant one. The condition is found occasionally in cases of chlorosis. There is sometimes associated a certain amount of hypertrophy of the heart. The other symptoms are anemia, and sometimes an imperfect

development of the body. A positive diagnosis during life is impossible.

Aneurism and Atheroma.—In early life chronic disease of the blood-vessels is exceedingly rare, yet a sufficient number of observations have been recorded to show that even young children are not exempt from this form of disease. Sanné records the youngest case, which occurred in a fetus born at about the eighth month, in whose body there was found a large aneurism of the abdominal aorta just below the origin of the renal arteries. Le Boutillier has collected seven cases of thoracic aneurism in children under ten years; the arch of the aorta was the usual seat.

Probably the most important etiological factor, as in adult life, is syphilis, but in only a few of the cases reported was the evidence of syphilis conclusive. In two cases there was general tuberculosis. In at least two cases whooping-cough appeared to act as a contributing cause. Aneurism may also be due to some local condition, such as an erosion from a bony growth, an abscess in the neighborhood, or to embolism. The symptoms and course of aneurism in young children do not differ essentially from those of the disease as seen in adults.

In addition to the cases of aneurism referred to above, we have found reports of seven cases of atheroma in very young subjects. In Sanné's case the patient was but two years old, and patches of atheromatous degeneration were found in several places in the aorta. In Hawkins' case, eleven years old, there was found extensive atheromatous disease of the aorta, subclavian and carotid arteries. In Filatow's case, atheromatous degeneration affected the arteries at the base of the brain, causing death from cerebral hemorrhage. It is interesting to note that in this patient, who was only eleven years old, there was also present chronic diffuse nephritis with contracted kidneys. A similar condition of the kidneys and arteries was observed by Dickinson in a girl of six years. We have seen extensive arterio-sclerosis the result of hereditary syphilis in a boy of five. Death occurred from hemorrhage into the lateral ventricle.

Embolism and Thrombosis.—Embolism is very rare in early life, even with acute endocarditis. The emboli are usually swept into the circulation from vegetations upon the valves of the heart. The symptoms which they produce will depend upon the nature of the emboli and the vessels occluded by them. If they lodge in the brain they may cause paralysis or convulsions; if in the spleen, pain and swelling of this organ; if in the kidneys, pain, tenderness, and sometimes hematuria; if in the lungs, cough, sometimes accompanied by hemoptysis and occasionally by a sharp thoracic pain. If the emboli are infectious, they may give rise to abscesses. The pathological results following embolism are similar to those which are seen in adults.

The most frequent form of thrombosis, that occurring in the sinuses of the brain, is discussed in connection with Diseases of the Nervous

System. Cardiac thrombi, especially of the right side of the heart, are not infrequently found in patients dying from cardiac disease, pneumonia, and occasionally also from other acute inflammatory processes and acute infectious diseases, particularly diphtheria. These thrombi are in most cases produced during the last few hours of life, or just at the time of death, and are of no clinical importance. They frequently extend from the heart into the large blood-vessels, particularly the pulmonary artery. Thrombosis occasionally occurs in any of the large vascular trunks in childhood as well as in adult life.

Thrombosis of the Internal Jugular Vein.—M. Pasteur has reported a case in a child two and a half years old, in which the middle of the vein was filled with an organized thrombus, and the lower portion obliterated and reduced to a fibrous cord. The symptoms were swelling, edema, and cyanosis of the face, and dilatation of the facial vein, but not of the external jugular. There were clubbing of the fingers and edema of the feet, but not of the arms. The heart was dilated and hypertrophied, but there was no valvular disease. The symptoms had existed since an attack of pneumonia, eighteen months before death.

Thrombosis of the Vena Cava.—Quite a number of cases are on record where this has occurred as the result of pressure from large abdominal tumors; it has followed new growths of the kidney and large masses of tuberculous lymph nodes. Neurutter and Salmon have recorded a case of thrombosis, apparently of marantic origin, in a child seven years old. The thrombus filled the vena cava, and extended to the origin of the hepatic veins and into both femorals. Death occurred from tuberculosis. In Scudder's case (seventeen years old) there was apparently obliteration (probably congenital) of the inferior vena cava; there was an extensive varicose condition of all the abdominal veins. The symptoms of thrombosis of the vena cava are swelling and edema of the feet—sometimes of the abdominal walls and the groin—and very great dilatation of the superficial abdominal veins.

Thrombosis of the Aorta.—A case has been reported by Leopold in a newly-born child who was delivered by version. The thrombus was of recent origin, and filled the lower aorta, extending into the femoral artery. A case of thrombosis of the aorta occurring in a girl of thirteen years has been reported by Wallis. The aorta was very narrow, and probably the seat of syphilitic disease. The thrombus extended from the origin of the renal arteries to the celiac axis.

Thrombosis in Infectious Diseases.—There is occasionally seen in typhoid fever, but more frequently in diphtheria, thrombosis of some of the large venous trunks, usually of one of the lower extremities. The symptoms are pain, localized swelling, and partial paralysis. If the artery is affected, there may be gangrene.

SECTION VI

DISEASES OF THE UROGENITAL SYSTEM

CHAPTER I

THE URINE IN INFANCY AND CHILDHOOD

WHILE a study of the urine is of much less importance in early life than of the symptoms referable either to the digestive or respiratory system, it is deserving of much more attention than it has generally received. In infancy especially it is attended with some difficulty, owing to the fact that it is by no means an easy matter to secure readily specimens for examination.

Methods of Collecting Urine.—In male infants this may be done by placing the penis in the neck of a small bottle or test tube which lies between the thighs, and is secured in position by pieces of tape passing over the hips and beneath the perineum. The urine of female infants can sometimes be collected in a similar way by placing a small cup or a large-mouthed bottle over the vulva and holding it in place by the napkin or by pieces of adhesive plaster. A plan often successful is to put the infant upon a chamber after a long sleep. It should be done at the instant of waking or the child may be wakened for the purpose. When an infant has not voided for one or two hours the application of a cold hand or a cloth wrung out of ice water to the abdomen or the buttocks will usually cause emptying of the bladder. A small amount of urine, sufficient to test for albumin, may often be obtained by placing absorbent cotton over the vulva or penis. The most certain of all means, however, is catheterization, which, however, should not be resorted to unless absolutely necessary. A soft-rubber catheter, size 6 or 7, American scale (9 or 11 French), should be used for infants.

Daily Quantity.—This is relatively much larger in infants than in older children and in adults, on account of the large amount of water taken with the food. The quantity fluctuates widely from day to day, according to the amount of fluid food taken and the activity of the skin and bowels. The figures following are the averages obtained by combining the results of the investigations of Schabanowa, Cruse, Camerer, Pollak, Martin-Ruge, Berti, Schiff, and Herter.

Average Daily Quantity of Urine in Health

Age.	Grams.	Ounces.
First twenty-four hours	0 to 60	0 to 2
Second twenty-four hours	10 " 90	⅓ " 3
Three to six days	90 " 250	3 " 8
Seven days to two months	150 " 400	5 " 13
Two to six months	210 " 500	7 " 16
Six months to two years	250 " 600	8 " 20
Two to five years	500 " 800	16 " 26
Five to eight years	600 " 1,200	20 " 40
Eight to fourteen years	1,000 " 1,500	32 " 48

Frequency of Micturition.—This is greatest in young infants, and diminishes steadily as age advances. In infancy, during the waking hours, the urine is passed very frequently, often two or three times an hour, while during sleep it is retained from two to six hours. By the third year the urine may be held during sleep for eight or nine hours, and at other times for two or three hours. Such control of the sphincter of the bladder is often obtained at two years, and sometimes even at an earlier period. From slight nervous disturbances or minor ailments of any kind, this control is impaired, and the urine may be passed by children of four or five years with the frequency seen in infants.

Physical Character and Composition.—The urine of the newly born is usually highly colored. During later infancy it is pale and frequently turbid, even when practically normal, owing to the presence of mucus; this turbidity often no amount of filtration will entirely remove. Less frequently the turbidity depends upon urates. The urine of the first few days of life often shows a deposit of urates or uric acid in the form of a pink or reddish-yellow stain upon the napkin. The reaction of the urine at this time is usually strongly acid, but throughout the rest of infancy it is faintly acid or neutral.

The specific gravity is higher during the first two days than at any time in infancy on account of the scanty supply of fluid taken; it is usually lowest from the third to the sixth day, but from this time it rises steadily until puberty is reached. The specific gravity varies with the quantity. From the writers already referred to, the following figures are taken:

	Specific gravity.
First to third day	1.010 to 1.012
Fourth to tenth day	1.004 " 1.008
Tenth day to sixth month	1.004 " 1.010
Six months to two years	1.006 " 1.012
Two to eight years	1.008 " 1.016
Eight to fourteen years	1.012 " 1.020

Microscopically, the urine of the newly born shows the presence of many squamous epithelial cells, mucus, granular matter, crystals of uric

acid and amorphous or crystalline urates and amorphous bilirubin crystals which are insoluble in urine not containing bile acids. It is not uncommon to find hyaline and even granular casts. Martin-Ruge found hyaline casts in the urine of fourteen out of twenty-four healthy nursing infants examined during the first week. Granular casts were much less frequent.

The inorganic salts (phosphates, chlorids, sulphates) are all present in the urine of the newly born, but in relatively small quantities, increasing as age advances. The coloring matters are also less abundant.

Albumin is often present in the urine during the first days, but usually in small amount. Cruse found it twenty-eight times in ninety observations upon healthy infants; usually the quantity was small, amounting to traces only, but in two cases it was quite large upon the second day. These observations are confirmed by the investigations of Martin-Ruge, and also of Pollak.

Sugar is frequently found in the urine of healthy infants during the first two months. It may be made to appear in the urine of healthy infants by simply increasing the quantity ingested. The different sugars vary as regards the amount which can be taken before it is thus eliminated. According to Grósz, lactose appears if the quantity is increased to three or four grams per kilo. of body weight; glucose, only when five grams, and maltose, not until seven and seven-tenths grams per kilo. are given.

LORDOTIC, ORTHOSTATIC OR CYCLIC ALBUMINURIA

This condition, although uncommon in young children, is frequently seen between the ages of six and fifteen years. It is much more common in males than in females. A recurrent but benign albuminuria in children has been recognized for many years and has been referred to numerous causes such as cold bathing, fatigue following muscular exertion, dyspeptic conditions or a diet rich in nitrogenous food. It is doubtful if any of these are of etiological importance, for the condition persists when their influence is entirely eliminated. The most important factor is undoubtedly a mechanical one. The albuminuria is due to the upright position. When this is not assumed there is no albuminuria, or the merest trace. There can also be no doubt that lordosis plays a very important part, if not the most important part. The majority of cases occur in children with a considerable degree of lordosis. How this acts, by producing congestion of the kidney by pressure on the vessels at the pelvis or otherwise is not quite clear.

Symptoms.—Some of the patients exhibiting orthostatic albuminuria are well nourished and have no other signs of disease; the majority, however, while they may be considered healthy, are not vigorous. They

are usually anemic and rather poorly nourished. They suffer from gastro-intestinal symptoms of which constipation is a frequent one and often have headaches and various neuroses. Cardiac palpitation and vasomotor symptoms are common. The trunk is usually long in proportion to the height and a degree of lordosis is the rule. The abdomen is usually prominent. Sometimes symptoms of angioneurotic edema have directed attention to the urine. Except for these there are no symptoms that would direct attention to the genito-urinary tract and the condition is usually discovered in an attempt to explain the poor general condition of the patient.

The urine is usually clear and that which is secreted while the child is lying down presents nothing abnormal. Shortly after assuming the upright position albumin appears in greater or less quantity. This is serum albumin plus a substance which is precipitated by acetic acid in the cold. Recent studies have shown that this is probably chondroitin sulphuric acid united with serum albumin. The amount of albumin present may vary from a trace to 50 per cent by volume or even more. The substance precipitated by acetic acid is never in large quantity. It causes clouding of the urine or an appreciable precipitate but no more. It is sometimes found alone and always when serum albumin is present. The assumption of a markedly lordotic position increases greatly the amount of albuminuria. Infrequently casts may be present; they are usually hyaline casts and few in number. They may occasionally be associated with a temporary glycosuria but the kidney does not show a greater permeability to other substances used for renal tests. Evidences of nephritis, high arterial tension, cardiac hypertrophy, etc., are absent.

Orthostatic albuminuria is not a dangerous condition, nor does it interfere with health. It disappears usually at or shortly after puberty. Occasionally it may persist well on into adult life.

It is important that orthostatic albuminuria should not be confused with nephritis. Children are not infrequently confined to bed for a long time and placed upon a rigid diet with the mistaken idea that nephritis is present. If, after repeated examinations, it is found that albumin is present only when the upright or lordotic position is assumed, if a substance precipitable by acetic acid in the cold is present and other evidences of nephritis absent, the diagnosis of orthostatic albuminuria may properly be made.

Treatment.—If lordosis is present, as is usually the case, much can be done to prevent the albuminuria. The abdominal muscles should be strengthened by appropriate gymnastic exercises. The children should practice assuming and maintaining a proper position in standing and sitting. Exercise is of value but prolonged standing should be avoided. If the lordosis persists in spite of these measures a light form of appara-

tus may be worn which prevents the lordotic position but does not interfere with exercise. Associated conditions such as anemia, constipation, and the various neuroses should receive their appropriate treatment.

HEMATURIA

Hematuria is characterized by the presence of red blood-cells in the urine, and is to be distinguished from hemoglobinuria where only blood pigment is present.

Hematuria may result from local or general causes. In infancy it may be due to new growths of the kidney. Such hemorrhages, though rare, may be abundant, and may be seen early. Hematuria may occur also as a symptom of acute nephritis, especially that complicating scarlet fever, or it may result from the irritation of a calculus in the kidney, the ureter, or the bladder. In rare instances its cause is a new growth of the bladder, and it may be due to traumatism. It may sometimes be produced by the irritation of a highly concentrated urine, owing to the fact that too little fluid is taken. We once saw a marked example of this in an infant eight months old, when no other explanation could be found. Hematuria is occasionally seen following uric-acid infarctions in the newly born. It may also occur at this age as one of the symptoms of sepsis. Among the general causes the most important are, the hemorrhagic disease of the newly born; the blood dyscrasiae, such as scurvy, purpura, and hemophilia, and infectious diseases, particularly typhoid, scarlet fever, influenza, and malaria. In most of these cases the amount of blood passed is small. When it is large it may appear in the urine as clear blood, or as clots, or it may impart simply a reddish or smoky color to the urine. The color, however, is not so reliable as a microscopical examination.

Large hemorrhages are much more likely to come from the kidneys than from the bladder. The presence of blood casts from the renal tubules, or larger ones from the ureter, are conclusive evidence of the renal origin of the hemorrhage.

The treatment of hematuria should be directed to the cause upon which it depends. In infancy scurvy especially should not be overlooked.

HEMOGLOBINURIA

In this condition blood pigment appears in the urine in large quantity, but red blood-cells are very few in number or are absent altogether. In severe cases the urine may be almost black. There is commonly a

small amount of albumin. This condition may be recognized by the appearance of granules of pigment under the microscope, or by Heller's test; the most conclusive means of diagnosis, however, is by the spectroscope.

Epidemic hemoglobinuria (Winckel's disease) has already been described in the chapter on Diseases of the Newly Born. Hemoglobinuria may be due to certain poisons, as carbolic acid or chlorate of potash, or to certain infectious diseases, as scarlet fever, typhoid fever, malaria, syphilis, or erysipelas.

Paroxysmal hemoglobinuria occurs in childhood, although it is an exceedingly rare condition. In most of the recorded cases there has been a history of syphilis and the Wassermann reaction has been positive. It is now regarded as a syphilitic affection. Paroxysms may be excited by exposure to cold, by chilling the surface of the body or by merely immersing the hands in cold water. Vigorous antiluetic treatment is indicated. It is not yet clear that it is always entirely successful; it may, however, greatly improve the condition. For further description textbooks on general medicine should be consulted.

PYURIA

Pus in the urine may exist as an acute or a chronic condition. In either case, in a child, it is much more likely to come from the pelvis of the kidney than from any other source. It may, however, come from any part of the genito-urinary tract—the kidney or its pelvis, the ureters, the bladder, the urethra, or the vagina. Sometimes it comes from an outside source, as when an abscess from perinephritis, appendicitis, or caries of the spine opens into the urinary tract.

Coming from the pelvis of the kidney, pus may indicate, if the condition is an acute one, pyelitis, pyelonephritis, or pyonephrosis; if it is chronic, it may point to renal tuberculosis or calculus. The amount of pus in any of these conditions may be quite large. The urine is turbid and usually acid in reaction. It contains many epithelial cells of the transitional variety. A urine containing much pus is always albuminous. It is rare that pus comes from the ureters except in connection with congenital malformations or the impaction of calculi. Pus from the bladder is not usually in large quantity, and may be mixed with mucus. The urine may be alkaline or acid in reaction; there may be associated the symptoms of vesical irritation or of cystitis. Pus from the lower genital tract is rare in children, and its causes may often be recognized by a local examination. When the cause of pyuria is the opening of an abscess into the urinary tract there is generally a sudden appear-

ance of pus in large amount. The pyuria is usually in such cases of short duration, possibly only a few days, and it may disappear quite rapidly.

The nature of the infection can be determined only by cultures made from a catheterized specimen. This information is of considerable aid both in diagnosis and prognosis.

The treatment of pyuria depends altogether upon its cause. Improvement in the symptoms sometimes follows the use of hexamethylenamin, which may be given in doses of from five to ten grains three times a day to a child of five years.

ANURIA

By this term is meant an arrest of the urinary secretion. To that form which occurs in the course of renal disease the term "suppression" is generally applied. Anuria is to be carefully distinguished from retention, from the scanty secretion which occurs whenever food is refused or withheld on account of illness, and also from that which accompanies acute diarrhea, with large, watery discharges. Anuria is sometimes seen in the newly born, where it depends upon some malformation of the genital tract; or, more frequently, upon uric-acid infarctions in the kidneys. The first urine passed after such an attack is very often highly acid, and may contain an abundance of uric-acid crystals and larger masses visible to the naked eye. Other cases admit of no such explanation. For the time, the secretion appears to be completely arrested, as the bladder, both by palpation and catheterization, is found to be empty. This condition is very uncommon in infancy, and it may continue for from twelve to thirty-six hours. So long as infants appear to be perfectly normal in every other respect, the suspension of the urinary secretion even for twenty-four hours need excite no anxiety.

The treatment consists in the administration of the acetate or citrate of potash, and plenty of water. To a newly-born infant one grain of the citrate of potash may be given every hour or two, in water, until the urinary secretion is established, which will usually be in six or eight hours. If the urine is very highly acid and stains the napkins, the potash should be continued for several days. Hot fomentations over the kidneys may be used.

DIABETES INSIPIDUS (POLYURIA)

This is a chronic disease characterized by the excretion of a very large amount of pale urine of low specific gravity. It is invariably accompanied by polydipsia. The disease is a rare one in children.

Etiology.—Of eighty-five cases collected by Strauss, twenty-one were in children under ten years of age and nine under five years. In Roberts's collection of seventy cases, the disease began in twenty-two children before ten years, and in seven during infancy. In some cases it begins soon after birth. Males are more frequently affected than females, and in certain cases heredity is an important factor. Weil has published a remarkable example of the disease existing in many members of a single family. Falls or blows upon the head, concussion of the brain, tumors of the brain, and chronic hydrocephalus, all have been found associated with diabetes insipidus. It sometimes has followed the acute infectious diseases; but in many cases no cause whatever can be found. The association of diabetes insipidus with lesions at the base of the brain has long been observed. More recently this symptom has been connected with lesions of the pituitary body. Since one of the most frequent lesions of the base is chronic syphilitic meningitis, syphilis must be considered a possible etiological factor. It is altogether probable that a number of quite distinct causes may produce diabetes insipidus.

Symptoms.—The quantity of urine is enormous, usually exceeding even that in diabetes mellitus. From five to twenty pints daily may be passed. The urine is pale, the specific gravity from 1.001 to 1.006, and it contains neither albumin nor glucose. In a few cases the presence of inosite (muscle sugar) has been found. Restricting the amount of fluid taken causes a very marked diminution in the amount of urine. The intense thirst leads patients to drink enormously of water and other fluids.

Nervous symptoms are usually present. There may be disturbed sleep from the frequent micturition, palpitation, flushing of face and other vasomotor disturbances, headache, restlessness, and neuralgia. There may be incontinence of urine. The bladder sometimes becomes enormously distended. In one of our cases it held forty-five ounces and reached above the umbilicus. The skin is pale and dry, and perspiration is scanty. The general health may not be much disturbed. In most cases, however, it is affected, and there may be the usual symptoms of malnutrition, and even neurasthenia. If it affects young children, their growth is generally retarded. The appetite usually remains quite good but anorexia may be marked. The temperature is at times slightly subnormal. The course of the disease is indefinite. It is very chronic, and may last for many years, death taking place from intercurrent affections.

Prognosis.—Occasionally a patient will recover spontaneously. Of the chronic cases in which the disease is well established very few are controlled. The prognosis is especially bad if there are marked disturbances of the digestive tract or organic brain disease.

Diagnosis.—This is easily made from the two marked symptoms, excessive thirst and polyuria. From diabetes mellitus it is easily distinguished by the lower specific gravity and the absence of sugar from the urine. In older children, chronic nephritis with contracted kidney may be confounded with it. Its occasional association with syphilis should be remembered and a Wassermann test made as a possible basis of treatment.

Treatment.—Fluids should be moderately restricted. It is a serious mistake to reduce the quantity of fluids too much, since the drinking is not the cause of the diuresis. The diet should be simple and nutritious. The general treatment should be directed to the condition of malnutrition. The clothing should be warm, and a moderate amount of exercise should be allowed. Drugs, in most cases, are of little use. Bromids and belladonna continued for many months are claimed to be of value. Codein too is said at times to cause decided improvement. It is doubtful if the prospect of cure justifies its use for a prolonged time. Treatment must be continued for many months to be of any value.

CHAPTER II

DISEASES OF THE KIDNEYS

MALFORMATIONS AND MALPOSITIONS

Malformations of the kidney are not infrequent. In seven hundred and twenty-six consecutive autopsies at the New York Infant Asylum malformations of the kidney or ureters were met with in seventeen cases. This does not represent the actual frequency with which they occur, for in about half that number of autopsies in two other institutions only a single example was seen. Adding to the cases mentioned two others seen elsewhere, there are twenty cases of renal malformation of which we have notes, classed as follows:

Fusion of the kidneys, or horseshoe kidney	4 cases.
Supernumerary ureters	4 "
Hydronephrosis (alone)	8 "
Congenital cystic kidney (alone)	2 "
Hydronephrosis and cystic kidney	1 case.
Single kidney	1 "

In all malformations the left kidney is much more frequently affected than the right, the proportion being nearly two to one. Malformations

are more often seen in males than in females. Only two of these conditions are of clinical importance—viz., cystic degeneration and hydronephrosis.

Cystic Kidneys.—Two varieties of this malformation are met with. In one the cysts are few in number and large; in the other they are very numerous and small. When the cysts are large the renal tumor may fill the abdominal cavity, even interfering with the birth of the child. The condition is generally bilateral, and the patients die in early infancy. The more common form, that with small cysts, also affects both sides as a rule. The organ often is not enlarged, and it may even be smaller than normal. The surface of the kidney is studded with small cysts, which usually vary in size from a pin's head to that of a pea. The entire organ may consist of nothing but a mass of cysts, held together by loose connective tissue. In other cases the cysts are less numerous, and much renal tissue remains. The cysts are formed by the dilatation of the uriniferous tubules owing to occlusion, which occurs in the development of the kidney. The large cysts are recognized as abdominal tumors; the small ones usually give no symptoms during infancy and childhood and are found accidentally at autopsy in patients dying from other diseases. In either form uremic symptoms may develop if an insufficient quantity of functionating renal substance remains.

Hydronephrosis.—This renal lesion in a mild form is not very uncommon at autopsy when no physical signs or symptoms have been given during life. In more severe form it is associated with many of the malformations of the organ such as horseshoe kidney, cystic kidney, etc. It may affect one or both sides and be found in both males and females. Hydronephrosis is undoubtedly the result of some obstruction to the outflow of urine from the kidney, ureter or bladder, but this obstruction may be very difficult to demonstrate. Obvious causes for hydronephrosis are stones in the kidney, ureter or bladder and pressure upon the urinary tract by tumors.

The ureter is generally dilated to a diameter of from one fourth to one half inch and it may be so large as to be easily mistaken for the intestine. Usually the ureters appear much elongated and sacculated; the pelvis and the calices of the kidney may be slightly dilated or the greater part of the kidney may be destroyed, leaving only a series of communicating pockets surrounded by a thin cortex of renal tissue. After a time chronic nephritis usually develops. This may involve both kidneys, even though the hydronephrosis is unilateral.

If hydronephrosis is unilateral there may be no symptoms until the dilatation of the pelvis of the kidney has reached a sufficient size to form an abdominal tumor. In most of the cases in children this condition has been noted between the third and the eleventh years. This

tumor may be situated in the lumbar region, or it may fill the abdomen. It is cystic, and may be confounded with a dermoid cyst of the ovary. On aspiration a fluid is withdrawn which may be clear, or of a brownish color, and recognized as urine by the fact that it contains urates and urea. After aspiration the urine passed *per urethram* may be bloody. Aspiration affords only temporary relief, as the tumor quickly refills. The treatment is surgical. When the other kidney is normal nephrectomy often results in a permanent cure.

Double hydronephrosis occurs much more frequently in the male. In infants and young children it not infrequently causes a definite and characteristic group of symptoms. It may be found in infants a few weeks old or throughout childhood. Double hydronephrosis, however, is generally associated with, or results in, such changes in the kidneys that the patients die during infancy.

The cause of double hydronephrosis is usually to be found in the posterior urethra. While several abnormalities have been described the most common one is an exaggeration of the normal folds of mucous membrane that lead from the verumontanum to the wall of the urethra. These folds are sometimes greatly hypertrophied and so situated as to make a diaphragm across the urethra in which there is usually a small, slit-like opening. There is thus produced a great obstacle to the passage of urine. The changes produced in the bladder, ureters and kidney are very extensive. The bladder is much increased in thickness but is not dilated. The walls of the bladder may be as much as a quarter or a third of an inch in thickness. The ureters are greatly dilated and are often an inch or more in diameter. They are tortuous, their walls are thickened and thrown into folds. The kidneys are increased in size, due entirely to the hydronephrosis, for, as a result of this, the renal substance may be reduced to a minimum. They consist of a mass of dilated, communicating cystic spaces surrounded by a shell of renal tissue. The structure of cortex and medulla may be indistinguishable. Secondary infection not infrequently occurs, in which case the bladder, ureters and kidneys may contain pus and there may be abscesses in the substance of the kidney. An excellent example of this condition is shown in Fig. 84. The damage to the kidneys may be so great that the infant dies shortly after birth. When it is less, life may be prolonged for months or years.

The history is at times quite characteristic. There may have been difficulty in urination and dribbling of urine from birth or it may not have been noticed until the child was a year or two old, or perhaps even later. With each attempt to pass urine only a small quantity is expelled after much straining. Examination of the abdomen shows a firm, globular mass in the hypogastrium which remains even after urination. Leading up from this into the loin on each side there may often be felt

masses sometimes elongated, sometimes globular, which are the twisted tortuous ureters. The kidneys may or may not be felt. In the bilateral form of hydronephrosis the renal tumors are usually not large, as life would be impossible with the destruction of much renal substance on both sides. The masses may vary in size but the tumor formed by the bladder is the most constant one.

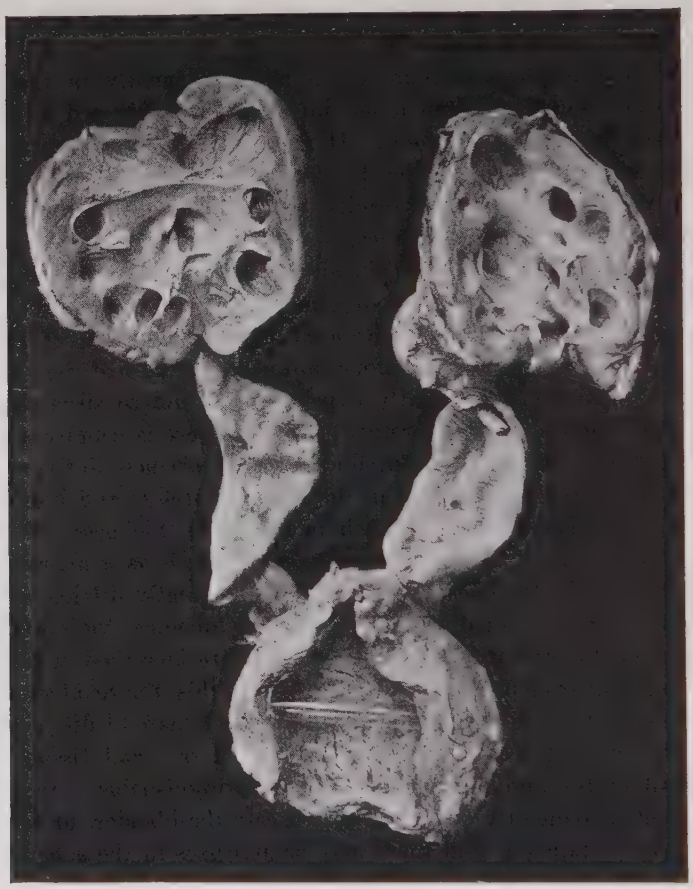


FIG. 84.—CONGENITAL HYDRONEPHROSIS, DILATED URETERS, AND HYPERTROPHIED BLADDER. (From a child one month old.)

Changes in the urine may not be present until the condition is far advanced. There may be all the symptoms of chronic diffuse nephritis or when infection of the genital tract occurs, there are added the symptoms of pyonephrosis. The course is usually progressive. More and more damage to the kidneys takes place until death results from uremia, from secondary infection, or from some intercurrent disease.

The treatment is surgical. The obstruction should be removed. If

this is done early before extensive changes in the kidneys have taken place life may be indefinitely prolonged. We have had two patients, three and four years of age, operated upon with very satisfactory results.

Movable Kidney.—This is a rare condition in young children. Comby has collected eighteen cases, of which sixteen were in girls and two in boys. Movable kidney was recognized before the tenth year in eight cases, and in two of these before the fourth month. It has been ascribed to too long a pedicle, which may be congenital; also to pressure from abdominal tumors and to injury. The most important symptoms are paroxysmal pain which may follow exertion, and a movable tumor. A twist in the ureter may produce hydronephrosis.

URIC-ACID INFARCTIONS

These consist in a deposit in the straight tubes of the kidneys of uric acid or of amorphous or crystalline urates; usually both kidneys are affected, and all the pyramids of each kidney. The infarctions appear to the naked eye as fine, brownish-yellow, fan-shaped striae. Associated with them there may be granular deposits of uric-acid salts in the pelvis of the kidney, and sometimes evidences of catarrhal inflammation of the pelvis, including even the presence of blood. This condition probably occurs, to some degree at least, in nearly all infants during the first ten days of life. It was formerly supposed that the discovery of these appearances was proof that an infant had breathed, and a certain medico-legal importance was therefore attached to them. This is now known not to be the case, as they are sometimes found in still-born infants.

The cause of this condition is the excretion of uric acid before there is sufficient water to dissolve it, so that the crystals are deposited in the tubes. Uric-acid infarctions are found chiefly in children dying before the end of the second week, although it is not uncommon to see them as late as the third or fourth or even the sixth month. In most of the cases, as the urinary secretion becomes more abundant, the deposits are washed out in the urine and appear as brownish-red or pink stains upon the napkins. Infarctions may give rise to a slight inflammation of the renal tubules, but very rarely to any serious lesion; sometimes they remain as deposits in the calices or the pelvis of the kidney or in the bladder, forming the nuclei of calculi. The symptoms to which they give rise are mainly scanty urination during the first week of life, and occasionally anuria for the first day or two. Sometimes there is evidence of severe pain; priapism may be present, and there is the stain upon the napkin already referred to. The treatment is to give water freely and some alkaline diuretic such as citrate of potash. One grain should be

given every two hours until the secretion is fully established; this in most cases will be within twenty-four hours.

CHRONIC CONGESTION OF THE KIDNEYS

This results from interference with the return circulation of the kidney, and may be caused by congenital malformation or valvular disease of the heart, chronic bronchopneumonia or chronic pleurisy; also by the pressure of any abdominal tumor upon the inferior vena cava or the renal veins.

The kidneys are generally enlarged, firmer than normal, and dark-colored. All the capillary vessels are swollen and distended with blood, and their walls are thickened. In addition to the symptoms of the primary disease, the amount of urine passed is usually scanty and of high specific gravity. Albumin and casts are generally present, but are not constant. The treatment should be directed toward the primary condition, and, in addition, an effort should be made to increase the amount of urine by alkaline diuretics, caffein, digitalis, and the sodium salicylate of theobromin.

ACUTE DEGENERATION OF THE KIDNEYS

In the succeeding pages devoted to the kidney Prudden's classification in the main has been followed.

In acute degeneration of the kidney the principal or only change is in the epithelium of the tubules. It is exceedingly common both in infancy and in childhood, being found to a greater or less degree in all autopsies upon patients dying of acute infectious diseases, but it is most marked in cases of scarlet fever, diphtheria, and acute pleuropneumonia. It may be found in any disease characterized by prolonged high temperature, and it is the explanation of the cases of so-called febrile albuminuria. The cause is in all probability direct irritation of the epithelium of the tubules by the toxins eliminated by the kidneys. It may also be induced by irritating drugs, such as cantharides or turpentine. By some writers these cases have been classed as examples of acute nephritis; hence the great discrepancy which exists in statements made as to the frequency of nephritis in the different infectious diseases.

The kidneys are usually slightly enlarged, softer, and paler than normal. On section the cortex may be somewhat thickened, and the straight tubules marked by yellowish-gray lines. It is the appearance commonly spoken of as cloudy swelling. The kidneys are seldom much congested. The microscope shows a granular degeneration of the epithe-

lium of the tubules, and when severe this may be accompanied by congestion and the exudation of serum.

Acute degeneration of the kidneys gives rise to no symptoms in addition to those of the original disease, except the appearance of a moderate amount of albumin in the urine, with a few hyaline, granular, or epithelial casts. It can not be said that such a condition adds much to the danger from the original disease. In cases that recover, the condition of the kidney becomes entirely normal. The development of the symptoms of degeneration of the kidneys in infectious diseases calls for no special treatment beyond a continuance of the fluid diet.

ACUTE DIFFUSE NEPHRITIS

(Acute Interstitial Nephritis; Acute Exudative Nephritis; Glomerulonephritis; Acute Bright's Disease.)

Etiology.—This variety of nephritis occurs apparently as a primary disease both in infants and in older children. Most such cases are undoubtedly of infectious origin, although the point of entrance of the infection it may be difficult or impossible to determine. Acute diffuse nephritis is very frequently secondary to the acute infectious diseases, especially to scarlet fever and diphtheria. It occasionally follows measles, varicella, empyema, typhoid fever, acute diarrheal diseases, pneumonia, meningitis, influenza, and malaria. It is the characteristic variety of secondary nephritis occurring in severe septic conditions. Some children exhibit a predisposition to this disease and develop acute nephritis with almost any infectious disease, however mild, which they contract. The exciting cause of the inflammation is in some cases the irritation from toxins; but usually there is in addition the entrance of pathogenic organisms carried by the circulation. Thus in post-scarlatinal nephritis, of which the one under consideration is the characteristic form, the cause is now generally admitted to be the toxins of the primary disease, to which in many cases is added infection by the streptococcus. While nephritis is more frequent after severe attacks of scarlet fever, it may occur after those which are very mild, even when patients have been kept in bed throughout the disease. The frequency of nephritis as a sequel of scarlet fever varies much in different epidemics; the average is from six to ten per cent. We have seen two cases of acute nephritis in infants, the apparent cause of which was the irritation of a highly concentrated urine. This was the result of the infants taking for a long time very little food and almost no water.

Lesions.—In severe cases the kidneys are usually enlarged, soft, and edematous. The capsule is non-adherent. The cortex is thickened, either

reddened or pale; frequently it is mottled with red, owing to the presence of small hemorrhages. There may be congestion of the entire organ; or the pyramids may seem unusually red by contrast with the pale and thickened cortex.

All the structures of the kidney—glomeruli, tubular epithelium, and interstitial tissue—are involved in the inflammatory process. The cells covering the glomerular tufts of capillaries are swollen and proliferated. They have frequently undergone fatty degeneration. The epithelial cells lining Bowman's capsule may undergo the same changes, but usually to a lesser degree. The space between the capsule and the tuft may contain exfoliated epithelium in considerable quantity, also cell-detritus, albuminous (granular) exudate, leucocytes, and red blood-cells. The tubular epithelium undergoes albuminous and fatty degeneration and may desquamate. Thus the tubules may contain epithelial fragments, serum, red blood-cells, leucocytes, and casts. The interstitial connective tissue is infiltrated with serum and in places with small round cells. In cases of longer duration a general increase of the connective tissue may take place, which is permanent.

When the glomerular changes are especially marked, as in acute nephritis following scarlet fever, the process is often spoken of as *glomerulonephritis*. If the degeneration of the tubular epithelium is extreme, as in severe cases of diphtheria dying shortly after the onset, the nephritis may be described as the *parenchymatous* or *degenerative* type. In the *hemorrhagic* form there are hemorrhages into the tubules, glomeruli, or interstitial tissue. In infants and young children the *exudative* type of acute diffuse nephritis is especially frequent. In this there is an exudative inflammation with large accumulations of leucocytes, serum, and red blood-cells in the glomeruli and tubules, the parenchyma and interstitial tissue sometimes being markedly and sometimes but slightly changed. Should the interstitial tissue suffer early and severely, the nephritis becomes of the *productive* or *interstitial* type. This form is most frequently seen with severe, protracted cases of scarlet fever and diphtheria, especially in older children. It sometimes occurs as an apparently independent process.

Symptoms.—1. *Primary Form in Infants.*—These cases are not common, and the symptoms are so obscure that they are often overlooked. A number of such cases have come under our observation. The inflammation in most of them was of the exudative type.

The onset in nearly every instance was abrupt, usually with high fever and vomiting, the temperature being in several cases over 104° F. Dropsy was exceptional; in most of these it was slight, and seen only toward the close of the disease. Fever was present in all cases. In those observed by us it was high and irregular in type, ranging from 101° to

105° F. The duration of the disease was from one to four weeks, the average being about two and a half weeks. Vomiting and diarrhea were noted in half the cases, but were rarely prominent, and marked either the onset of the attack, or were traceable to indigestion accompanying the fever; very rarely did they exist as symptoms of uremia. Anemia was a prominent symptom in nearly every case, and it was this which called attention in several instances to the renal condition. Nervous symptoms were usually prominent. In several patients there was dyspnea without pulmonary disease and without cyanosis, partly due perhaps to the marked anemia, but probably due chiefly to the development of acidosis. In nearly all cases there was marked restlessness or muscular twitchings, and in three there were convulsions. Dullness and apathy were present in the majority of the fatal cases, but deep coma was never seen. The urine was rarely scanty until near the close of the disease, and sometimes not even then. Suppression of urine was seldom seen. Albumin was frequently absent early in the attack, but was invariably present at a late period, although rarely in large amount. Casts were found in all cases that were carefully examined microscopically. They were not usually numerous, and were chiefly of the hyaline, granular, and epithelial varieties. No blood casts were seen. There were usually many pus cells and renal epithelial cells, together with red blood-cells in moderate numbers.

Of the thirty-four cases collected, including our own, twenty-five died and only nine recovered. Whether these figures represent the actual mortality of the disease it is difficult to say. No doubt there are many mild cases which are unrecognized. The severe ones, however, are quite uniformly fatal, chiefly on account of the tender age of the patients.

2. *Primary Form in Older Children.*—This also is a rare form of renal disease. The onset is usually less abrupt than in infants, the febrile symptoms are less marked, and the termination is less frequently fatal. Dropsy is rarely marked, and often is absent. The urine is only slightly diminished in quantity; the amount of albumin is small; casts are not numerous, and usually hyaline, epithelial, or granular; very rarely is there much blood present. Uremia is infrequent, and the prognosis is better than in infancy. The course may be very prolonged; but even when albuminuria has lasted several months recovery may be complete.

The interstitial type may begin abruptly with febrile symptoms, dropsy, headache, lumbar pains, scanty urine, and often with vomiting; or it may come on somewhat insidiously with few constitutional symptoms, but with dropsy and changes in the urine.

3. *Secondary Form.*—The secondary nephritis of acute infectious diseases may occur at the height of the febrile process or at a later period, and its severity is generally proportionate to the intensity of the infection.

The general symptoms of nephritis are often not marked, and dropsy is rare; so that unless the urine is examined the condition may be overlooked. The urinary changes are essentially the same as those already mentioned in the primary cases. Suppression of urine and the development of the symptoms of acute uremia are infrequent. While nephritis adds considerably to the danger from the primary disease, it is seldom itself the cause of death, although this is sometimes the case in scarlet fever and diphtheria.

The characteristic type of nephritis which follows scarlet fever most frequently develops during the third or fourth week of the disease. The onset may be gradual, dropsy being first noticed. Or it may begin abruptly without dropsy, but with headache, vomiting, scanty urine, fever, and even convulsions. The temperature generally ranges from 100° to 101.5° F., but in very severe attacks it may be 104° or 105° F. While dropsy is usually present, it may be slight or absent in severe and even in fatal cases. It is first seen in the face, next in the feet, legs, and scrotum: there may be general anasarca, with dropsy of the serous cavities of the body, the pleura, or the peritoneum, rarely the pericardium. As the disease progresses there is always a very marked degree of anemia.

The urine is, as a rule, greatly diminished in quantity, and may be suppressed. Albumin is invariably present, although not always at first; it is usually in large amount, often enough to render the urine solid upon boiling. The urine is of a dark, reddish-brown or smoky color, owing to the presence of red blood-cells or hemoglobin. The specific gravity may be low, even though the quantity is very small. Casts are present in great numbers, chiefly hyaline, granular, and epithelial casts from the straight tubes; not infrequently there are blood casts. Red blood-cells are present in great numbers; also many leucocytes, and renal epithelium.

The duration of the active symptoms in cases terminating in recovery is from one to three weeks. The temperature and dropsy gradually subside. Improvement in the urine is shown by an increase in quantity, by an increased elimination of urea, and by a diminution in the amount of blood, albumin, and the number of casts. A few casts may persist for several weeks, and a small amount of albumin for two or three months.

In the graver cases, when the onset is accompanied by high temperature, pain in the back and loins, and a rapid, full pulse of high tension, the urine is very scanty and is often suppressed. Then follow the symptoms of uremia. In children this is usually manifested by vomiting, great restlessness or apathy, and often by diarrhea. Hyperpnea is not infrequent and is usually evidence of acidosis. Less frequently there is headache, dimness of vision, stupor developing into coma, or convulsions. If the secretion of urine is re-established, the nervous symptoms

abate and the patient may recover. This has been known to occur after complete suppression has lasted thirty-six hours. Care should be taken not to mistake retention for suppression. If doubt exists, percussion of the bladder and the use of the catheter will quickly settle the question.

There are several complications for which the physician must constantly be on the lookout during attacks of acute nephritis; the most frequent are pneumonia, pleurisy, pericarditis, and endocarditis; more rarely there may be meningitis and edema of the glottis. It is from complications or acute uremia that death usually occurs.

Prognosis.—This is to be considered from two points of view: first, the danger to life during the acute stage of the disease, and, secondly, the danger of the development of chronic nephritis. The majority of patients survive the acute stage, and not infrequently even those recover who have presented grave symptoms of uremic poisoning. The quantity and specific gravity of the urine, the delayed elimination of phenolsulphonephthalein, and the number and variety of the casts, are a much better guide in prognosis than the amount of albumin. The existence of acidosis and of severe nervous symptoms, such as stupor, intense headache, dimness of vision, and persistent vomiting, add much to the gravity of the case, as does also the presence of any serious complication. In general it may be said that if there is no suppression of urine, or if there are no symptoms of uremia and no complications, recovery is almost certain if the child is over three years old; in younger children the outlook is less favorable. The general opinion prevails that acute diffuse nephritis in childhood, whether it is primary or occurs as a complication of scarlet fever, is rarely followed by the chronic form of the disease; and such was the view we formerly held. Larger experience, however, has convinced us that this sequel is not very uncommon. The interval of apparent health may sometimes cover a period of several years, and the later nephritis may be attributed to other causes; but all cases of scarlatinal nephritis should be carefully watched for a long time, and after a severe attack a guarded prognosis should always be given as regards the ultimate result.[1]

Treatment.—Prophylaxis is important, and relates principally to the

[1] The following case may be cited as an illustration of this point: A girl at the age of seven years had scarlet fever, followed by nephritis; the dropsy having lasted, it was reported, for three months. She was believed to have recovered perfectly, and remained in apparent health until she was sixteen, when, as a supposed result of a severe chilling, she developed dropsy and all the symptoms of acute nephritis. From that time, although she lived for three years, and was often for months at a time seemingly in the best of health, her urine was never free from casts and albumin, and she finally died in uremic convulsions.

secondary form which occurs in the course of infectious diseases, especially to post-scarlatinal nephritis; but the measures here outlined apply equally to all varieties. The inflammation of the kidney being in most of these cases the result of direct irritation by the toxins which are eliminated by them, it follows that elimination through the skin and intestines should be increased, and that the urine should be rendered as little irritating as possible by largely increasing its quantity. The first indication is met by frequent sponging, warm baths, and keeping the bowels freely opened by saline cathartics, sufficient being given to produce one or two loose movements daily. To meet the second indication, the patient should be kept upon a diet of milk and farinaceous food, at least for the first three weeks of the disease, and, if possible, for a full month. At the same time he should drink very freely of alkaline mineral waters, or of plain water. If milk is not well borne, kumyss, whey, or buttermilk may be used, or thin gruels mixed with milk. When the first trace of albumin appears in the urine this plan of treatment should invariably be followed. In addition to these measures, after an attack of scarlet fever the patient should be kept in bed for at least a week after the temperature has become normal.

The mild cases of acute nephritis tend to spontaneous recovery under the hygienic and dietetic treatment outlined, i. e., rest in bed, the diet mentioned, the drinking of large quantities of water, and attention to the action of the skin and bowels. These measures should be continued so long as the urine contains any considerable amount of albumin, or so long as the patient's general condition will permit. Should he become very anemic, or lose much in weight, it may be necessary to enlarge the diet by the addition of more solid food. An increase in the diet and exercise should be made very gradually, and the effect upon the urine carefully watched.

The severe cases, with scanty urine, fever and marked dropsy, require more active treatment. Free diaphoresis should be maintained by the hot pack or vapor bath. Active counter-irritation should be used over the kidneys by dry cups followed by poultices, or the mustard paste. Two or three loose movements from the bowels should be secured by the administration of calomel or, better by Rochelle or Epsom salts. Harm is sometimes done by carrying this depletion too far, and its effect upon the patient's general condition must be closely watched. If suppression of urine occurs with the development of uremic symptoms—delirium, vomiting, diarrhea, and a pulse of high tension—venesection should be practiced; from three to six ounces of blood may be drawn from a child of five years, according to his general condition and the urgency of the symptoms. The depressing effect may largely be overcome by immediately following this with an intravenous injection of a normal salt

solution. Twice as much as the fluid drawn should be introduced. This will almost invariably give at least temporary relief, which may afford time for the operation of other measures, such as catharsis and diaphoresis. Pulmonary edema is rather an indication to bleeding; the best of all guides as to its use is a pulse of very high tension.

In addition to these measures rectal injections of a normal salt solution may be given high in the colon, at a temperature of from 104° to 108° F. At least two quarts should be given several times a day, to be continued until a free flow of urine is established. This is one of the most valuable means we possess of increasing elimination by the kidneys and skin.

The nervous symptoms of uremia are best relieved by chloral, which should be given per rectum. When such symptoms are marked, from six to ten grains are required for a child of five years, to be repeated in two hours if no improvement is seen. Uremic convulsions may sometimes be averted by the use of morphin hypodermically.

One should always be on the lookout for complications, especially dropsy of the serous cavities, pericarditis, and edema of the lungs. Convalescence is nearly always slow, and a patient who has suffered from nephritis needs careful attention for a long time. Anemia is always present, and iron is required. The diet should be carefully restricted for several months; much nitrogenous food should be avoided. If the disease tends to pass into a subacute form, the child should, if possible, be sent to a warm climate, and kept there during the succeeding winter, and every means taken to improve the general nutrition. Flannels should be worn next to the skin, and special precautions taken against any exposure which might cause an exacerbation of the disease.

CHRONIC NEPHRITIS

Chronic inflammation of the kidney is an infrequent condition in childhood. In infancy it is almost unknown, except in connection with congenital hydronephrosis or other malformations of the kidney. Two pathological varieties are met with: (1) chronic diffuse nephritis of the parenchymatous or degenerative type; (2) chronic diffuse nephritis of the interstitial or productive type. As the disease progresses the former may assume the characteristics of the latter variety.

Etiology.—Chronic nephritis is most frequently seen as a sequel of the acute nephritis of scarlet fever, less often after other acute infections. The only other important causes in early life are hereditary syphilis, chronic tuberculosis, and valvular disease of the heart. Nearly all the cases occur in children over five years of age.

Lesions.—The lesions of chronic nephritis in childhood do not differ essentially from those seen in later life. In the chronic parenchymatous type the kidneys are usually enlarged, the surface is smooth or slightly nodular, and the thickened cortex yellowish-white on section. These are often called "large white kidneys." On the other hand, the kidneys may be nearly normal in appearance, or smaller and with a thinner cortex than is usual. In the so-called "large red kidneys" the cortex is red or mottled red and yellow, owing to hemorrhages into the tubules or interstitial tissue. The microscope shows that the renal epithelium is swollen, granular, fatty, and degenerated. The tubes contain leucocytes, red cells, cast matter, and the detritus of broken-down epithelial cells. In some places they are dilated, in others atrophied. In the glomeruli there is a growth of cells, compression and atrophy of the tufts, with the formation of new connective tissue.

In the chronic diffuse nephritis of the interstitial type (granular kidney) the organs are smaller than normal, with a nodular surface and adherent capsule. The cortex is thinned, and the color is gray or red. In addition to the lesions found in the preceding variety, there is an extensive production of new connective tissue, which is irregularly distributed throughout the kidneys. The tubules in some places are dilated to form cysts of considerable size, while in others they have completely disappeared. The glomeruli may be atrophied to little fibrous balls; or if chronic congestion has preceded the inflammation, some may be large and the capillaries dilated with hyaline degeneration of their walls.

Symptoms.—1. *Chronic Nephritis of the Parenchymatous Type.*—This form of the disease may be chronic from the outset, or follow an acute attack from which the patient is often believed to have recovered completely. The symptoms sometimes immediately follow the acute attack; at others there is an interval of apparent recovery, extending over a few months or even years. Very rarely no such history of an antecedent acute attack can be obtained and the symptoms come on gradually and insidiously. Such cases occur chiefly in older children, and their clinical features do not differ essentially from those of adult life.

As a rule dropsy is present, although it is variable in amount, and fluctuates considerably from time to time. There may be not only edema of the cellular tissue, but effusion into the pleura, the peritoneum, and even the pericardium. As the case progresses, anemia is always a marked symptom. There are various disturbances of digestion—loss of appetite, occasional vomiting, and attacks of diarrhea. From time to time nervous symptoms may be quite prominent, such as headaches, sleeplessness, neuralgia, fatigue upon slight exertion, and dyspnea. Acidosis

may develop as it does in the nephritis of adults. Attacks of epistaxis are not infrequent.

For the greater part of the time the urine contains albumin and casts. They vary much in amount at different periods in the disease, according to the rapidity of its progress. During periods of exacerbation, both albumin and casts are very abundant, while in the intervals the amount of albumin may be small and the casts few. The casts are hyaline, granular, epithelial, and fatty. The daily quantity of urine is much reduced during the periods of exacerbation, while at other times it may be nearly normal. The specific gravity is usually normal or high.

If amyloid degeneration is present, there are generally associated with the renal symptoms, others dependent upon amyloid changes in other organs. The spleen and liver are enlarged; there may be ascites and diarrhea, and there is usually present a peculiar alabaster cachexia.

The duration of this form of chronic nephritis depends much upon the surroundings of the patient and the treatment. It is rarely shorter than two years, and it may last for many years. The progress is always irregular and marked by periods of exacerbation and remission. The patients die from acute uremia, from some intercurrent disease, or from complicating pneumonia, pleurisy, pericarditis, endocarditis, or from pulmonary edema.

2. *Chronic Nephritis of the Interstitial Type.*—This is a very rare disease in early life, being much less frequent even than the preceding variety of nephritis. In some cases there is a history of hereditary syphilis; in others, of chronic alcoholism. The early symptoms are few, and the disease usually develops insidiously. The urine is pale, excessive in amount, and of low specific gravity—1.001 to 1.008. Albumin is often absent, and, when found, the quantity is small. Dropsy likewise is rare, and never marked. Nervous symptoms are often prominent, such as headache, attacks of spasmodic dyspnea resembling asthma, neuralgias, and disturbances of vision. High blood-pressure and hypertrophy of the left ventricle are regular symptoms; and even atheromatous degeneration of the arteries may be present. Dickinson reports an instance of this in a patient only six years of age. Late in the disease, hemorrhages may occur, and these may be the cause of death. Filatow has reported a cerebral hemorrhage in a child of eleven years. Acute uremia with acidosis is, however, the usual termination of this form of nephritis. The course is slow, and the disease may be overlooked until the final uremic symptoms occur.

Prognosis.—The prognosis of chronic nephritis as to complete recovery is always unfavorable; and although cases are seen in which symptoms are absent for several years, they almost invariably return. As to the duration of the disease, no exact prognosis can be given, because

from the symptoms it is difficult or impossible to determine exactly the extent of the disease in the kidney and the rapidity of its progress. The continued passage of a large amount of urine of low specific gravity is invariably to be interpreted as evidence of fibroid changes in the Malpighian tufts, and is a bad symptom. A large amount of dropsy, the coexistence of valvular disease of the heart, and marked renal insufficiency, as shown by the quantitative examination of the urine and by the phenolsulphonephthalein test, are all very unfavorable symptoms.

Diagnosis.—Chronic nephritis, like the acute forms, is likely to be overlooked because of the failure to examine the urine in children. Regular and frequent examinations should be made in all cases of convulsions, of persistent or frequent headaches, severe anemia, hypertrophy of the heart, high blood-pressure and of general malnutrition, as well as when the more obvious symptoms of renal disease, such as dropsy and scanty urine, are present. Nor should one be too ready to make the diagnosis of functional albuminuria because he finds albumin only occasionally and in small quantity. All such cases demand most careful observation and the closest attention for a long period before excluding organic renal disease.

Treatment.—Children with chronic nephritis are to be treated on the same general plan as adults. The purpose of treatment is to retard as much as possible the progress of the disease and to relieve the symptoms as they arise. It is of the greatest importance to remove the patient from conditions in which exacerbations are liable to occur. If it is possible, he should be sent to a warm, dry climate in winter, and all exposure to cold avoided; an out-door life is desirable. Most patients require general tonic treatment with very moderate but regular exercise, never carried to the point of fatigue, as much rest as possible in a recumbent position, a fluid diet, consisting largely of milk as long as this can be borne, and the administration of iron. Dropsy calls for a salt-free diet, diuretics, saline cathartics, and vascular stimulants. If uremia develops, with high arterial tension and stupor, headache, and convulsions, venesection should be resorted to, or nitroglycerin used. Morphin may be given hypodermically if the nervous symptoms are very marked.

Decapsulation of the kidney is to be considered in cases growing progressively worse in spite of medical treatment. The immediate risks of the operation are rather less than would be expected. We have seen striking temporary benefit in several cases when this operation was done upon young children. In no case, however, was the improvement permanent, all the patients dying within a year after it was performed.

TUBERCULOSIS OF THE KIDNEY

In general tuberculosis, miliary tubercles are frequently seen both upon the surface of the kidney and in its substance. These give rise to no symptoms and are of no clinical importance. Larger tuberculous deposits are extremely rare in early life. They usually occur in patients who are the subjects of general tuberculosis, and are associated with tuberculosis of other parts of the genito-urinary tract, or they may exist as apparently the primary and only tuberculous lesion in the body. Ascending infection occurs occasionally but it is rare; nearly all cases are of the descending type, i. e., primary in the kidney. Infection of the kidney therefore generally takes place through the circulation and not from the bladder. Aldibert's figures show that in children the bladder usually escapes even when the kidneys are tuberculous, for of thirteen cases of renal tuberculosis the bladder was involved in but two. The disease when primary begins in the cortex, but soon extends to the mucous membrane of the pelvis and the calices of the kidney, and also to the pyramids. As a rule, but one kidney is affected. The process may be confined to the pyramids, where are found cheesy nodules which may be single or multiple. These ultimately break down and form abscesses. The process may result in almost complete destruction of the pyramids, and even of portions of the cortex, so that the kidney may consist of a mere shell of renal tissue. Suppuration in the neighborhood of the kidney (perinephritic abscess) often coexists.

The symptoms are quite indefinite. There may be localized pain and tenderness in the region of the kidney, and a tumor if there is perinephritis. The symptoms of irritability of the bladder may be almost as severe as in cases of calculus. Pus usually appears in the urine as a constant symptom, and blood is often present. But the only thing that is diagnostic is the discovery of tubercle bacilli in the urine.

The treatment is the same as in adults.

TUMORS OF THE KIDNEY

In the great majority of cases tumors of the kidneys are malignant. Of fifty-one cases collected by Aldibert which were operated upon, forty-eight were malignant, and three benign.

Malignant growths are almost invariably primary. In children under five years, although not common, they are yet more frequent than any other variety of malignant tumor of the abdomen. Nearly all these tumors belong to the class of embryonal adenosarcoma. They contain

remnants of fetal tissue and in many instances are undoubtedly congenital. Tumors growing from the adrenals belong to a different group—hypernephroma. Renal tumors may grow from the cortex of the kidney, or from the pelvis, sometimes from the adrenals. They may infiltrate the whole kidney, so that there is no trace of renal structure remaining, or they may form an immense tumor on one side of the kidney, which is only partially invaded. These tumors are very rarely cystic, but they are quite soft, and hemorrhages often occur into their substance. There may be secondary growths in the liver, the lungs, the retroperitoneal glands, in the opposite kidney, the intestines, the pancreas, and rarely in the skull. Pressure of the tumor upon the ureter may lead to hydronephrosis, and upon the inferior vena cava, to thrombosis of that vessel. As it grows, the tumor sometimes becomes adherent to nearly all the abdominal organs by localized peritonitis. It may lead to ascites, but it very rarely causes general peritonitis. The growth may reach a great size, usually from five to fifteen pounds, but in one case reported by Jacobi it weighed thirty-six pounds. In Seibert's collection of forty-eight cases the right kidney was involved in twenty-four, the left in twenty-two, and both kidneys in two cases.

Etiology.—These tumors of the kidney may be congenital. This was true of 5 cases in a series of 55 collected by Jacobi. The majority occur in early childhood. In the collection of 130 cases by Longstreet Taylor in which the ages are given, 106 were observed during the first five years, and 57 of these in the first two years of life. The sexes were about equally affected.

Symptoms.—The principal symptoms are tumor, hematuria, and cachexia. The tumor is usually first noticed. It is in most cases discovered in the loin, but grows forward toward the median line. Its surface may be lobulated and irregular or quite smooth; and although solid, it is sometimes so soft as to give an obscure sensation of fluctuation. It may grow to an enormous size, causing displacement of the liver, spleen, intestines, and lungs. The progress of the growth is usually rapid, so that from the size of a fist, the tumor may grow in the course of five or six months so as nearly to fill the abdomen. By careful palpation it will be found—certainly when the tumor is small—that although it may be quite freely movable, its attachment is near the lumbar spine.

Hematuria may in rare cases be the first symptom noticed. The amount of blood passed is sometimes quite large, but is usually small, and blood may be discovered only by the microscope. Pain is rare, and is due to localized peritonitis. Constitutional symptoms are usually absent until the tumor has attained a large size, when a cachexia develops and the patient wastes steadily. The pressure effects are dyspnea, from compres-

sion of the lungs; edema of the lower extremities, from pressure upon or thrombosis of the vena cava; vomiting and indigestion from pressure upon the stomach and intestines. Tumors of the suprarenals have a marked tendency to produce metastaes in the skull. The tumor may remain small and the metastasis may be considered the primary growth. Precocious sexual development is often seen with suprarenal tumors.

The course of the disease is steadily from bad to worse. The usual duration of life in patients not operated upon is from three to ten months after the tumor is large enough to be discovered.

FIG. 85.—SARCOMA OF THE KIDNEY. Child thirteen months old. Weight of tumor, seven pounds. This patient was followed for sixteen years and there was no recurrence.

Diagnosis.—The important points are, the position and attachment of the tumor, its steady growth and solid character, hematuria, and the age of the patient (under five years). It may be confounded with hydronephrosis, dermoid cyst of the ovary, enlargement of the spleen, retroperitoneal sarcoma, tumors of the liver, or even of the abdominal wall.

Treatment.—Nothing is to be said regarding the medical treatment of these cases. Unless operated upon, they invariably terminate fatally. Some of the results of operation during recent years have been encouraging and no case should be abandoned, no matter how young the patient; but a recurrence in a few weeks or months is the usual result.

Benign Tumors.—These are very rare. They are distinguished by their slow growth, and by the fact that the constitutional symptoms are mild or wanting. Of the three cases mentioned by Aldibert, one was adenoma, one fibroma, and one was fibrocystic.

PYELITIS—PYELOCYSTITIS

Pyelitis is an inflammation of the mucous membrane lining the pelvis of the kidney; cystitis is an inflammation of the mucous membrane of the bladder. They may exist separately or together. With pyelitis there may be inflammation of the ureter or of the kidney itself (pyelonephritis), and it may be acute or chronic. It may result in the accumulation of pus in considerable amount in the pelvis of the kidney (pyonephrosis).

Etiology.—Pyelitis may be secondary to local conditions in the genito-urinary tract. It is regularly present with renal calculi. It is also frequently associated with congenital malformations of the kidneys or ureters, with renal tuberculosis and renal tumors. It may result from an extension of inflammation from the tissues surrounding the kidney (perinephritis), or from an abscess opening into the pelvis of the kidney. Acute pyelitis sometimes occurs as a complication of scarlet or typhoid fever, diphtheria, influenza, or pyemia. The organisms found in the urine in these cases are the streptococcus, the staphylococcus, the tubercle bacillus, the typhoid bacillus, the bacillus pyocyaneus, and very rarely the diphtheria bacillus and other bacteria alone or in combination with the colon bacillus.

All these forms, however, are very infrequent compared with the form of pyelocystitis which often occurs apparently as a primary affection. It may be found, however, in the course of any disease, and frequently follows acute disturbances of the gastro-intestinal tract, especially diarrhea. In these cases the evidences of inflammation of the bladder are slight or, more frequently, entirely wanting. This form of inflammation occurs with by far the greatest frequency in female infants. Male infants and older girls occasionally are the subjects of pyelitis. The organism present with great uniformity is the colon bacillus, usually alone. Pyogenic cocci are occasionally associated with it.

The infection has been assumed to be an ascending one, through the urethra, chiefly because of the great preponderance of the cases in girls; but this is by no means established. That infection may take place through the intestinal walls into the genito-urinary tract seems probable in view of the frequency with which pyelitis follows diarrhea and by its occasional presence in boys. Infection through the blood does not seem to be a likely method, for blood cultures in these cases are uniformly negative. Pyelitis is quite frequent in the first two years, after that time the number of cases diminishes, but they may be found at any age.

Lesions.—When pyelitis develops from a local cause it is usually uni-

lateral; otherwise both sides are involved. In the cases of acute pyelitis or pyelocystitis there are the usual appearances of an acute catarrhal inflammation of the mucous membrane with congestion, swelling and sometimes minute hemorrhages. There may be an accumulation of pus of considerable size distending the pelvis and calices (pyonephrosis).

In most of the severe cases of pyelitis there is also present a certain amount of nephritis. This may be merely degeneration or there may be collections of polymorphonuclear leucocytes and even the formation of numerous small abscesses throughout the parenchyma of the kidney. If the condition is one depending upon a calculus or congenital deformity, and in all protracted and severe cases, the mucous membrane of the pelvis is extensively altered. It may be granular, irregularly thickened and present more or less ulceration. In the rare cases of diphtheritic pyelitis there is a false membrane. The kidney in all these forms is involved to a greater or less degree; the extent of the nephritis will depend upon the nature of the exciting cause and the duration of the process.

Symptoms.—There are few diseases in which there is such a great difference in the severity of the symptoms. In perhaps the majority of cases pyelitis is so mild as to cause no symptoms but a slight elevation of temperature of one or two degrees, which may be very temporary. It would entirely escape detection but for an examination of the urine. The pus may be present only in small amount, i. e., four to six cells in each microscopical field of uncentrifugalized urine, and for only a few days.

In other cases the symptoms may be quite severe. The history of the following case illustrates the main clinical features of acute pyelitis, in this instance occurring apparently as a primary disease:

A previously healthy female infant of eight months was taken suddenly with a chill, followed by a very high fever. The child was ill for ten days before the nature of the disease was suspected. During this time the temperature ranged between 101° and 106° F., touching 105° nearly every day; but the chill was not repeated. The other constitutional symptoms were not severe. At the first examination of the urine there was found a large amount of pus, which on standing was equal to one-twelfth of the volume of the urine passed; the reaction was strongly acid. There were no signs of vaginitis or vulvitis, no *ardor urinae,* no evidence of local pain either in the bladder or kidney, no abnormal frequency of micturition, no localized tenderness, and no vomiting. At later examinations there were found in moderate numbers epithelial cells from the bladder, and the tubules and pelvis of the kidney, also a few hyaline casts, but not more albumin than would be explained by the amount of pus. Under no treatment except alkaline diuretics, the tem-

perature gradually fell to normal, and the pus steadily diminished in quantity, and at the end of five weeks had practically disappeared from the urine. The child remained well and entirely free from urinary symptoms.

In some cases there are recurring chills, with wide fluctuations in temperature; in others there may be only pyuria, with moderate fever and few other constitutional symptoms. The course of the temperature is a very irregular one. The fever is seldom continuous, but may be interrupted by periods of normal temperature, lasting several days. A polymorphonuclear leucocytosis is present. The number of cells is usually from 15,000 to 30,000. An agglutination reaction of the colon bacillus with the patient's blood can usually be obtained, often in high dilution. The duration of the acute attack may be from a few days to six or eight weeks, and pus cells may be found microscopically for a much longer time. If the disease complicates one of the acute infectious diseases, pyuria may be the only symptom. If cystitis is also present micturition is frequent, and may be painful. The urine in acute pyelocystitis is turbid from the presence of pus, the amount of which may be from one to fifty per cent of the volume of the urine. The amount of pus varies greatly from day to day. It is often abundant when the temperature is low, and almost absent when the temperature is high, this fluctuation depending upon the accumulation or the discharge of the pus. The quantity of urine is generally somewhat diminished, and it may be quite scanty. The reaction is usually acid, even though the amount of pus is large. Albumin is present in proportion to the amount of pus or the degree of nephritis. Red blood-cells are found under the microscope in most of the very acute cases, and may be in sufficient number to color the urine. The pus cells in recent cases are usually well preserved, but in old cases they may be degenerated. There are many epithelial cells—conical, fusiform, and irregular cells with long tails. There may be renal epithelium and hyaline, granular, or epithelial casts, varying in number with the severity of the nephritis. In a catheterized specimen the colon bacillus is usually present in pure culture.

There is at times seen a particularly severe form of pyelitis. It affects boys as well as girls, usually in the first two years of life. The onset is sharp with fever, gastro-intestinal symptoms, occasionally convulsions, and the temperature is often continuously high. The prostration is extreme, the loss of appetite marked, and anemia develops very rapidly. There is irritability and hyperesthesia, sometimes so marked as to suggest meningitis. The urine contains besides the pus, granular casts in large numbers. The course is prolonged and the mortality relatively high. About 10 per cent of such severe cases prove fatal from exhaus-

tion, from complications affecting the gastro-intestinal tract or the lungs. Thiemich and Göppert have reported a series of such cases that seem to be particularly prevalent in certain localities. We have ourselves observed a small number. The severity of the disease is undoubtedly due to the fact that the kidneys, as shown by autopsy, are severely involved. They are really cases of pyelonephritis.

Pyelitis in older children usually gives more local symptoms. There is frequently pain on urination. Pain in the abdomen or loins may be marked and there may be tenderness and even muscular rigidity. When the right side is involved it may be difficult to exclude appendicitis.

Pyelitis has a marked tendency to recur. It may do this after a few weeks or months or perhaps not for several years. Some children may suffer from a number of attacks. Others show few, if any, constitutional symptoms, but their urine for a long period may never be free from pus cells and there may be exacerbations with fever from time to time for many months.

In pyelitis depending upon congenital malformations, pyuria is usually the only symptom, unless pyonephrosis is present. With calculi there is an acute or chronic pyelitis; there may be localized pain, tenderness, sometimes a tumor, occasionally hematuria, and perhaps a history of renal colic or the passage of gravel. With tuberculosis, there is chronic pyuria and the presence of tubercle bacilli in the urine. The symptoms of general tuberculosis are commonly associated. If there is perinephritis, the inflammation is usually acute, and there are present the local symptoms of the original disease. If an abscess opens into the pelvis of the kidney, there may be a sudden discharge of pus in large quantity with a subsidence of previous local symptoms, including the tumor. With neoplasms, both pus and blood may be found in the urine, but the latter is more frequent.

Diagnosis.—The characteristic symptoms of acute pyelitis are chills, which may be repeated, high and fluctuating temperature, scanty urine containing pus, and occasionally pain and tenderness over the kidneys. All of these may be absent, however, except the fever and the pyuria, and both the fever and the pyuria may be intermittent. The diagnosis of pyelitis is made only by an examination of the urine, which, particularly in infancy, should never be omitted in cases of obscure high temperature, whether prolonged or only temporary. If pus is not found the examination should be repeated several times. When cystitis is associated, the only additional symptoms may be pain and other signs of vesical irritation. These symptoms, with an acid urine containing more or less pus and numerous epithelial cells, are sufficient to establish the diagnosis of pyelocystitis. If the pus comes from the opening of an

abscess into the bladder, ureter, or pelvis of the kidney, the local signs of such abscess will usually be present.

Prognosis.—In cases apparently primary, and especially in those due to the colon bacillus, the prognosis is good. The danger is chiefly from the nephritis which follows or complicates the process and to very young and poorly nourished infants who may die from exhaustion as the result of gastro-intestinal disturbance. The prognosis in the malignant form is always doubtful. In cases depending upon local conditions, the prognosis will depend upon the nature of the exciting cause. Here, also, the principal danger is from nephritis. If calculi are present and if pyonephrosis occurs, the patient may die from exhaustion before a serious degree of nephritis has developed.

Treatment.—Water should be given freely, and alkalis up to the point of neutralizing the excessive acidity of the urine. A large amount of alkali is necessary to accomplish this. Citrate of potash sufficient to render the urine alkaline in this condition is apt to cause diarrhea or vomiting. It is therefore wise to give not more than five or ten grains of this three times a day, but to give bicarbonate of soda from twenty to thirty grains every four hours, according to the age of the patient. The urine should be kept alkaline for some time after the subsidence of all symptoms. The most widely used remedy is hexamethylenamin (urotropin), which may be given in doses of one or two grains every three hours to an infant of a year, and proportionate doses to older children. In order that this drug should have an antiseptic action the urine must be acid. It is improper, therefore, to combine hexamethylenamin with alkalis. We have seen it used in large and small doses in cases of acute pyelitis, but have not been convinced of its value. Occasionally pyelitis is very resistent to any form of treatment, the exacerbations and remissions continuing for many weeks. For such obstinate cases vaccines, preferably the autogenous variety, should be tried. Striking benefit has sometimes followed their use. If calculi are present or other conditions, such as perinephritis, etc., the methods of treatment applicable to these diseases are indicated.

RENAL CALCULI

Small renal calculi are very common in infancy. In the autopsy room we frequently see, on opening the kidneys of young infants, fine brown granules in the pelvis and calices, and occasionally a calculus as large as a small pea is found. They are usually composed of uric acid. Only once in over two thousand autopsies of which we have records, was a stone of any considerable size seen in an infant. In this case it was an inch in length and half an inch wide. It is surprising that these are

so rare, when we consider how very frequently the minute calculi are met with. The probable explanation is, that the majority of them are dissolved or washed down into the bladder and passed *per urethram* because of the fluid diet of the first two years. The granular deposits are usually lodged in the pelvis of the kidney, and are generally seen upon both sides. With the larger collections there is often a slight catarrhal pyelitis.

Symptoms.—The small deposits give no symptoms, and even quite large calculi may be found at autopsy when no indication of their presence had existed during life, as in the case above mentioned. In some cases symptoms are produced which resemble those of renal calculi in the adult. In infants less definite symptoms are often passed over as merely intestinal colic.

In well-marked cases in older children there is tenderness, pain localized over the affected kidney, or radiating to the bladder, the perineum, and even the opposite kidney, and there may be irritation and retraction of the testicle. The urine may show, especially after exercise, a trace of blood; there may be the added symptoms of pyelitis, with some fever, localized tenderness, and the appearance in the urine of pus and epithelial cells from the pelvis of the kidney.

Renal colic is produced when a stone of any considerable size passes from the kidney to the bladder. It is characterized by symptoms similar to those seen in the adult. There are sudden attacks of severe sickening pain in the loins, shooting down the thigh or to the testicle. There may be vomiting and even collapse. The urine is passed frequently, in small quantities, and contains blood. The symptoms quickly subside when the stone reaches the bladder. The calculus may sometimes become impacted in the ureter and give rise to hydronephrosis or pyonephrosis, which soon becomes pyelonephritis.

The existence of small calculi may be suspected from the symptoms above mentioned; the diagnosis is made positive by the appearance of gravel in the urine. The use of the X-ray is of service in recognizing even small calculi.

Treatment.—The only medical treatment consists in a fluid diet, the free use of alkaline mineral waters, and a sufficient quantity of some drug to render the urine alkaline. Such measures will relieve only the milder conditions. With larger calculi and more marked symptoms, a surgical operation should be considered and should be urged in proportion to the severity of the symptoms and the clearness of the diagnosis. If calculous pyelitis exists, it is certain sooner or later to lead to serious nephritis, and it is only a question of time when the kidney will be disabled. The same is true of hydronephrosis from the impaction of a calculus in the ureter. Aldibert has collected four cases of nephrectomy in

children for renal calculi in which the kidney was healthy, with three recoveries and one death from shock. In nine cases of operation for calculous pyonephrosis, there were six recoveries and three deaths. The earlier the operation the greater the chances of success, because of the better condition of the other kidney. Although the continued use of water and the use of drugs may relieve some of the symptoms, it is very questionable whether they do more.

PERINEPHRITIS

This consists in an inflammation in the cellular tissue surrounding the kidney, which may terminate in resolution or in suppuration. It is not of very uncommon occurrence. Perinephritis may be secondary to suppurative processes in the kidney itself, whether from calculi or tuberculous deposits, or it may be primary. In children the latter is the common form. Primary perinephritis is attributed to traumatism, cold, or exposure, or it may develop without assignable cause. It usually runs an acute or subacute course; very rarely it may be chronic.

For the clinical picture of this disease we are chiefly indebted to a paper by Gibney, who has published a report of twenty-eight cases of primary perinephritis in children. The ages of these patients were between one and a half and fifteen years, the majority being between three and six years. The two sides and the two sexes were about equally affected. About one-third of the cases were clearly traceable to traumatism; in the others no adequate exciting cause could be discovered. The majority of the cases were referred to the hospital with the diagnosis of hip-joint disease or caries of the spine. Resolution followed in twelve of these cases, and sixteen terminated in suppuration.

When abscess forms, it usually burrows between the lumbar muscles and comes to the surface posteriorly near the middle of the iliocostal space; it may burrow forward between the abdominal muscles and point just above Poupart's ligament; very rarely it may follow the psoas muscle and appear at the upper and inner aspect of the thigh, like an ordinary psoas abscess; or it may open into the peritoneal cavity.

Symptoms.—The onset of acute perinephritis may be quite abrupt, with chill, fever, and localized pain; or it may be gradual, with stiffness of the spine, lameness referred to the hip, and deformity due to the contraction of the flexors of the thigh. The pain is usually felt in the loin, but may be referred to the groin, to the inner side of the thigh, or to the knee. It is often severe, and increased by using the limb. It is in most cases accompanied by localized tenderness in the neighborhood of the kidney. There is lameness upon the affected side, which may come on

gradually, being sometimes referred to the hip and sometimes to the spine. These symptoms often develop slowly in the course of two or three weeks. They are usually accompanied by a slight elevation of temperature. In the most acute cases the temperature is high (102° to 104° F.), and prostration severe.

As the disease progresses, fever is a constant symptom, the temperature usually varying between 101° and 103° F. There is in most cases increasing deformity, and finally the patient may be unable to walk at all. On examination at the height of the disease, there is found in a typical case a deviation of the spine with the concavity toward the affected side; the thigh may be held flexed to a right angle; passive extension is resisted and causes pain, although all the other movements at the hip joint are normal. In the lumbar region there is tenderness, and there may be an area of infiltration filling the iliocostal space. At first this is only appreciable by percussion, but later a distinct tumor is present. In addition to the tumor in the usual region, there is sometimes one at the upper and inner aspect of the thigh, owing to a burrowing of pus, and the sacs may communicate.

Lameness, pain, deformity, and fever sometimes exist for two or three weeks before any tumor can be made out. The constitutional symptoms are often severe. The size of the abscess is sometimes very great. In one case we saw it extend from the spine to the median line in front, and from the crest of the ilium nearly to the free border of the ribs. The amount of pus varies from a few ounces to two or three pints. Urinary symptoms are sometimes wanting; at other times there is increased frequency of micturition, accompanied by pain from an irritation referred to the bladder. The urine may contain pus from a complicating pyelitis. In only one of Gibney's cases was this present. It developed in the fourth week, and the child recovered.

The duration of the disease in the acute cases varies from three to eight weeks; in the subacute it may be five or six months. When suppuration occurs the symptoms subside quite rapidly after the pus has been evacuated, and recovery is complete. When resolution takes place, there is a gradual subsidence of the symptoms, and often some stiffness of the thigh, with slight lameness for several months. In the series of cases above referred to, sixty-five per cent recovered completely in three months.

Diagnosis.—In many cases a diagnosis of hip-joint disease is made, but that disease develops more insidiously, is very much more chronic, and rarely produces so great deformity in a year as is often seen in perinephritis in two or three weeks; abscess is infrequent during the first year of the disease. In perinephritis, on the other hand, we have a tolerably acute onset, sometimes with chill, fever, marked lameness, and

deformity, developing in two or three weeks; abscess often forms in a month, and complete and permanent recovery usually follows after a few months at most; the deformity is due solely to flexion of the thigh; all other movements at the hip may be free, and joint tenderness is absent. Psoas abscess from Pott's disease may cause deformity, tumor, and lameness similar to that seen in perinephritis, but on examination there is found the angular prominence and other signs of disease of the lumbar vertebrae. In cases of doubt the tuberculin test may give important aid in diagnosis.

Prognosis.—Primary perinephritis in children almost invariably terminates in complete recovery. Of the twenty-eight cases referred to, and eight subsequently observed by Gibney, all recovered perfectly. The only condition likely to prove fatal is rupture of the abscess into the peritoneal cavity.

Treatment.—The patient should be put to bed and kept as quiet as possible throughout the attack. In the early stage, hot fomentations or an ice-bag should be applied over the affected side; heat is generally to be preferred. Abscesses should be opened early, to prevent burrowing and the danger of a possible rupture into the peritoneal cavity.

CHAPTER III

DISEASES OF THE GENITAL ORGANS

MALFORMATIONS

Adherent Prepuce.—This condition is sometimes called false phimosis. It is so constantly present that it can hardly be regarded as a malformation. It is, however, a condition often needing attention in male infants. The prepuce should be retracted so as to expose the glans completely. The smegma should then be washed away, the glans covered with a drop of oil, and the skin drawn forward. This should be repeated daily until there is no disposition to a recurrence of the adhesions.

Phimosis.—This is such a narrowing of the prepuce that it can not be retracted over the glans. The degree of phimosis varies greatly. In very rare cases there is no preputial opening. In other cases the orifice is so small that no part of the glans can be exposed, and there is obstruction to the outflow of urine; but usually a small part of the glans can be seen. Phimosis may be complicated by an elongated prepuce (hypertrophic phimosis), and the elongation may exist without any narrowing of the orifice, although this is usually present to some degree.

The presence of phimosis makes cleanliness impossible in many cases, and want of cleanliness leads to infection and to balanitis. This is quite frequent, even in infants. It may be complicated by urethritis, and even by cystitis. Another consequence of the straining induced by phimosis is hernia, which may be either inguinal or umbilical. To cure the hernia is often impossible, unless the phimosis is relieved. The list of reflex phenomena which have been ascribed to phimosis is a long one. There has been a disposition on the part of some to attribute nearly all the nervous disturbances of boyhood to phimosis, and an exaggerated importance has certainly been attached to this condition. A very marked degree of phimosis often exists in children without producing any symptoms. That phimosis is an etiological factor in many neuroses is certainly to be doubted. Our experience with circumcision as a cure for such conditions has been very unsatisfactory. When cleanliness is impossible the irritation and resulting pruritis may cause frequent priapism and may at times encourage masturbation. Phimosis may rarely lead to vesical spasm and retention of urine, but more frequently to nocturnal incontinence.

Treatment.—Phimosis should receive attention in infancy. Often very little treatment is needed. When there is a very long prepuce with phimosis, the operation of circumcision should be done, even when the degree of phimosis is slight. Many cases of phimosis in which the prepuce is not long can be relieved by stretching. If no part of the glans can be exposed, the simplest plan is to slit up the dorsum of the prepuce with a pair of scissors and break up the adhesions. The corners of the flaps thus made can then be snipped off and one stitch inserted on either side. To promote cleanliness in older boys or in cases of hernia or prolapse and when phimosis is present, circumcision should be performed.

Hypospadias.—In this condition the urethra is not continued to the tip of the penis, but opens on the inferior surface some distance back, being represented in front of this only by a shallow furrow. In more severe cases there is a deep fissure which divides the scrotum, and sometimes even the perineum. Into this fissure the urethra opens. This is a condition likely to be mistaken for that of hermaphrodism, especially as the testicles are frequently in the abdominal cavity.

Epispadias.—This is a condition in which the urethra opens on the dorsal surface of the penis. It is much less frequent than hypospadias. There may be simply a division of the glans, or the fissure may extend the whole length of the organ and be complicated by exstrophy of the bladder.

Exstrophy of the Bladder.—In the complete form there is a median fissure from the umbilicus to the tip of the penis. It includes the an-

terior abdominal wall, the pelvic bones, and the urethra. The bones are entirely separated at the symphysis, or connected behind the bladder by a fibrous band. The hypogastric region is occupied by a red, mucous surface, slightly corrugated, which is all there is of the bladder. In the lower lateral portions of the red mucous membrane two slightly rounded elevations are seen, from which urine oozes. These are the openings of the ureters. The penis is short, and presents a shallow furrow on its dorsal surface. The testes are often in the abdominal cavity.

An analogous deformity is sometimes seen in girls. There is a division of the clitoris and the labia minora and majora. The fissure may be so deep as to reach nearly to the anus. The vagina is usually absent. The rectum may open into the prolapsed bladder.

All these deformities are compatible with long life. In exstrophy of the bladder, whether complete or partial, patients are a nuisance to themselves and to all about them. It is almost impossible to prevent the clothing from being soaked with urine, which gives everything connected with the patient a strong ammoniacal odor. The skin is often excoriated. Operation for the relief of these cases should always be undertaken. The operation to be recommended is the transplantation of the ureters into some part of the large intestine, usually the rectum. The results are often most surprising. The rectum soon becomes tolerant of the urine, holds it for hours without difficulty and evacuates it without discomfort. Ascending infection of the kidney seldom occurs.

Undescended Testicle—Cryptorchidism.—In fetal life the testes are situated in the abdominal cavity below the kidneys. They usually descend into the scrotum during the ninth month, but in children born at term the testicles may be in the inguinal canal, or even in the abdomen. The former condition is quite frequent, being present in fully ten per cent of all male children. In most of these the descent takes place without difficulty during the first weeks of life, and causes no symptoms In others the condition may persist. Spontaneous descent may take place at any time before puberty, the chances, however, steadily lessening as age advances. When in the inguinal canal, on account of its exposed situation, the testicle may be injured, or become painful and tender as puberty approaches. In any abnormal position it probably will not develop properly, and may remain without function, but interference with the development of the body is rare. Hernia is a frequent complication.

When in the inguinal canal, descent of the testicle may sometimes be facilitated by manipulation. If the condition is unilateral, operation is unnecessary except for relief of pain. If it is double, operation should be performed before puberty, preferably from the ninth to the eleventh year. Transplantation into the scrotum is at this time simple, and usually successful. Should pain be persistent, and transplantation impossi-

ble, the testicle may be replaced in the abdominal cavity. Removal is indicated only when degeneration has taken place.

With the exceptions already mentioned, deformities of the female genitals belong rather to gynecology than to pediatrics, since they are chiefly of the internal organs, and do not usually give symptoms before puberty.

DISEASES OF THE MALE GENITALS

Balanitis.—Balanitis, or inflammation of the prepuce, is one of the results of phimosis. It may follow decomposition of the smegma, infection of the mucous membrane, injury, or masturbation. The parts are swollen, edematous, red, painful, and sometimes bathed in pus. Retraction of the prepuce is impossible. Under proper treatment the inflammation usually subsides in two or three days, but there may be some discharge for a considerable time. Abscess may follow, and even gangrene of the prepuce. The most severe cases are likely to be complicated by anterior urethritis. We have frequently seen erysipelas start from balanitis, and occasionally diphtheria occurs here.

The object of treatment is to remove the irritating and infectious material lodged beneath the foreskin. This may be quite difficult. It is best accomplished by syringing with a 1-5,000 bichlorid solution, and the constant application of a wet antiseptic dressing. Ice is often useful when the edema is great. It is sometimes necessary to slit up the prepuce before the parts can be thoroughly cleansed, and in severe cases this is often the quickest method of cure. Circumcision should not be done during an attack.

Urethritis.—This, like the same disease in females, may be simple or specific. Both forms are much less frequent in little boys than in the other sex. In simple urethritis the inflammation usually affects only the anterior part of the canal, the fossa navicularis. There is a slight discharge of pus, and sometimes pain on micturition. The most frequent cause is want of cleanliness.

Gonococcus inflammation is more common. This occurs even in infants, but most of the cases are in those over seven years old. The usual cause is direct contagion. The symptoms are more severe than in the simple form, and resemble the same disease in the adult, with the exception that constitutional symptoms are usually absent. A microscopical examination of the discharge is the only positive means of diagnosis between the two varieties. In these cases it reveals the gonococcus in great numbers. Conjunctivitis and arthritis are seen as complications, just as in the female. Epididymitis is rare, but balanitis and bubo are not infrequent. Poynter has reported a case in a boy of three years, who,

when five years old, required treatment for a urethral stricture. He was infected by a nurse.

The first thing in the treatment is always to keep the parts covered, otherwise the infection may be carried by the hands to other mucous membranes, usually the conjunctiva. In other respects the treatment is the same as in the adult.

Hydrocele.—Hydrocele consists in an accumulation of serum in some part of the serous pouch brought down by the testicle in its descent. In infants it is usually due to the imperfect closure of this pouch at some point, where a fluid accumulation occurs. Four varieties of hydrocele are met with in young children.

1. *Congenital Hydrocele.*—In this the condition is a congenital one, although the tumor is not necessarily present at birth. The tunica vaginalis communicates with the general peritoneal cavity. There is present an elongated tumor, extending from the bottom of the scrotum throughout the whole length of the cord. The tumor is reducible, sometimes spontaneously by position, sometimes, when the opening is smaller, only by pressure. It reduces slowly, without gurgling, never going back *en masse* like a hernia. The tumor is translucent, and is flat on percussion. The testicle is above and posterior, and usually indistinctly felt. Congenital hydrocele may be complicated by hernia.

2. *Hydrocele of the Tunica Vaginalis with the Canal Closed.*—In this form the accumulation of fluid is in the scrotum, communication with the peritoneal cavity having been entirely cut off by the complete obliteration of this pouch in the canal in the normal way. This is one of the most frequent forms. It gives rise to an oval or pear-shaped tumor, quite tense and firm, usually about two inches in length. The cord is distinctly felt above it, the testicle is behind and somewhat above it, and not always felt very distinctly. This variety gives translucency and the usual elastic feeling of a hydrocele.

3. *Hydrocele of the Cord.*—This is one of the rare forms. The serous pouch which accompanies the spermatic cord is open above, and communicates with the peritoneal cavity; but below it is closed. The scrotum is normal, and the testicle is in its usual position. The tumor is small, elongated, reducible, and entirely above the scrotum. Usually it stops at some point in the inguinal canal. This hydrocele also may be completed by hernia. The diagnostic points are the same as in the form first mentioned.

4. *Encysted Hydrocele of the Cord.*—The peritoneal pouch of the cord in this variety is closed for some distance above, and again below, but somewhere in its course it is open, and here the fluid accumulates in the form of a cyst. When small it resembles an undescended testicle; but on examination this organ is found below and in its normal position.

When in the canal, it is often mistaken for a lymph gland, sometimes for a small hernia. The tumor is usually about the size of an almond. It is elastic and irreducible, and translucent like the other varieties.

Treatment of Hydrocele.—In the congenital form the application of a truss will sometimes cause obliteration of the canal, so as to shut off the hydrocele sac from the general peritoneal cavity. It is subsequently managed like an ordinary hydrocele of the tunica vaginalis. In infants and young children it is rare that active operative measures are called for in any variety of hydrocele, as these usually tend to disappear spontaneously in the course of a few months. Iodin may be applied locally over a hydrocele of the cord, but should not be applied to the scrotum. Some cases are cured by a simple puncture with a needle, allowing the fluid to drain off into the cellular tissue of the scrotum from which it is absorbed; others by a single aspiration with a hypodermic syringe. It is seldom necessary to resort to the injection of irritants like iodin or carbolic acid, but they may be used if the fluid returns after repeated aspirations.

DISEASES OF THE FEMALE GENITALS

VAGINITIS

This is a catarrhal inflammation usually affecting only the vaginal mucous membrane, but may involve the urethra, bladder, and, in older girls, the lining membrane of the uterus, the tubes, and even the peritoneum. It may be either simple or specific (gonorrheal); the purulent form is almost invariably specific.

Simple Vaginal Catarrh.—This may be seen at any age, even in infancy, but is most frequent after the second year. It occurs especially in girls suffering from malnutrition and anemia, and whose personal cleanliness is neglected. It may follow any of the infectious diseases, particularly measles. It sometimes complicates varicella with a local lesion in the vagina. It may be traumatic, as from attempted rape or the introduction of foreign bodies. Other causes are pinworms and scabies. It is sometimes the cause, sometimes the result of masturbation.

The disease generally begins as a subacute catarrhal inflammation, the discharge being the first, and in mild cases the only symptom. It is of a white or yellowish-white color and not very abundant. If the parts are not kept clean the odor of the discharge is quite foul. In severe cases the discharge is abundant, and may excoriate the skin of the labia and thighs. The mucous membrane is swollen and red, but there is only a moderate secretion. Microscopical examination of the discharge shows bacteria in large numbers and of many varieties, but they are chiefly the ordinary cocci. With proper treatment and in children who

are in good general condition, the disease usually lasts but a few weeks. Under unfavorable conditions a leucorrheal discharge may continue for a much longer time.

Cases of simple vaginal catarrh should be irrigated daily with a warm saturated solution of boric acid or 1 to 5,000 bichlorid. Cleanliness should be secured by frequent bathing and the skin protected by ointments. In more severe cases, astringent injections, such as sulphate of zinc and tannic acid (of each one dram to a pint of water) should be used. The general health should be built up by iron, cod-liver oil, and other tonics.

Gonococcus Vaginitis.—This disease once considered rare in children has been shown to be exceedingly common in girls of all ages, even in young infants. Its control has become a social problem of much importance, and one that is beset with peculiar difficulties. Gonococcus vaginitis is an especial scourge in institutions, in homes and asylums for older girls, and in those for infants as well; also in hospitals, particularly those in which prolonged residence is necessary. Routine examinations made in large institutions for children have revealed the presence of this disease, often, it is true, in a mild form, in from 2 to 10 per cent of the female inmates. In a single year, of 1,200 children under three years, chiefly infants, applying for admission to the Babies' Hospital, 63, nearly one per cent of the females, were found to be suffering from gonococcus vaginitis. Epidemics in institutions are frequent and very difficult to control. Before means of prevention were so well understood as they are now, four epidemics were observed in the Babies' Hospital in five years, with 273 cases.[1] Day nurseries are another common agency of spreading the disease.

But gonococcus vaginitis is by no means confined to the classes mentioned. In out-patient practice and among the poor who live in tenements, it is common in girls of the school age who have never been exposed in institutions. Even in private practice among the well-to-do, cases are not very rare.

The ultimate source of infection in children with this disease in most cases is undoubtedly contact in the home with adults suffering from it. In several series of cases carefully investigated fully one-third have been definitely traced to a mother or sister suffering from the disease, with whom the young child has slept. In the home, infection may also take place by baths, clothing, dirty toilets, etc. Among companions infection may take place by manual contact, masturbation being frequent among infected persons; in schools and other public places it may unquestionably be spread by the toilet seat. Criminal assault is a rare cause among children.

[1] "Gonococcus Infections in Institutions," N. Y. Medical Journal, March, 1905.

In institutions for infants and young children the disease is most often acquired through the medium of diapers. Other possible sources of contagion are towels, sponges, wash-cloths, clothing, bed linen, thermometers, syringes, bath tubs, and bath water. Even when the most careful attention is given to all these matters we have sometimes seen ward epidemics continue. The most probable explanation of such a condition is that the disease is spread by the hands of the nurse in washing, dressing, or the changing of napkins. In such cases nurses as well as infected children must be quarantined. In some instances it is impossible to trace the mode of spreading.

The susceptibility of the vaginal mucous membrane to gonococcus infection is very great in young children, which in part accounts for the prevalence of this disease. A further reason for the frequency of infection is probably to be found in the want of protection of the mucous membrane owing to the small size of the labiae. Vaginitis should not in early life be regarded as a venereal disease.

The constant presence in cases of vaginitis in children of an organism which morphologically and culturally is identical with the gonococcus found in acute inflammations in the adult, has led to the belief that the two diseases were identical. But the mildness of the local inflammation in the great majority of the cases in young children, the absence of constitutional symptoms and of serious complications has led to the suspicion that there might be important differences in the infecting agent in the two groups of cases. Pearce, of the Rockefeller Institute, has recently shown by immunological tests (agglutination and complement fixation) that the type of organism in the two groups is quite distinct. Not a single exception was found in the cases studied. The infrequency of ophthalmia as a complication in little children has often been noted. In our own experience it has been rare. In this connection it is interesting to note that in cases of ophthalmia in infants studied by Pearce the organism corresponded in every instance to the adult type. Should these differences in type prove to be the rule, we may find that gonococcus vaginitis in young children, though a most troublesome condition, is not so serious a matter as many have been inclined to regard it.

Symptoms.—In the mild cases the disease is limited to the mucous membrane of the vagina. There is a moderate yellow discharge, smears of which show pus cells and gonococci. There is very little redness of the mucous membrane and no local symptoms of discomfort. In the more severe form the discharge is copious, often thick and of a yellowish-green color. It may be tinged with blood from slight erosions. It causes excoriation of the labiae and inner surface of the thighs. Micturition may be frequent and painful owing to the involvement of the urethra. If a small speculum is introduced and the parts examined with

a good light, the extent and severity of the disease can be determined. It is usually seen that the inflammation is a general one affecting the urethra, vagina, hymen, and the cervix uteri. The parts are intensely congested, granular in appearance and the purulent discharge may be seen coming from the cervix. With these severe local symptoms there may be in the acute stage some constitutional symptoms as in the adult. But the cases seen in little children are seldom of this severe form.

In the most severe cases, usually seen in girls past the age of six or seven years, the inflammation may involve not only the cervix, but the entire endometrium; it may extend to the Fallopian tubes and even the pelvic peritoneum. Cases of this severity may be seen, though very rarely, in children of only three or four years. We have never met with them in infants. Swelling and suppuration of the inguinal glands are very rare. Other complications are conjunctivitis, arthritis, endo- or pericarditis, meningitis, and proctitis. Conjunctivitis is surprisingly infrequent in very young patients. Arthritis is usually multiple and involves especially the small joints of the fingers, toes, wrists, or ankles, but the large joints may also be attacked. Symptoms of pyemia are usually associated. These cases are more fully considered in the chapter on Acute Arthritis in Infants. The diagnosis in all the complicating conditions rests upon the presence of the gonococcus. Masturbation is not uncommon in these cases and occasionally it is associated with sexual precocity.

Diagnosis.—A positive diagnosis between simple and gonococcus vaginitis can be made with certainty only by a microscopical examination of the discharge, though in default of such examination an abundant purulent discharge may be assumed to be due to the gonococcus. In simple catarrh the discharge is made up of epithelial and pus cells with quite a wide variety of bacterial forms, chiefly cocci and bacilli, occasionally a few diplococci. In gonococcus vaginitis the gonococci are found in large numbers, and are usually the only bacteria present. To be diagnostic, they should be demonstrated within the pus cells as well as outside them. The gonococcus decolorizes when stained by Gram's method, which fact distinguishes it from the other organisms likely to be present in the vagina. The staining is quite as diagnostic as the cultural characteristics of this organism. Cases of vaginitis are to be regarded as suspicious if pus is found and few organisms are detected; in such conditions subsequent examination usually reveals the gonococcus. In our hospital experience the gonococcus cases have outnumbered the simple purulent forms, fully twenty to one.

Since the diagnosis rests upon the microscopical examination of smears made from the vaginal secretion, the manner in which smears are taken is important. A moist swab or a platinum loop may be used,

the latter being preferred, or a few drops of a 1 to 10,000 bichlorid solution may be instilled into the vagina and withdrawn with a pipette; after evaporating the fluid the residue is stained. The smear should be taken far inside the vagina, preferably through a small speculum, such as a female urethroscope. Unless these precautions are used a good many cases will be missed, especially since smears from the cervix are sometimes positive when those taken from the vagina may be negative. When properly made and examined by an experienced person the results of the examination may be relied upon for diagnosis. In a certain proportion of the cases, usually those of a severe type with constitutional symptoms, a positive result is obtained by the complement fixation test. This reaction is also at times of value in establishing the fact of cure. In cases complicated by multiple arthritis the gonococcus is usually found by blood cultures, even though the vaginal smears may be negative.

Prophylaxis.—The problem of controlling this disease is a difficult one owing to its great frequency, its extremely contagious character, its protracted course, and the unsatisfactory results of treatment. Educational measures come first in importance. Mothers, nurses, social workers, matrons of institutions, hospital and school authorities should all be made acquainted with the prevalence of the disease and the means by which it is usually spread. The attitude of the public toward the problem would be more intelligent if the idea that vaginitis in young children is a venereal disease could be gotten rid of. Even girls themselves who are likely to be exposed, should be instructed as to the dangers of infection and the means of its avoidance. The importance of proper cleansing of the genitalia is the first lesson to be taught. In the home, essential measures of prevention are that an infected person should sleep alone, should wear a vulvar pad of such a character that it can be destroyed, that sheets and clothing should be washed separately from those of the household, and that especial care be used about both bath tubs and bath water and the toilet seat. In the school the greatest danger is probably from the common toilet; scrupulous cleanliness of this should be secured; only the U-shaped toilet seat should be used, not merely in schools but in all public places. Another chief source of infection being contact with infected companions, this should be limited so far as possible. To make the disease a reportable one and exclude infected children from public schools does not seem a practicable measure, since this would involve the examination of smears from all the girls attending school. The importance of the disease does not justify such radical measures.

It is in institutions for children that the problem of prevention is most difficult and also most important. In all day nurseries, hos-

pitals and homes similar means must be employed, viz., the examination of vaginal smears from every child on admission should be a matter of routine; cases showing the gonococcus should not be received into the same ward or dormitory with others, and even cases showing only pus cells but no gonococci should be quarantined. In hospitals for children, routine smears should be taken from all female children at least once a week. In no other way is it possible to recognize cases early and prevent ward epidemics.

The attendants, both day and night nurses, as well as the affected children, should be quarantined. Napkins, underclothing, and sheets from the beds of such patients, also their towels and wash-cloths, should not go into the common laundry, but should be first soaked in a strong solution of carbolic acid, and afterward boiled. In wards or institutions where cases have occurred, washable napkins should be discontinued and old muslin and absorbent cotton substituted. These are to be destroyed after using. All articles connected with the children's toilet, also syringes, thermometers, etc., should be carefully disinfected. But often this is not enough. Separate articles should be furnished for each child. The essential measure is a prompt recognition and isolation of the first case in the hospital. The danger to life in this disease is not great, and is from the serious complications mentioned above, all of which are very infrequent in young children. In very many cases, however, the disease lasts for years even in spite of treatment and the question of the ultimate damage to the general health or, what is more important, to the organs involved must be considered. At present we have not enough knowledge to warrant positive statements upon this point. It is possible that many of these protracted cases ultimately recover spontaneously, or that after long continuance of the disease the organisms present have such a low virulence that their capacity for injury is very slight indeed. The disease is not a new one and it is very prevalent; were the ultimate dangers as great as some have asserted more evidence of this would exist than now appears to be the case. Facts now at hand do not justify the belief that the ultimate dangers from vaginitis in children are great, or in any way comparable to acute gonococcus vaginitis acquired in adult life. Some reason for this may be found in the biological difference in the gonococci from adult and infantile cases which has been already referred to.

Treatment.—On account of its very chronic character and its prevalence chiefly among the poor, most cases of vaginitis must be treated in out-patient clinics. Special clinics for such cases should be established in every large city, attached to which should be a visiting nurse who should see that proper treatment is carried out in the home. To be at all successful local treatment must be thoroughly carried out by a

physician and for a long period. The first essential is local cleanliness which must be secured by bathing the external organs twice a day with a solution of boric acid or some similar preparation. In spite of the obvious objections to their use, irrigations are probably the most valuable of the local measures we possess. These should be made daily if possible and through a catheter whose tip is carried well into the vagina. Boric acid solution or permanganate of potash 1-2,000 to 1-5,000, ichthyol 1-1,000, or bichlorid 1-10,000 may be used. Following the irrigation local applications should be made every second or third day of nitrate of silver 10 per cent, or argyrol 20 per cent strength. These should be made with an applicator through some sort of a speculum—the female urethroscope answers very well for small patients—and the child kept upon the back with the thighs in contact for a short time. If the cervix is involved local applications made in the manner indicated are essential if anything is to be accomplished.

Regarding the value of vaccines there is still much difference of opinion. Some writers have reported excellent results while others with considerable experience have seen little benefit from their use. Our own experience is that their effects are very uncertain; that, while in some instances striking improvement has been seen, in the great majority of cases this does not occur. The best results are seen in the most recent cases. Regarding the value of vaccines in some of the complications, especially arthritis and general sepsis, there can be little question. The autogenous appear to have no advantage over stock vaccines. The dosage of vaccines is still empirical. It is customary to give from 50,000,000 to 75,000,000 as an initial dose, to repeat every five or six days, gradually increasing this to 100,000,000. If no improvement is seen after six or eight injections, their continuance is useless. In connection with the administration of vaccines careful bathing of the external organs should be combined, but irrigations may be omitted. Because of the favorable results sometimes seen, the use of vaccines is to be advised in all recent acute cases of the severe form. The prolonged use of irrigations has serious objections in girls of seven or eight years or older, in that it tends to develop sexual consciousness and may lead to masturbation.

On the whole, it must be stated that the results of treatment in cases which have reached the chronic stage by any measures yet proposed are very unsatisfactory, largely owing to the difficulty of controlling the patients for the tedious period of local treatment which is necessary.

Relapses are exceedingly common even in cases in which there has been no discharge for weeks or even months. Of twenty-six cases carefully followed up by Spaulding and subjected to thorough treatment, all but two relapsed after variable periods from one to six months.

That such cases are reinfections seems improbable. It would rather appear that the disease may have long periods of latency and recrudescence for an indefinite time. It is therefore difficult to say when a given case is actually cured. Under most conditions one is safe in pronouncing a case cured when there has been no discharge for three months after the discontinuance of special treatment, and when smears from the deeper parts continue to be negative.

GANGRENOUS VULVITIS (NOMA)

This is the same process as that seen in the mouth and known as cancrum oris. It usually follows one of the infectious diseases, most frequently measles, occurring in patients whose general vitality has been greatly reduced. There is first noticed a tense, brawny induration, the skin being shiny and swollen over a circumscribed area. In the center of this there soon appears, usually upon one of the labia majora, a dark, circumscribed spot. Day by day the gangrenous area advances, preceded by the induration. It may involve the whole labium, extending even to the mons veneris and the perineum. These cases are generally fatal. If recovery takes place, it is with considerable deformity of the parts in consequence of the extensive sloughing and cicatrization. As sequelae, there may be fistulae, stenosis, or atresia of the vagina. The only radical treatment is early excision, and the application of the actual cautery, carbolic or nitric acid.

CHAPTER IV

DISEASES OF THE BLADDER

ENURESIS

(*Incontinence of Urine; Bed-wetting*)

Enuresis may be due to some malformation of the genital tract, such as an abnormal opening of the bladder into the vagina, to extroversion of the bladder, or to the persistence of the urachus; in the latter case the urine is discharged from the umbilicus. It also occurs in organic diseases of the central nervous system, such as idiocy, cerebral palsy, acute meningitis, tumors of the brain, certain forms of myelitis, spina bifida occulta, and in injuries of the cord. In many of these conditions there is associated incontinence of feces. Both of the groups of cases mentioned are quite distinct from the ordinary form of incontinence of

urine which is seen in childhood. The latter is the only variety which will be considered here.

It is in many cases possible to teach infants to control the evacuation of the bladder before the end of the first year; usually, however, control is not acquired even during waking hours until some time during the second year, and in some healthy infants not before the end of the second year. The time depends very much upon the training. If a child during its third year can not control the evacuation of the bladder during its waking hours, incontinence may be said to exist.

Etiology.—Incontinence of urine may be due to a continuance of the infantile condition, to anything which increases the irritability of the spinal center, or which interferes with the cerebral control over this center, or to anything which increases the irritability of the terminal filaments of the vesical nerves or of those in the neighborhood. The causes of incontinence thus may be in the central nervous system, in the urine, in the bladder, or in any of the adjacent organs.

The causes relating to the central nervous system are in the main those of the other neuroses of childhood; these are anemia, malnutrition, an inherited nervous constitution, or a condition of extreme nervousness or neurasthenia, the result of the child's surroundings. In such cases incontinence is often associated with chorea, epilepsy, hysteria, headaches, neuralgia, and other nervous symptoms. In these conditions there is assumed to be not only an increased irritability of the nerve centers, but also of the peripheral nerves, accompanied by loss of tone of the vesical sphincter. A similar condition may exist with almost any form of acute illness, this usually, however, being only temporary.

Incontinence may be caused either by a highly acid, concentrated urine when an insufficient amount of fluid is taken, or by the opposite condition, when owing to the drinking of a large quantity of water, often only a matter of habit, the amount of urine is very greatly increased and passed at frequent intervals.

In the bladder itself, cystitis and vesical calculus, although infrequent, should not be overlooked as possible causes. In a few cases, where incontinence has existed a long time, the bladder becomes so contracted that it will hold only an ounce or two of urine. This condition, although not the primary cause of enuresis, may be enough to continue it.

Local irritation in the neighboring organs may be due to adherent prepuce, balanitis, phimosis, or to a narrow meatus. All of these conditions are frequently associated with incontinence. Rectal irritation may be due to pinworms, anal fissure, or rectal polypus; and vaginal irritation to vulvovaginitis or adherent clitoris; but these are rarely the only cause. Often there is incontinence as the result of a combination of several causes, no one of which alone would have been sufficient to produce

it. In many cases heredity seems to be a factor of some importance, parents often having suffered in their childhood from the same condition; quite frequently two and sometimes even three children in the same family are affected. In many cases the condition seems to be mainly the result of habit, and in all cases habit is a potent factor in continuing the incontinence, sometimes after the original exciting cause has been removed. Frequently no adequate cause can be found. Both sexes are about equally liable to enuresis; it may be seen in all ages up to puberty and even to adult life.

Symptoms.—Enuresis may be nocturnal or diurnal, or both. Of 184 cases, 73 were nocturnal, 9 diurnal, and 102 were both nocturnal and diurnal. Cases differ greatly in severity. Incontinence may be habitual, occurring every night, often several times during the night, and frequently during the day; or it may be only occasional under the influence of some special exciting cause, when it continues a few days or weeks until the cause is removed. In a considerable number of cases, the condition lasts from infancy until the sixth or seventh year. It may even continue until puberty; but it generally ceases at that period, unless its cause is mechanical or depends upon some organic disease of the brain or cord. In ordinary enuresis there is never dribbling of the urine, but usually a contraction of the walls of the bladder follows almost immediately upon the desire before the patient can make his wants known or reach a convenient place for micturition. At night the same thing may occur without wakening the child, the contraction being of purely reflex origin.

Prognosis.—The condition is usually hopeless when it depends upon organic disease of the brain and cord; also in cases due to malformation, unless these are amenable to surgical treatment. In the ordinary cases seen, the prognosis depends upon the age of the child, the duration of the symptom, and the nature of the exciting cause. In children of from three to five years a cure can in most cases be accomplished with proper management. Those who are older are much less amenable to treatment, especially if the condition has persisted since infancy; but if the incontinence has begun after seven or eight years of age and lasted but a few weeks or months, the outlook is much more encouraging. There are, however, some cases in which no other cause than habit can be discovered which resist all treatment, the condition finally ceasing spontaneously about puberty; rarely does it continue beyond this period.

Treatment.—The first indication is to remove the cause, when one can be found. If there are preputial adhesions, they should be broken up and irritating smegma removed. If phimosis is present, it should be relieved by circumcision. If stone in the bladder is suspected, as it should be when the incontinence is worse by day and accompanied by

straining and painful spasm of the bladder, the patient should be sounded for stone. Pinworms in the rectum should receive the appropriate treatment by injections. While the local conditions mentioned should always be attended to, the fact remains that few cases are cured simply by relieving them, except those due to vesical calculi. The explanation of this is that habit is the important factor in keeping up incontinence.

A concentrated urine of high acidity with deposits of uric acid is an indication for alkalis and the free use of all fluids, especially water. On the other hand, when there is passed a large quantity of urine of low specific gravity, the amount of water and other fluids should be greatly restricted. During the night water should be forbidden. In these cases the incontinence is often simply the result of the polyuria, which in turn depends upon polydipsia.

In most cases the condition is purely a habit, often associated with other habits which indicate an unstable or highly susceptible nervous system. It is therefore of the greatest importance that a proper general régime should be instituted. Care should be taken to secure for the child a simple, natural life, with no overtaxing of the nervous system at home or in school. Every cause of unnatural excitement should be avoided. Early hours and plenty of sleep should be insisted upon. Certain articles of diet are to be avoided, and coffee, tea, and beer should be absolutely prohibited. Sweets and all highly seasoned food should be very sparingly allowed, or not at all. The exclusion of meat from the diet seems to us to be of no special advantage. Measures directed toward improving the general muscular and nervous tone are of the greatest importance. Anemia, malnutrition, indigestion, and constipation should each receive careful attention.

Punishments, whether corporal or otherwise, do little good, and usually they are harmful. Rewards are sometimes more efficacious than any other means of treatment. One should first find out what it is that the child desires most—a new doll, a bicycle, etc.—and allow him to have it if the bed is dry, taking it away if it is wet. A reward of five cents for every dry night sometimes works marvels. Any measures that produce a marked impression upon the mind of the child sometimes have a beneficial effect. The inspiring of confidence that the physician will bring about a cure is oftentimes the most efficacious method of treatment. Bad-tasting drugs and mechanical measures, such as the passing of sounds, probably owe their occasional success to the mental impression that they produce.

After all local and general causes which can be discovered are so far as possible removed, there remains the large majority of the cases of enuresis in which the condition is simply the continuance of a bad

habit. To break the habit, training is of the first importance. The regulation of the amount of fluids is indispensable. Fluids should be given freely up to 4 P. M., but those who have nocturnal incontinence should have no fluids after that hour, a dry supper being given before retiring. These children are often heavy sleepers and the distention of the bladder does not produce a sufficient impression to waken them. Training should be begun during the day by voiding at regular intervals, and gradually lengthening the interval to accustom the bladder to distention. At night also the child should be wakened regularly at certain hours to void his urine. This should be done by an alarm clock if necessary; e. g., a child who is put to bed at 7 is at first wakened at 10 P. M. and at 1 and 4 and 7 A. M., a record being kept of the times when the bed is found wet. When he goes three hours regularly at night without voiding, the time is lengthened to three and a half and finally to four hours. A child can in this way usually be trained in a few weeks to hold his urine with but one waking from 10 P. M. until morning; and in a few months this can be omitted. The number of cases which can be permanently cured by such simple means is most surprising. The faithful coöperation of the mother or nurse is essential to make the cure permanent.

The measures described—removal of local causes, improvement of the general health, the institution of a proper régime and training—constitute the most important part of the treatment and in the majority of cases suffice for a cure. Drugs are at times useful as accessories; alone they seldom cure and, on the whole, they are disappointing. Belladonna is the most effective one. Atropin, either in solution or in tablet form, is the most convenient method of administration. For nocturnal incontinence, 1-1,000 of a grain for each year of the child's age up to seven years is a suitable dose. A child of five would thus be taking 1-200 of a grain. At first, a single dose should be given at bedtime; after a few days a second dose may be given three or four hours earlier; still later a dose may be given at 4 P. M., 7 P. M., and 10 P. M. To push the drug further than this may cause much discomfort and is of doubtful advantage. After the habit is under control, the drug should be continued for some time and the dose reduced.

Strychnin is sometimes advantageous when there is diurnal as well as nocturnal incontinence, for under these conditions there is usually a lack of tone in the sphincter, as well as increased irritability in the mucous membrane of the bladder. Full doses are necessary; beginning with 1-100 of a grain twice daily it may be gradually increased to 1-50 of a grain three times a day to a child of five. Intelligent, systematic training is the most important of all measures for the relief of this very annoying condition.

VESICAL CALCULUS

Vesical calculus is a very rare condition in children in New York. The nucleus of the calculus is usually a renal calculus which has passed the ureter, but has been prevented by its size from going farther. Stone in the bladder is extremely rare in infancy, probably owing to the fluid diet, but it is not infrequent in children from two to ten years of age. The most common variety of calculus at this time is the uric-acid.

The symptoms in children are somewhat different from those in adults, and the condition is often overlooked. There is frequently pain upon micturition, especially at the close of the act, which may be felt at the end of the penis or in the perineum. There may be a sudden stoppage in the flow of urine. The straining often leads to rectal tenesmus and even to prolapse. This complication is so frequent that, in a case of persistent prolapse, stone should always be suspected. Incontinence of urine is a prominent, and often the principal symptom; in many cases it is noticed only during the day. The urinary changes are not generally marked; hematuria is rare, and mucus and pus are infrequent and in small quantity. The genital irritation may lead to the habit of masturbation. A stone of any considerable size may often be felt by a bimanual examination, one finger being placed in the rectum and the other hand above the pubes. This is easier in males than in females, but it is not very trustworthy, and not conclusive when it gives a negative result. A positive diagnosis is made only by exploring the bladder with a sound or by the X-ray.

The treatment of calculus is purely surgical.

SECTION VII

DISEASES OF THE NERVOUS SYSTEM

CHAPTER I

The Weight of the Brain.—From ninety-eight observations made in the post-mortem room of the New York Infant Asylum, the following were the average weights noted:

At three months	21	oz.	(602 grams).
At six months	25½	"	(712 ").
At twelve months	32½	"	(916 ").
At two years	35	"	(990 ").

The following are the figures given by Boyd and Schäfer.

Age.	Males.		Females.	
	Ounces.	Grams.	Ounces.	Grams.
At birth (full term)	11½	330	10	283
Under three months	17½	500	16	450
From three to six months	21	602	20	560
From six to twelve months	27	776	26	727
From one to two years	33	941	30	843
From two to four years	39	1,110	35	990
From four to seven years	40	1,138	40	1,135
From seven to fourteen years	46	1,301	40½	1,154
From fourteen to twenty years	48½	1,374	44	1,244

At birth the weight of the brain to that of the body is nearly 1 : 8. During infancy and childhood the following is the ratio, according to Bischoff: during the first year, 1 : 6; the second year, 1 : 14; the third year, 1 : 18; at the fourteenth year, 1 : 15 to 1 : 25; in adult life it is 1 : 43.

The Spinal Cord.—The weight of the cord to the weight of the body at birth is 1 : 500; in adult life it is 1 : 1500. According to Kölliker, the spinal cord and the vertebral column are the same length until the end of the third month of fetal life, there being at this time no cauda equina. At the ninth month the lower end of the cord is opposite the third lumbar vertebra; in the adult it is opposite the first.

Some Peculiarities in the Diseases of the Nervous System in Infancy and Childhood.—The relatively large size, the rapid growth, and the immaturity of the brain and cord during early life, explain much that is peculiar to the nervous diseases of this period.

At this time, apparently trivial causes are enough to produce quite profound nervous impressions, because of the instability of the nervous centers and the greater irritability of the motor, sensory, and vasomotor nerves. These are conditions which are very much increased by all disturbances of nutrition. These disturbances may be manifold in character, but they lie at the root of very many of the neuroses of early life, e. g., extreme nervousness, disorders of sleep, stuttering, chorea, incontinence of urine, tetany, and convulsions. The great liability to convulsions depends not only upon the greater irritability of the peripheral nerves, but upon the instability of the nervous centers and the lack of inhibition over the motor ganglion cells of the spinal cord. The nervous centers are more easily exhausted than later in life.

Another peculiarity is the serious consequences which often follow reflex irritation, although this is rarely the only factor in the case. Conditions which in adult life produce almost no effect may in infancy be the cause of most alarming symptoms.

As a third point of importance may be mentioned the grave permanent results which often follow relatively small organic lesions. A good illustration is seen in the lesions which produce cerebral birth-palsy. Here the damage is only in small part the immediate effect of the hemorrhage, for this often is not great, but it is the interference with the development of certain parts of the cortex that makes the condition so serious.

From what has been said, it follows that the hygiene of the nervous system is of the utmost importance in infancy and childhood. It is essential for the healthy development of the nervous system that all stimulants should be avoided—not only tea, coffee, and alcohol, but undue and unnatural excitement, the effect of which in infancy is almost as serious. A normal development can take place only in the midst of quiet and peaceful surroundings, with plenty of time for rest and sleep. The conditions of modern life, especially in cities, are such that these laws are almost invariably violated, and the consequences of this are seen in the marked and steady increase in nervous diseases among children of all classes.

CHAPTER II

GENERAL AND FUNCTIONAL NERVOUS DISEASES

CONVULSIONS

All young children, but especially infants, are extremely prone to convulsive disorders. In certain infants, especially those who are rachitic, this susceptibility is much heightened.

Under the head of convulsions are included attacks of acute transient nervous disturbance, characterized by involuntary rhythmical spasm of the muscles, either of the face, trunk, or extremities, or all of them, usually accompanied by loss of consciousness. They may be regarded as "motor discharges" from the cortex of the brain.

Etiologically, convulsions may be divided into those of organic and those of functional origin according as to whether a pathological lesion is or is not demonstrable. It must not be overlooked, however, that what we now consider functional may, with improved methods, be shown to depend upon an actual change in the tissue of the brain. Under the head of organic, or those due to direct irritation of the cortex of the brain, may be included all convulsions occurring with the various forms of cerebral disease. The most frequent are meningitis, meningeal or cerebral hemorrhage, tumor, abscess, hydrocephalus, embolism, and thrombosis. Developmental defects of the brain, especially microcephalus, are frequently the cause of repeated convulsions that are usually classed under epilepsy. Convulsions due to organic disease may be found at any time during infancy and childhood. Because of their dependence upon traumatism at birth they are frequent in the first few weeks of life.

Convulsions functional in origin are, in the overwhelming majority of cases, dependent upon tetany which may be either active or latent. It is only in the last few years that this has been sufficiently recognized. As will be emphasized under Tetany, the symptoms of this disease and the irritation of the nervous system accompanying it are not usually manifest before the end of the first half year. For this reason, functional convulsions are much less frequent during the early months of life.

It has been held that the most important predisposing cause of convulsions in infancy is the instability of the nerve centers, which is dependent upon a lack of development of the voluntary centers of the cortex. It should be emphasized, however, that while convulsions of functional origin are exceedingly common in infancy, they are not so in the first three or four months of life when instability of the centers might be assumed to be the greatest. It is quite evident that the instabil-

ity depends not upon the normal insufficiency of cerebral development, but upon the acquisition of tetany, which causes cerebral instability.

It has long been held that convulsions were caused by materials absorbed from the gastro-intestinal tract. It is certainly true that overfeeding or indigestion may excite convulsions. This is usually, however, in children suffering from tetany and it is very likely that the convulsions are not due to any specificity of the material absorbed, but that any irritation to the child's nervous system is likely to be followed by convulsions. Convulsions are sometimes seen, it must be admitted, in infants when no evidence of organic disease can be detected, nor any symptoms of tetany and no hyperexcitability of the nervous system as shown by electrical examination. The cause of these is not clear.

Convulsions are apparently at times of toxic origin. They may result from conditions like uremia and asphyxia and also at the onset or in the course of various infectious diseases. They are more frequent in children who have or have had tetany, but may be found without any evidences of this. They are very frequent at the onset of certain diseases, particularly pneumonia, scarlet fever, malaria and acute indigestion. In pertussis, which, of all the infectious diseases, is the one in which convulsions are most frequent, several factors may be present: asphyxia, due to a severe paroxysm, cerebral congestion or hemorrhage resulting from such a paroxysm, or simply a peculiar susceptibility of the patient brought about by the disease itself. One attack of convulsions, whatever the cause, renders the patient more liable to a second attack and when there have been several, they occur from causes which are less and less marked.

An infrequent cause of convulsions in young children is an encephalopathy due to lead poisoning. We have seen four such cases, three of which were fatal. The poisoning was caused in each instance by the child's nibbling and swallowing the paint from his crib or furniture.

Convulsions ending fatally are not infrequently associated with enlargement of the thymus gland. We have seen many such where there was found at autopsy great enlargement of the thymus, and the lymphatic structures. Some of these infants were previously healthy; some were rachitic. The similarity of all these cases indicated that the convulsions were in some way associated with the enlarged thymus, but the exact explanation of such cases is not understood. In infants who die during convulsions the brain may be the seat of punctate hemorrhages, and sometimes of more extensive ones. The lungs are also deeply congested, and the right heart is generally distended with dark clots. The other lesions found are accidental.

Symptoms.—In some cases prodromal symptoms are present, such as extreme restlessness, irritability, slight twitchings of the muscles of the

face, hands, feet, or eyelids. More frequently, however, the attack comes quite suddenly with little warning. Usually the first thing noticed is that the face is pale, the eyes fixed, sometimes rolled up in their orbits; in a moment or two, convulsive twitchings begin in the muscles of the eye or face, or in one of the extremities, which usually rapidly extend until all parts of the body participate. In most cases the convulsions become general, but they may remain unilateral even when not due to a local cause—a point which is often forgotten. The contraction of the facial muscles causes a succession of grimaces; the neck is thrown back; the hands are clenched; the thumbs buried in the palms; and a quick spasmodic contraction of the extremities occurs. There may be some frothing at the mouth, and in all true convulsions there is loss of consciousness. Respiration is feeble, shallow, and may be spasmodic. The pulse is weak; it may be slow or rapid; often it is irregular. The forehead is covered with cold perspiration. The face is first pale, then becomes slightly blue, especially about the lips. Unnatural rattling sounds may be produced in the larynx. The bladder and rectum may be evacuated. The convulsive movements consist in an alternation of flexion and extension occurring rhythmically. All varieties of tonic and clonic spasm may be seen, and in all degrees of severity. The contractions of the two sides of the body are usually synchronous. After a variable time, from a few moments to half an hour, the convulsive movements gradually become less frequent, and finally cease altogether, usually leaving the patient in a condition of stupor. They may recur after a short time or there may be but one attack. A period of general relaxation usually follows the convulsive seizures, frequently accompanied by marked evidences of prostration. Transient paralysis, apparently due to exhaustion of the nerve centers, is not an uncommon sequel.

Death may take place from a single attack; this, however, is rare except in very young infants, or those with status lymphaticus. There may be no sequel to the convulsions if the cause is a temporary one, or they may produce some serious brain lesion, particularly meningeal hemorrhage. Death from convulsions is generally due to asphyxia, or to exhaustion from the rapidly recurring attacks. Many cases recover in which the children for several minutes had the appearance of being moribund.

One attack of convulsions is very apt to be followed by others, especially if tetany be the cause. The longer the interval which has passed, the less likely is there to be a repetition, especially if the child has passed his third year.

Diagnosis.—There can rarely be any difficulty in recognizing an attack of convulsions. The difficulty consists in determining with which of the many possible exciting causes we have to deal in the case before

us. If it comes with acute symptoms does it depend upon a cerebral lesion, or does it mark the onset of some other acute disease? Is it due to tetany? If there are no acute symptoms, is it epilepsy? To answer these questions a careful history must be obtained, and all the circumstances surrounding the patient, the character of the convulsions, and all the other symptoms present must be taken into consideration. Tetany is easy to recognize if there is carpopedal spasm, Chvostek's sign, laryngospasm, or Trousseau's sign. If these are absent, it can only be determined by the electrical reactions. Tetany is to be considered the most likely cause, however, in the absence of the evidence of organic cerebral disease.

In infancy, epilepsy is the least probable diagnosis. In older children the important points indicating that disease are: a history of previous attacks, a distinct aura preceding the seizure, or a sudden onset with a cry or fall, biting of the tongue, a deep sleep following the seizure, and, finally, perfect recovery in the course of a few minutes or hours. Convulsions which come on with high fever, even though a patient may have repeated attacks, are seldom epileptic. However, in some cases only prolonged observation can enable one to decide positively whether or not epilepsy is present.

Convulsions occurring in brain disease, except acute meningitis, are not as a rule accompanied by any marked rise in temperature. Focal symptoms are often present, such as localized paralysis or rigidity, changes in the pupils, and strabismus. The convulsive movements are frequently limited to one side of the body. It should, however, be borne in mind that unilateral convulsions, even when repeated, do not always mean a local lesion, as we have seen proved by autopsy more than once. In hemorrhage or meningitis, convulsions are likely soon to recur. In tumor they may recur after a longer interval.

Convulsions may be thought to indicate the onset of some acute disease when they occur in a child over two years old, and when they come on suddenly or with only slight premonition in a child previously well; but the most important point is that they are accompanied by a high temperature—104° to 106° F. Acute meningitis is the only other condition likely to produce these symptoms. Whether the convulsions mark the onset of lobar pneumonia, scarlet fever, or some other disease, can be determined only by carefully watching the patient's symptoms for twenty-four or thirty-six hours or possibly longer.

In the first weeks of life one may often be in great doubt as to the cause of convulsions. Such attacks may be due to some disorder of the digestive tract, to a recent cerebral lesion like hemorrhage, or to a defective brain development. Apparently prolonged pressure in a difficult labor may produce temporary, perhaps circulatory, changes in the brain

sufficient to cause convulsions during the first few days of life. We have seen them in a number of children whom we have had an opportunity to follow for several years. Their physical and mental development has progressed in a perfectly normal manner.

Examination of the urine should not be omitted in any case of convulsions of doubtful origin. Asphyxia may be suspected in the case of convulsions occurring in the newly born, late in pneumonia, in some cases of pertussis, in spasmodic or membranous laryngitis, or with laryngospasm. It is altogether improbable that dentition and worms play any part in the causation of convulsions except perhaps that of the slight irritant which is sufficient to excite convulsions in a child suffering from tetany.

Encephalopathy due to lead should be kept in mind as a rare cause of convulsions in children. The blue punctate line in the gums can usually be found, though not around each tooth. There is also stippling of the red blood-cells. The cerebrospinal fluid is under increased pressure, the cells are slightly increased in number and there is a positive reaction for globulin. There is frequently pallor of the optic discs and hemorrhages into the retina may be seen.

In all cases of convulsions occurring in infants in which the cause is not readily apparent, tetany should be suspected as the underlying condition.

Prognosis.—This depends upon the cause of the convulsions, and differs with each underlying cause. In general it may be said that convulsions in themselves are seldom fatal unless they occur as a terminal condition. Especially fatal are the convulsions of pertussis and of asphyxia when they occur late in any form of laryngeal or pulmonary disease. The conditions during an attack which should lead one to make a bad prognosis are when the convulsions are prolonged or recur frequently; also the presence of very great prostration, a feeble pulse with cyanosis, or deep stupor.

In the prognosis one must take into account not only the immediate result of the attacks, but the possible outcome. In a highly nervous or susceptible child a convulsion often means very little. Permanent injury to the brain, simply as a result of an attack, is very rare. The possibility of epilepsy is to be borne in mind in all cases where children over two years old have occasional attacks of convulsions. The farther apart the attacks are and the more definite the exciting cause, the less likely is this to be the case.

Treatment.—Summoned to a child in convulsions, a physician should go at once and remain until the attack has subsided. He should take with him chloroform, a hypodermic syringe with morphin, a soft catheter or rectal tube, and a solution of chloral. In order to treat convul-

sions intelligently one must have in mind the prominent pathological conditions. These are acute cerebral hyperemia, a more or less severe asphyxia with pulmonary congestion, an overtaxed right heart, and a tendency to congestion of all the internal organs. The nervous centers are in a condition of such unnatural excitability that the slightest irritation may bring on convulsive movements when they have temporarily subsided. The patient should therefore be kept perfectly quiet, and every unnecessary disturbance avoided. Cold should be applied to the head—best by means of an ice cap or cold cloths—and dry heat and counter-irritation to the surface of the body and extremities. The time-honored mustard bath causes so much disturbance of the patient that it can usually be dispensed with and the mustard pack substituted. The feet may be placed in mustard water while the child lies in his crib. The mustard pack and footbath should be continued until the skin is well reddened. The degree to which counter-irritation of the skin should be carried will depend upon the condition of the pulse and the cyanosis.

In controlling convulsions the remedies which may be depended upon are the inhalation of chloroform, chloral per rectum, morphin and magnesium sulphate hypodermically. Chloroform is undoubtedly the most reliable remedy for an immediate effect, and may be used even in the youngest infant. At the same time that it is being administered, chloral may be given. The initial dose should be, at six months, four grains; at one year, six grains; at two years, eight grains, dissolved in one ounce of warm milk. It should be injected high into the bowel through a catheter, and prevented from escaping by pressing the buttocks together. It may be repeated in an hour if necessary. The effect of the drug is generally obtained in twenty or thirty minutes. If, in spite of the chloral, the convulsions show a marked tendency to continue as soon as the chloroform is withdrawn, or if the enema of chloral has been expelled, morphin may be given hypodermically. When the heart's action is weak, this is probably the best of all remedies. To a well-grown child two years old, $\frac{1}{16}$ grain may be given; one year old, $\frac{1}{20}$ grain; six months old, $\frac{1}{40}$ grain. This dose may be repeated in half an hour if no effect is seen. The tolerance of opium in cases of convulsions is very marked, and sometimes double the doses mentioned may be required. For frequently recurring convulsions magnesium sulphate, hypodermically, is a valuable remedy. It has the advantage over morphin in that it does not constipate. Eight or ten grains of Epsom salts may be given to an average infant of three or four months, and from fifteen to twenty grains to one of six or eight months. It does not act so promptly as does morphin. The dose may be repeated in two hours if necessary. The only other agent of much value is oxygen. We have occasionally seen convulsions which continued in spite of all other treatment yield imme-

diately to oxygen. This is most likely to be valuable in cases of convulsions due to asphyxia.

In infancy it is wise in every case to irrigate the colon thoroughly with warm water, to remove any possible source of irritation. If there is reason to suspect the presence of undigested food in the stomach, this may be washed out. Much more frequently it is in the intestines, and free purgation by calomel is advisable. If there is high temperature, this should be reduced by the cold bath or pack.

When once under control, the recurrence of the convulsions may be prevented by keeping the patient for two or three days under the influence of chloral with bromid of sodium, the amount of chloral being gradually reduced. If it is badly borne by the stomach and not easily retained by the rectum, either antipyrin or phenacetin may be used with the bromid. As soon as the convulsions have ceased, the cause should be sought and treated.

TETANY

Several clinical conditions, formerly described under different names, are now regarded as manifestations of tetany: *arthrogryposis* or carpopedal spasm, *laryngismus stridulus* or laryngospasm, holding-breath spells, etc.

Tetany is a disease characterized by an extreme irritability of the nervous system to mechanical and electrical stimulation. It is frequently accompanied by more or less prolonged contractions of the muscles of the extremities. Spasm of the glottis and also general convulsions are very common. It was formerly believed that tetany was rather infrequent and was manifested only by muscular spasm. Studies by electrical methods, however, have shown that in infancy and childhood the disease is exceedingly frequent and that it may exist without giving any symptoms, i. e., in a latent form. To the latent form of the disease as well as to all the manifestations, the term "spasmophilia," or "spasmophilic diathesis," has been applied by many.

Etiology.—While tetany is found with the greatest frequency during the latter half of the first and during the second year, it is very rarely seen in the first three months of life. It may occur at any time during childhood but its frequency diminishes rapidly with age. Tetany is rare in summer and early autumn, but it is very common in winter and early spring. The association of tetany with rickets is a very close one. Not only is it found at the time of year when active rickets is most common, but almost all children with tetany show some of the symptoms of rickets. While cases are observed in which no rachitic manifestations are present, rickets cannot be entirely excluded, for, as has been stated

elsewhere, the first evidences of rickets in the bones escape clinical observation. Symptoms of both rickets and tetany begin to be seen at about the same age. While tetany may occur in the breast-fed, this is relatively infrequent. The disease evidently depends for its development largely upon artificial feeding but occurs even when this has been apparently proper.

Tetany seems to be closely connected with changes in the calcium metabolism, although these are not yet entirely clear. It has been shown in a certain number of patients that with active tetany, just as with active rickets, there is a negative calcium balance—more calcium being eliminated than is ingested with the food. There has also been found post mortem a deficiency in the calcium content of the brain. Mariott and Howland have demonstrated a marked reduction of the calcium of the blood of infants with active tetany. MacCallum and Voigtlin have shown a deficiency of calcium in the blood of animals with experimental tetany. It is therefore clear that there is some alteration of calcium metabolism in tetany.

The removal of the parathyroids in animals and the occasional accidental injury of these in human surgery produces a condition closely akin to tetany. The work of Erdheim, Escherich and Yanase indicated that the parathyroids might be diseased in tetany, the changes consisting in hemorrhages and their remains. Later observations have shown that these alterations may be found in children who, during life, have given no evidence of tetany and also that the glands may be normal when definite tetany has been present. It is as yet impossible to say whether the parathyroids play an important part in the disease. There is, however, sufficient evidence to indicate that they may have some influence upon its production. Tetany is at times hereditary. There may be a history of the disease in one of the parents and occasionally families are found with several children who have suffered from tetany. Acute disease, especially when accompanied by fever, is sometimes the exciting cause. It must be assumed that up to the onset of the acute disease tetany has been latent, the new condition providing the necessary irritation to make the tetany active. Thus, tetany is seen with acute diseases of the gastro-intestinal tract, pneumonia and the acute infectious diseases.

There are no characteristic pathological changes other than those of the associated rickets. In a certain proportion of the cases alterations in the parathyroids are found. One or more of the four glands may be enlarged and red as a result of extravasation, or the changes may only be evident under the microscope and consist in small hemorrhages, and the remains of hemorrhages.

Symptoms.—One of the most characteristic and striking is carpopedal spasm. It is, however, by no means the most common manifesta-

tion, and is seen in only a small percentage of the cases. The spasm of the hands and feet may develop abruptly, or it may be preceded by sensory disturbances. The upper extremities are usually first affected and both sides equally. The position is very characteristic: The fingers are

Fig. 86.—Tetany, showing the Characteristic Position of the Hands and Feet. In a child two years old.

flexed at the metacarpophalangeal joints and the phalanges extended; the thumbs are adducted almost to the little finger; the wrist is flexed acutely and the hand drawn somewhat to the ulnar side. If the spasm is very marked no motion is allowed at the wrist. The feet are strongly extended, sometimes in the position of equinovarus. The first phalanges

of the toes are flexed, and the second and third rows extended; the plantar surface is strongly arched and the dorsum of the foot is very prominent, standing out like a cushion. The typical position of the hands and feet is well shown in Fig. 86. Motion at the elbow, shoulder, hip and knee is generally free. The spasm in many cases is limited to the hands and feet; more rarely the muscles of the thigh, usually the adductors, may be involved. In rare cases the muscles of the trunk or the face may be affected. The spasm can be voluntarily overcome to a certain extent; thus a child may open his hands to grasp objects or feed himself. As soon as active motion ceases, the hands resume their former characteristic attitude.

Evidences of pain are frequent; it may be so severe as to cause children to cry out. Pain may be induced by any attempt to overcome the spasm, and sometimes it is constant. There is no loss of consciousness and no fever. The duration of carpopedal spasm may be from a few hours to several days. The muscular contraction is generally continuous, although there are often periods of remission. There may be only a single short attack. Of this we have seen several striking instances. One child seven years old who had always been well was operated upon for enlarged tonsils. The night following operation she cried out with pain and her hands and feet were found in the typical position of tetany. In four or five hours this completely disappeared and did not return. This was the only symptom of tetany that she ever manifested. Carpopedal spasm may come on spontaneously but is more frequently found in the course of some febrile illness. It is found in no other disease and is diagnostic of tetany.

Disturbances of respiration are exceedingly common in tetany. The most typical of these is spasm of the glottis or laryngospasm. This consists in a contraction of the laryngeal muscles of such intensity as partially to obstruct inspiration or for a time to arrest it. When the obstruction is partial there is a very characteristic crowing sound with each inspiration, especially if the child is disturbed or crying. There may be a succession of these sounds, followed by an intermission, or the condition may last in a mild form for several minutes or hours. The severe attacks of obstructed respiration usually come on suddenly. The child throws back his head, the face becomes pale, then livid, and for the time there is complete arrest of respiration. This continues for a few moments, during which the cyanosis deepens, and the child seems in great distress, making violent efforts to breathe. If the paroxysm is very severe, the asphyxia may be so great as to lead to loss of consciousness, or the attack may terminate in general convulsions. It may even be fatal. In less severe attacks, after fifteen or twenty seconds the muscular spasm relaxes, the glottis opens, and a long, deep inspiration occurs, with the production

of a crowing sound. Such forms of spasm often come on without evident cause, and may be repeated from two to twenty times a day. Between them the condition of the child may be normal or carpopedal spasm and other evidences of tetany may be present. Not all the paroxysms are equally severe. A child may have in the course of a day a great many mild attacks, but only a few severe ones. General convulsions are seen in over one-third of the severe cases. Laryngospasm is most common in children from six to fifteen months of age.

Attacks closely related to those which have just been described are met with in which respiration entirely ceases for a time; there are temporarily no attempts at inspiration. It has been assumed that the diaphragm participates in the spasm. Attacks with temporary arrest of respiration are seen most frequently in the latter part of the first and during the second year, but beginning in infancy they may recur from time to time until the age of four or five years. They affect children of an extremely nervous type. Several attacks may occur in a single day, or they may occur at intervals of several days or weeks. In susceptible children almost any form of excitement may precipitate one. They are often known as "holding-breath spells." In older children by far the most frequent exciting causes are temper and fright. If anything is attempted to which the child strongly objects, e. g., a cold bath, inspection of the throat, or taking away a toy, an attack may ensue. The child's face becomes flushed, then livid; there is general rigidity of the trunk and extremities, but very rarely clonic spasms. This rigidity is usually followed by complete relaxation with loss of consciousness. The entire attack usually lasts about half a minute. There may be a crowing sound as the child catches his breath or there may be none. After a few minutes of quiet the child gets up and in a short time is apparently as well as ever. Many of those who are subject to attacks of this sort sooner or later have one or more general convulsions, but in some only the mild attacks are seen though they may recur at intervals for years. Death occasionally occurs with severe attacks, there being no renewal of respiration and all attempts at resuscitation failing.

Lederer has described a complex of pulmonary symptoms closely simulating asthma. This he has termed *broncho-tetanie*. It is not clear that the symptoms which he describes are necessarily dependent upon tetany.

General convulsions are exceedingly common with tetany in infancy. After that they are less frequently seen. They differ in no respect from those that have been described in the previous chapter. The more frequent the convulsions, the milder they usually are. From the character of the convulsions alone, it is impossible to differentiate them from epilepsy. They may occur without any exciting cause or the least stimu-

lus may be sufficient to cause an attack. Thus we have seen a child who repeatedly had convulsions whenever cold was applied to the skin. The number of attacks may be very great. In one case that we saw, an infant during the latter part of his second year had, during six months, over 3,500 distinct attacks of convulsions. For a considerable period they reached the almost incredible number of 80 a day. After improvement occurs, the number may gradually diminish or more frequently they may cease almost at once. Death is infrequent during a convulsion but occasionally occurs, apparently from exhaustion, when severe convulsions are frequently or uninterruptedly repeated.

When tetany is suspected, three confirmatory signs should be sought: Chvostek's sign or the facial phenomenon, Trousseau's sign, and Erb's sign. *Chvostek's sign* consists in a momentary contraction of the muscles of the face when a branch of the facial nerve is tapped with the percussion hammer or with the finger. The nerve may be tapped anywhere, but usually best about the middle of the cheek. The contraction may affect only the mouth and the alae nasi, or it may involve any of the muscles supplied by the nerve. This sign is not found in the first two years of life, except in cases of tetany. Later, it is of more frequent occurrence and less reliance can be placed upon it as an evidence of tetany, particularly after the fifth year. Thiemich, however, maintains that it always indicates tetany. But it is found in such a large proportion of older children in whom no symptoms or history of tetany can be obtained that it is generally believed to indicate in them only a neurotic constitution.

Trousseau's sign is elicited by pressure by the hand or a bandage upon the blood vessels of an extremity with sufficient force to stop the circulation temporarily. The sign is most easily elicited in the upper extremity when pressure is made above the elbow. The radial pulse should be obliterated for several minutes. Then the hand may assume the typical position of carpopedal spasm. The sign is often absent in well-marked tetany, but when present is to be regarded as positive evidence of tetany.

Erb's sign or the quantitative reaction of the nerves to the galvanic current.[1]—Muscular contractions are produced by the application of the

[1] For the electrical determinations a galvanic battery with a milliampèremeter graduated in fifths up to five milliampères is necessary. The measurements are usually made upon the peroneal nerve. The large indifferent electrode should be placed upon the abdomen, the stimulating electrode upon the peroneal nerve in the outer part of the popliteal space near the head of the fibula.

The *cathodal closure contraction* is often obtained with a current less than 5 milliampères in strength in normal children under six months of age, and after this time it is regularly present with a current of this strength or a weaker one. No evidence in regard to tetany may be obtained from the C.C.C. The *anodal*

galvanic current to the nerves. These contractions occur with the making or breaking of the current and are called "closing" and "opening" contractions, respectively. The nerves react differently to the different poles and also to the making or breaking of the current. Age also has an important influence in the character of the electrical response. The nerves of the newly born and of infants during the first year are less responsive to the current than those of children who are older. The excitability increases with age up to about five years, after which there is little if any difference between the child and the adult. Closing contractions occur in early childhood with a weaker current than do opening contractions.

In the first six months of life any contraction with a current of less than 5 milliampères, except that of cathodal closure, points to tetany; while an opening contraction, either cathodal or anodal, with a current weaker than 5 m. ap. is positive evidence of tetany.

Under two years of age an A.O.C. with a current of less than 5 m. ap. and weaker than one which will cause an A.C.C., is presumptive but not positive evidence of tetany. C.O.C. or C.C. tetanus with a current of less than 5 m. ap. in a child under five may be considered hyperexcitability due to tetany. Repeated measurements upon the same child often give different results in the course of a few days. For this reason several electrical examinations are frequently necessary to determine or exclude tetany.

---

closure usually requires more than 5 m. ap. of current with infants less than six months of age. From that time up to two years the A.C.C. is frequently, and after two years regularly, obtained with a current less than 5 m. ap. strength. An A.C.C., therefore, with a current of less than 5 m. ap. is suggestive of tetany only in the first six months.

The *anodal opening contraction* in the first six months of life occurs with normal children only with a current of more than 5 m. ap. strength and up to two years it almost always requires a current of more than this. It also usually requires more current to produce an A.O.C. than an A.C.C. until the second or third year. After five years of age the A.O.C. is regularly obtained with a current of less than 5 m. ap., and less than is required to produce an A.C.C. An A.O.C. therefore in the first six months of age obtained with a current less than 5 m. ap. is strong evidence of tetany and under two years of age is suggestive of tetany, especially if the A.O.C. takes place with a current less than is required to produce an A.C.C. This was called by von Pirquet "anodal hyperexcitability." We cannot regard it as more than highly suggestive of tetany after six months of age, for it sometimes occurs with children that are apparently entirely normal. After two years of age it is often present and after five years of age regularly so with normal children.

A *cathodal opening contraction* or *cathodal closing tetanus,* occurring with a current of less than 5 m. ap. in children under five years of age, is positive evidence of tetany. After that time such values may occasionally be found with quite normal children.

The conception of "latent" tetany was gradually reached when it was appreciated that muscular spasm of the extremities, laryngospasm and general convulsions were all symptoms of the same basal disorder. The electrical reactions also were shown to be in many instances the same in children that had suffered from no spasmodic symptoms, as in those who were the subjects of frank tetany. If the former were followed carefully it was often noticed that, sooner or later, convulsions, laryngospasm or carpopedal spasm developed. It is therefore apparent that there is an instability of the nervous system that, without electrical measurements, may exist unsuspected until suddenly it becomes clinically evident. Electrical measurements upon a large number of children in hospital and out-patient practice have shown that latent tetany is a frequent condition and that undoubtedly only a small percentage of these children show symptoms by which the disease is recognizable.

Various other symptoms have been ascribed by writers to tetany. Thus, Ibrahim has emphasized spasm of the pylorus producing vomiting, of the intestines, causing pain and meteorism, and of the anal sphincter leading to obstinate constipation. The occasional retention of urine in tetany has been referred to spasm of the vesical sphincter. The fatal outcome in some cases of general convulsions or those with laryngospasm it is claimed results from tetany of the cardiac musculature. The relation of all of these conditions to tetany is very doubtful.

From what has been stated it is evident that the variations in the course of the disease may be extreme. Tetany may entirely escape observation or it may give symptoms for months or even years. There is a surprisingly close connection between the condition of the bowels and the symptoms of tetany. In most patients tetany is aggravated by the existence of constipation. A sharp attack of diarrhea or free purgation by medicine regularly causes a diminution and often a complete disappearance of all symptoms including the abnormal electrical irritability. As the result of dietetic treatment, a marked diminution in the intensity and frequency of the attacks may be observed. They often cease altogether in a short time. Other cases are observed, however, in which improvement is very slow. In those children that suffer from malnutrition a proper growth and gain in weight may be difficult to obtain.

Diagnosis.—This may be easy or so difficult as to be possible only after prolonged observation. Carpopedal spasm, laryngospasm, Trousseau's sign and Chvostek's sign under five years, are pathognomonic symptoms. But in perhaps the largest number of children with tetany none of them is present. The electrical reactions are usually conclusive, but at times may be of little assistance. If an infant with no evidences of an organic brain lesion has repeated attacks of convulsions tetany should

always be suspected. If there are symptoms of rickets and if the attacks are frequent the probabilities of tetany are greatly increased. The chief difficulties in diagnosis are with older children who suffer from occasional convulsions. It may be almost impossible without prolonged observation to decide between epilepsy and tetany. Electrical reactions at this age offer little assistance. The older the child the greater are the chances in favor of epilepsy.

Prognosis.—The prognosis of tetany varies greatly with the age of the patient, the type of the disease and its severity. The prognosis of latent tetany is always good, with proper treatment. In general, the younger the patient the more severe the manifestations of tetany are likely to be and the more difficult to control. After two years, except in markedly rachitic children, the prognosis as to life is always good. The chances are always in favor of recovery when there are only occasional attacks of general convulsions. With frequently repeated convulsions there is danger to life, not only from the convulsions themselves, but from the frequent association of severe attacks of laryngospasm. This must always be looked upon as a dangerous manifestation of tetany and infants may die during such attacks.

Tetany complicating gastro-intestinal or any acute infectious disease makes its prognosis less favorable. According to Thiemich and Birk, the mental development of children who have suffered from severe tetany is often greatly retarded and, in many cases, permanently interfered with. The physical development also suffers. More observations are required definitely to settle this point. It is apparent, however, that tetany may leave permanent effects.

Treatment.—Prophylaxis should be emphasized. Tetany does not often occur with breast feeding. Maternal nursing is not only the best preventive, but feeding with woman's milk is also the best means of stopping the further progress of tetany when it has once developed. It does not, however, rapidly cure the disease. With infants under eight months of age who give symptoms of tetany woman's milk should be supplied if possible. Treatment should be directed not only against the manifestations of tetany but also against the fundamental metabolic disturbance upon which they depend. The treatment of this basal condition is the treatment of the associated rickets. It differs in no respect even though we recognize that the two conditions are not similar. This has been discussed in the Chapter on Rickets. The only exception to this general statement is that during the presence of attacks of convulsions and laryngospasm or carpopedal spasm it may be advisable to remove all cow's milk temporarily from the food. While it is true that overfeeding with cow's milk apparently causes and certainly aggravates tetany, in the event that breast feeding is impossible, cow's milk cannot

be altogether removed from the diet, except for a short period. There is no advantage in excluding it for a long period. The most satisfactory results are generally obtained when feeding is carried on according to the indications afforded by the child's digestive symptoms. There is a distinct advantage in providing a mixed diet with a minimum amount of milk as soon as the child's digestion will allow it.

The specific treatment of tetany by parathyroid extract has not been followed by any appreciable benefit, nor has the administration of calcium in our hands given favorable results. There can be no doubt that the prolonged administration of cod-liver oil and phosphorus is beneficial in a certain number of cases. They are to be used as in rickets.

General convulsions are to be treated according to the methods given in the previous chapter. Chloroform, chloral, morphin and magnesium sulphate are all useful and are to be employed for rather different indications. In an average case in an infant the last mentioned remedy is to be preferred. It is given subcutaneously in doses mentioned under Convulsions. If the convulsions are frequent it is advisable to withdraw cow's milk from the diet entirely for a time. Gruels may take its place for several days. When milk is again included in the diet it should be added very gradually and in minimum amount.

Laryngospasm, if severe, requires the administration of calcium bromid by mouth or chloral by rectum until the frequency and severity of the attacks are controlled. Antipyrin at times seem to be more effective than bromid or chloral. If during attacks there are no efforts at inspiration, artificial respiration should be performed and possibly intubation may be of value. The dietetic treatment should also be the same as when general convulsions are severe. Carpopedal spasm is often relieved by prolonged warm baths or by the application of warm compresses. Bromids, chloral or antipyrin are also to a certain extent useful in relaxing the spasm. Latent tetany requires no treatment other than the general dietetic and hygienic treatment directed toward the correction of the basal disturbance of metabolism.

EPILEPSY

Epilepsy cannot be considered a sharply limited disease. Rather it is to be looked upon as consisting of certain symptom-complexes that are frequently repeated and arise as the result of widely different causes, some known and some unknown. Moreover, these symptom-complexes are to a certain extent interchangeable. Epilepsy is manifested by repeated general or localized muscular spasm with or without loss of consciousness and by peculiar mental states, the so-called "equivalents."

A distinction must be made between cases of so-called "idiopathic"

epilepsy, or those without gross anatomical basis, and those which are secondary to a definite lesion of the brain, such as tumor, sclerosis or abscess. Convulsions of the latter character are designated as "symptomatic" epilepsy, and are discussed in connection with the various diseases in which they occur. The nature of the attack may, however, be identical in both varieties, and may not differ from an ordinary attack of convulsions or eclampsia. The proportion of idiopathic cases in children is not so large as was formerly supposed; many of these have been shown to depend upon lesions once overlooked, particularly mild infantile cerebral paralyses.

Etiology.—From a consideration of 1,450 cases of epilepsy, Gowers states that 12 per cent begin in the first three years of life, and 46 per cent between ten and twenty years. The greatest tendency to the development of the disease is shown about the time of puberty. Females are rather more liable to be affected than males, although the difference in sex is slight. Heredity plays the most important rôle in the production of the disease. It is estimated by various authors that from 35 to 65 per cent of epileptics come from epileptic families. Echevierra investigated the families of 135 epileptics and found that of their 533 children, 78 were epileptic and that 126 manifested various forms of nervous and mental diseases. The influence of alcoholism in the parents upon the production of epilepsy cannot be estimated with certainty. It is hardly to be doubted that it is a factor of importance in at least a certain percentage of cases. Syphilis also must be looked upon as the cause of some of the cases. Whether, in the absence of definite anatomical lesions, it so affects the brain as to lead to epileptic seizures cannot be stated at the present time. Further studies with the assistance of the Wassermann reaction are necessary to decide this question.

It was formerly believed that infantile convulsions were not infrequently followed by epilepsy in later years. There are numerous causes for convulsions in infancy. By far the greatest number not due to organic brain disease depend upon tetany. Not sufficient time has elapsed nor sufficient observations been made since the more recent knowledge of tetany to say whether it is likely to induce epilepsy. There is no good reason, however, to suppose that it does. Convulsions in infancy that are followed by epilepsy are probably epileptic from the beginning.

An innumerable number of other causes have been suggested, such as autointoxication from the intestinal tract, worms, adenoid vegetations of the pharynx, phimosis, masturbation, etc. That poisons absorbed from the intestinal tract can cause convulsions is probably true, but that epilepsy results in this way is very much to be doubted. The influence of the other factors suggested awaits any definite proof.

Pathology.—If one includes in the pathology of epilepsy the symp-

tomatic cases the changes in the brain are striking and of the greatest variety. These, however, do not concern us here. There has been much written and many careful observations made upon the changes in the so-called idiopathic cases. While it is perhaps true that, with improved technic and new methods, more definite and conclusive alterations in the brain will be found, it must be admitted that at the present time in the opinion of very competent authorities certain alterations can be demonstrated in the majority of instances. These are chiefly lesions in the cortex that can only be observed microscopically. A generalized gliosis has been described by Bleuler, Alzheimer and Chaslin. Meynert has observed a sclerosis in the cornu ammonis and Redlich and others have demonstrated various degenerative changes in the ganglion cells as well.

It seems probable that a great variety of lesions, many of which are apparently slight, may produce this disease.

Symptoms.—Two distinct types of epileptic seizures are met with: the major attacks, or *grand mal*, in which there are severe convulsions lasting from two to ten minutes, with loss of consciousness, etc.; and minor attacks, or *petit mal*, in which the convulsive movements are slight and may be absent, and in which the loss of consciousness is often but momentary. Between these two extremes all gradations are seen.

Grand Mal.—The onset may be sudden, without premonition, or it may be preceded by certain prodromal symptoms known as the aura. The aura may be motor, such as a local spasm of the hand, face, or leg; or sensory, such as numbness and tingling in any part of the body, or some abnormal sensation rising gradually to the head, at which time loss of consciousness occurs. The variety of sensations described by patients as indicating an attack is endless. There may be a sensation in one finger, in the face, tongue, eye, or in any part of the body; or the warning may be of a general character, like a tremor or a shivering sensation, or a feeling of faintness. There has also been described a visceral or pneumogastric aura, in which there is epigastric pain, sometimes nausea, and a sensation of a ball in the throat; or there may be palpitation, or cardiac distress. There may be general giddiness or vertigo, or a sensation of fulness in the head; or feelings of strangeness, or a dreamy, dazed condition; and, finally, the aura may have reference to any of the special senses, most frequently to sight. Sparks may appear before the eyes, or flashes of light or color, or strange objects may be seen; or there may be a momentary loss of hearing; or strange sounds may be heard. In most cases the aura is peculiar to the individual.

At the beginning of the seizure the face becomes pale, the pupils widely dilated, the eyes rolled up in their orbits and fixed. Speedily there is loss of consciousness. Simultaneously with these symptoms, or immediately following them, there occurs a violent tonic muscular spasm

to which are due the characteristic symptoms of the early part of the seizure, viz., the fall, cry, biting of the tongue, cyanosis, and evacuation of the bladder or rectum. The fall is forcible, violent; in fact the patient is precipitated, usually forward, and frequently suffers injury, never sinking down as in a faint. The head is often strongly rotated to one side. The position of the hands is frequently that assumed in tetany. The cry is a hoarse, inarticulate sound, not very loud, and is due to forcible expiration, owing to spasm of the muscles of respiration with the glottis partially closed. The cyanosis is the result of tonic spasm of the muscles of respiration; it may be quite intense, so that the face is livid, bloated, and the features distorted. The spasm of the muscles of mastication causes the biting of the tongue. Evacuation of the bladder and rectum may result from contraction of their walls, or from spasm of the abdominal muscles. The violence of the muscular spasm in this stage may be very great; it has caused fracture of bones, rupture of muscles, and even dislocation of joints.

The stage of tonic spasm may be only momentary, the patient passing almost at once into the stage of clonic convulsions. The usual duration is from ten seconds to half a minute. In the stage of clonic spasm which follows, the symptoms are those of an ordinary attack of convulsions. The muscular contractions are violent, and there is often frothing at the mouth. Gradually the muscles of respiration relax, air enters the lungs, and the cyanosis passes off. After the clonic spasm has continued for a variable time—from two to three minutes to half an hour—the muscular contractions become less and less frequent, and finally cease altogether. In a few minutes the patient may regain consciousness, look vacantly around, and in a dazed way perhaps ask what has happened, he being completely oblivious to all that has occurred. More frequently, however, he passes at once into a deep sleep, which continues for an hour or more, but from which he can be aroused. From this he usually wakens with a severe headache, which may continue for several hours. After this he often feels better than for several days preceding the attack. During the seizure the temperature may be elevated one or two degrees, but rarely more. The attack may be followed by a slight temporary paresis, aphasia, hysterical phenomena, vomiting, and intense hunger. In very rare cases the urine may contain a trace of sugar.

Petit Mal.—The minor attacks of epilepsy may present a very great variety of symptoms, and at times it is almost impossible to decide that these are epileptic, except from their periodical occurrence. They pass under the names of "spells," "attacks of dizziness," "fainting turns," etc. In recent years the term "absences" has been employed to designate them. The most striking thing which stamps them as epileptic is the

loss of consciousness, and this may be of short duration, sometimes only momentary, and so pass unnoticed; in some cases there is none. There is no fall, but there may be a slight dropping of the head, a fixed stare for a moment or two, and that is all. The muscles are often firmly fixed so that the child stands straight and stiff. Occasionally there are one or two contractions of the arms or a violent bending forward or nodding movement. These attacks may or may not be preceded by aura. After such a mild attack the patient's mind may be somewhat confused or he may become sleepy. One of the most striking things about attacks of petit mal is the frequency of their repetition. There may be as many as thirty or forty attacks a day. Petit mal is a serious form of epilepsy and after a time is usually associated with grand mal.

"Equivalents" are attacks in which only an abnormal mental state is manifested. They may come on after an attack of grand mal or petit mal or they may occur with no previous attack, apparently taking the place of one of them. Sometimes they are the first evidence of epilepsy. There may be for a time a complete alteration in the disposition of the child. He may have uncontrollable fits of anger, be disobedient or destructive, run away, and, in rare instances, even acts of violence have been committed. Upon recovery from such a state, which is usually sudden, there is generally no recollection of what has occurred.

The Mental Condition of Epileptics.—A careful distinction should be made between cases in which epilepsy is secondary to some organic brain disease, and the mental disturbances seen in cases of idiopathic epilepsy. The children who are the subjects of the latter disease, and who are perfectly normal mentally, are certainly few. All degrees of disturbance may be seen, from those who are simply dull, apathetic, backward in development, and uncontrollable in temper, to those who are melancholic, idiotic, and even maniacal. The earlier in childhood epilepsy develops, the greater is usually the mental disturbance seen, because of the effect upon the brain during its period of active growth. Mental deterioration with repeated attacks of petit mal may be rapid.

Symptomatic Epilepsy.—This occurs most frequently in children as a sequel of cerebral palsy, usually with hemiplegia, and it may follow either the congenital or acquired form. Epilepsy may come on at any time after the onset of the paralysis,—from a few months to five or six years. At first the attacks may be separated by long intervals, but they gradually become more frequent as time passes. The convulsions in posthemiplegic epilepsy begin, as a rule, on the paralyzed side, and for a long time they may be confined to that side; but later they may become general, in which case they are indistinguishable from attacks of idiopathic epilepsy. Severe seizures are more likely to be seen than are the mild ones. Children with microcephalus often regularly suffer

from repeated convulsions that differ in no way from epileptic seizures.

Jacksonian epilepsy consists in localized spasms of groups of muscles in the face, arm or leg with retention of consciousness. The most frequent lesion producing this form of epilepsy is a cerebral tumor, but almost any abnormal process involving the cortex may be the cause. Jacksonian epilepsy is described under the diseases in which it may be found.

Course of the Disease.—In most cases seizures at first occur at long intervals, of perhaps a year, but later they become more and more frequent. Either the mild or the severe attacks may be first seen, and may remain throughout as the only type present, or they may be associated in the same case. There are most frequently seen occasional major attacks with a large number of minor ones. The interval between the epileptic seizures in most cases is from two to four weeks, although they may be of daily occurrence. Sometimes three or four seizures will follow one another closely, and then there will occur a long interval. The seizures may come on either during sleep or in the waking hours, and in some cases for a long time they may occur only in sleep. Such cases present peculiar difficulties in diagnosis, and are often long unrecognized as epileptic. The general health of patients may be quite normal.

Death rarely, if ever, results from epilepsy, except from some accident at the time of the seizures, or from the condition known as *status epilepticus;* in this the attacks come on with great frequency and severity, the patient at times passing rapidly from one convulsion into another, the temperature rising to 105° or 106° F., and death occurring either from exhaustion or in coma.

Diagnosis.—In most cases there is little difficulty in recognizing the major attacks when they occur by day. Nocturnal attacks may be diagnosticated by the cry, the biting of the tongue, blood upon the pillow, sub-conjunctival extravasation, evacuation of the bladder or rectum, and the severe headache. Minor attacks present the greatest difficulties, and a positive diagnosis is often impossible until the patient has been watched for a long time. The most important points to be noted are sudden pallor, dilatation of the pupils, temporary loss of consciousness, or simply mental confusion, and sometimes the evacuation of the bladder. Psychic equivalents can only be suspected unless there is a history of attacks of grand or petit mal.

It is not always possible to distinguish between secondary or symptomatic epilepsy and the idiopathic or hereditary form, particularly if the case comes under observation late in the course of the disease. The points which go to establish the first form are: that the convulsive movements are partial, or limited to one side; that when they are general, they always begin in the same part of the body; or that there is a history

of partial or unilateral attacks for some time before the occurrence of any general convulsions. It is important in all cases to examine the patient carefully for signs of an old hemiplegia, the symptoms of which may be so slight as to be readily overlooked. A marked increase in the reflexes of one side is quite as conclusive evidence as is a distinct weakness of the arm or leg. In idiopathic epilepsy some of the stigmata of degeneration are usually present. The sudden development of epileptiform seizures in a child previously healthy, and in whom there is no hereditary history of the disease, should always arouse the suspicion of some organic brain disease, especially tumor.

Prognosis.—The danger to life in epilepsy is very slight. Death is generally due to some accident, particularly drowning, at the time of a seizure. The tendency to spontaneous cessation of the attacks is small, while the tendency to recurrence is very great. It should be recognized, however, that instances are not infrequently met with in which apparently clear cases of epilepsy recover. This may happen without any treatment. This is more common when the attacks have been of the grand mal type but even petit mal may cease spontaneously. The attacks may gradually become less and less frequent or may cease suddenly without recurrence.

The prognosis in any given case depends upon the cause of the disease and the duration of the symptoms. When the cause can be removed, which is infrequently the case, and when the symptoms have lasted less than a year, the prospects of permanent cure are fairly good. If an hereditary tendency to the disease is marked, if the epileptic seizures have developed apart from any adequate exciting cause, and if they have continued untreated or in spite of treatment for two or three years, the symptoms may perhaps be relieved, but there is little prospect of permanent cure. In the cases also which are due to local irritation, like that resulting from an old meningeal hemorrhage, the prognosis is invariably bad, and only temporary relief is to be expected. A few cases of traumatic epilepsy have been cured and many have been greatly improved by a surgical operation.

Treatment.—The general hygienic and dietetic measures are of equal importance with the use of drugs. The most common mistake is to rely only upon drugs, ignoring the other measures mentioned. It not infrequently happens that drugs are without any effect when they are the only means of treatment employed, whereas in conjunction with other measures marked improvement is seen. The general hygiene of the patient must receive careful attention. He should lead a simple, regular life, as much as possible out of doors, away from all sources of excitement. Particular attention should be given to the digestive organs. Meat should be allowed once a day and in moderate quantity. Milk

should be given, also buttermilk or kumyss. Green vegetables, peas and beans, may be given freely; also all fresh fruits. Tea, coffee, and alcohol in every form must be absolutely prohibited. Under no circumstances should a condition of chronic constipation be neglected.

Evidences of syphilis, in the history, by physical examination and by the Wassermann reaction should be carefully sought. If these are present or if there is only a suspicion that syphilis may be the cause a thorough trial of antisyphilitic treatment should be made.

The bromids are unquestionably the best means of combating the epileptic habit. Either the sodium salt alone or a combination of the sodium and ammonium bromid is to be preferred. The purpose should be to give the smallest doses which will control the seizures. Children require proportionately larger doses than adults, and in most cases a child of five years will need from twenty-five to fifty grains a day. The method of administering the bromids is of some importance. The larger part of the quantity for twenty-four hours should be given shortly before the time when the seizures have usually occurred; in the interval much smaller doses. In most cases it is desirable to give a full dose at bedtime. Bromids should always be given largely diluted—in from three to four ounces of water. It is believed by many that more satisfactory results are obtained with the bromids and a smaller quantity required if the sodium chlorid in the diet is restricted to a minimum. A combination of opium with the bromids is warmly recommended by some authors. The opium must be given in full doses and preferably for some days or weeks before giving the bromid.

Cases of petit mal are especially difficult to control. For such there is often an advantage in combining belladonna with the bromids. In all cases the treatment must be continued for a long time if anything is accomplished. The bromids should be gradually reduced after the attacks are controlled, but must be given in moderately large doses for at least two years after the seizures have ceased. Sometimes the combination of chloral or antipyrin with bromids is advantageous, particularly if the latter are badly borne or cause an annoying amount of acne. Cases have been reported of very striking benefit following the use of calcium lactate. It is deserving of trial and should be given in full doses, at least thirty grains a day for a considerable period.

The surgical treatment of epilepsy has of late attracted much attention. An operation is to be considered in cases in which the paroxysms are very frequent and severe, when they are limited entirely or chiefly to one side of the body and when there is present a definite local cause, such as an old fracture of the skull, or when epilepsy has followed an injury to the head even without fracture. The results of operation are, in many instances, disappointing. There may be a diminution of the

attacks for a time, but they usually recur. There are sufficient instances on record, however, of permanent improvement or even definite cure to warrant operative procedure for very frequently repeated epileptic attacks, especially if there are any evidences of localization of the lesion. *Status epilepticus* requires prompt and active treatment. A high cleansing enema should be given followed by chloral and bromid by rectum in full doses. Morphin hypodermically, or veronal in full doses, trional or amylene hydrate by mouth may be given in addition.

The education of epileptic children is a subject of great difficulty and is often neglected. There are many reasons why it is impracticable to send them to ordinary schools, and it is therefore very desirable that special schools and colonies for them should be established.

The Management of the Attack.—Abortive measures are sometimes successful in cases with a distinct aura, the most reliable being the inhalation of nitrite of amyl. While the seizure lasts, the patient should be prevented from injuring himself. The clothing should be loosened, a spool or cork should be placed between his teeth to protect the tongue, but no effort made to restrain his movements unless he is likely to do violence to himself. An epileptic child should never be without some companion.

CHOREA

(*Saint Vitus's Dance*)

Chorea is a functional nervous disease characterized by aimless, irregular movements of any or all the voluntary muscles. Choreic movements are of a somewhat spasmodic character, often accompanied by an apparent or real loss of power in the groups of muscles affected, and by a mental condition of extreme irritability.

Etiology.—Chorea is most frequently seen between the ages of seven and fourteen years. Of 146 cases, 6 were under five years, 72 between five and nine years, and 68 between ten and fourteen years. The youngest case of which we have records was that of a child four years old. It is extremely rare before the third year, although it may occur even in infancy. Our own observations coincide with those of nearly all writers, that the disease is more than twice as frequent in females as in males. While chorea may be seen at all seasons, it is much more frequent in the spring months. Of 717 attacks studied by Lewis (Philadelphia), the largest number began in March, and the next largest number in May; in our own cases May stands first.

The relation of chorea to rheumatism is of much importance. The investigations of different writers have given results which are somewhat

contradictory. Some have found evidences of rheumatism in but a small proportion of the cases—in not more than five or ten per cent—while the statistics of others have placed the percentage with rheumatism as high as fifty or even sixty per cent. The question hinges largely upon what is to be admitted as evidence of rheumatism in a child; if cases of acute articular inflammation only, then the number will be very small; if subacute cases with joint swellings are included, the proportion will be considerably larger; while if we admit cases of acute endocarditis without articular symptoms, and those of articular pains and joint stiffness but without swelling, the proportion will be very much increased. Our own belief is that there is a very close connection between chorea and the rheumatic diathesis as manifested by all the symptoms above noted, and accompanied by a family history of rheumatism. There seems to be a large group of cases, therefore, which may be classed distinctly as rheumatic. There are, however, a few others in which no such element can be found.

Crandall has analyzed 146 cases of chorea treated in an out-patient clinic and in private practice, with the following results: Of 111 cases in which the question of rheumatism was investigated there was a definite history of it in 63. In 41, articular symptoms occurred before the chorea; in 13, the first evidence of rheumatism was coincident with the chorea; and in 9 it first occurred subsequent to the chorea, usually within three months. In about one-third of the cases, attacks of rheumatism occurred during or subsequent to the chorea as well as before it. It may then be stated that previous rheumatism was evident in 37 per cent, concurrent rheumatism in 24 per cent, and subsequent rheumatism in 15 per cent of the cases. Excluding cases mentioned twice, and also all those in which there was a history only of "growing pains," there was evidence of articular rheumatism in 56.7 per cent of the cases. Many of these patients were under observation for several years, and it was interesting to see, as time passed, how the evidences of rheumatism multiplied the longer the cases were followed.

In the above statistics only articular symptoms have been accepted as evidence of rheumatism. If the cases of endocarditis without articular symptoms were included, as they might fairly be, it would raise the proportion of rheumatic cases still higher. The great proportion of constant cardiac murmurs persisting after chorea, if not all of them, should be classed as rheumatic, even if no articular symptoms have been present.

Overpressure in school is often an important element in the production of chorea. Anemia, if not an essential factor, is certainly a very important one, and the great proportion of cases present very distinct evidences of it. Chorea may develop as a sequel of any of the infectious

diseases, more particularly scarlet and typhoid fevers. Among the reflex causes that have been suggested, but whose influence is doubtful, may be mentioned phimosis, either lumbricoids or pinworms, delayed menstruation, and ocular defects. The latter frequently cause a local spasm of the muscles of the eyes, which can hardly be considered choreic. Hereditary influence is of considerable importance in the production of chorea. It is much more frequent in children of neurotic families, and very often several successive generations, or several children in the same family, may suffer from the disease.

The exciting cause of chorea in a certain proportion of cases is fright; occasionally it arises from imitation, and the disease has been known to occur epidemically in institutions.

The rôle of bacteria in the production of rheumatic chorea is still unsettled. The organism which Poynton and Paine have described as the cause of acute articular rheumatism has been found in the meninges of the brain in a few fatal cases of chorea, but in three of our own it was impossible to obtain any growth from the brain or other organs.

Pathology.—The exact pathology of chorea is at the present time not settled. The seat of the morbid process is undoubtedly the central nervous system, probably the motor areas of the cortex. The cases associated with rheumatism are now generally regarded as of infectious origin. In some severe cases which were fatal, owing to association with acute endocarditis, capillary emboli have been found in the brain. However, it is by no means established that this is the condition present in most of the rheumatic cases. The fact that in the great majority of such cases complete recovery occurs in the course of a few weeks or months, speaks strongly against any important structural change in the nervous centers.

Symptoms.—An attack of chorea generally comes on gradually. At first the child may be considered simply as unusually nervous; if at school, there may be noticed a difficulty in writing, drawing, or in using the hands for other delicate operations. At home, the child is continually dropping things, has difficulty in feeding himself, sometimes in buttoning his clothes, and very frequently he is not brought to the physician until the symptoms have lasted a week or two. Sometimes the legs are first affected, and a history is given of frequent falls, a stumbling gait, difficulty in going upstairs, etc. At other times the spasm is first seen in the facial muscles, with disturbance of articulation, twitchings of the eye muscles, and the child may be punished for making grimaces. In most cases the spasmodic movements soon extend to all parts of the body. They remain limited to one side of the body (hemichorea) in about one-third of the cases. When fully developed, the movements of chorea are quite unmistakable. They are irregular, jerking, spasmodic, never

rhythmical, rarely symmetrical, and vary in intensity from an occasional muscular contraction to almost constant motion. The movements are not under the control of the patient's will, and are usually intensified by efforts to repress them. They are increased by excitement, embarrassment, or fatigue, but do not continue during sleep.

Very often there is weakness of the affected muscles, which may be so great as to lead to the suspicion that actual paralysis exists. Not infrequently we have had patients brought to the clinic for supposed paralysis, either of one extremity or of one side of the body, where the choreic movements have not been severe enough to attract the attention of the mother. This paralysis usually disappears in the course of a few weeks.

In severe forms of chorea the patient may be unable to walk, to speak intelligibly or even to sit up in bed. The movements may be so violent that it is necessary to pad the bed and to wrap the child's extremities in cotton. Control of the bladder or rectum may also be lost. The symptoms may be so intense as even to threaten life. Such cases, however, are usually dangerous, not from the choreic movements, but from the acute endocarditis with which they are frequently associated. We have seen fatal cases, however, in which the outcome was not determined by the endocarditis. The temperature usually rises to 103° F. or more and remains constantly high. The choreiform movements are almost impossible to control even with sedatives in enormous doses, and death takes place after several days, apparently as the result of exhaustion.

The mental condition of choreic patients is one of marked irritability. They are fretful, emotional, easily provoked to tears or laughter, and difficult to control. In extreme cases a mental disturbance bordering upon acute mania has been observed. In other cases the facial expression and manner of speech strongly suggest beginning imbecility. All degrees of speech disturbances are seen from the slight difficulty in articulation due to inability properly to control the movements of the tongue and lips, to a condition in which speech is almost impossible. In severe cases speech may be temporarily lost.

Cardiac murmurs are frequent in chorea. Some of these are of anemic origin, but a large number, probably the majority, are due to concurrent endocarditis, as is shown by the fact that they are permanent, and are followed by all the signs of organic heart disease. During every attack the heart should be closely watched, especially in children in whom there is a strong predisposition to rheumatism.

The general condition of choreic patients is usually much below normal. They are anemic; the appetite is poor, often capricious; they sleep very badly; they suffer frequently from headaches; they are easily fa-

tigued by slight muscular exertion; and in short they have all the symptoms of a greatly disturbed nutrition.

Course and Duration.—The ordinary form of chorea tends to spontaneous recovery in from six to ten weeks. Exceptionally it may last for three or four months. In a small number of cases the disease may continue for a much longer period with remissions and exacerbations. Certain forms of local spasm, particularly choreiform movements of the muscles of the face, eyes, or neck, may be permanent. In any case of chorea which lasts longer than the usual time, the patient should be carefully examined for some cause of peripheral irritation. The tendency to relapses and second attacks is very marked. Later attacks are likely to occur in the spring succeeding the first illness, and in a small number of patients attacks may come every year for four or five years.

Diagnosis.—There is little difficulty in recognizing chorea from the sudden, irregular, spasmodic contraction of the muscles coming on under other circumstances. No other movements of childhood are likely to be confounded with it. The form of chorea following hemiplegia is usually more athetoid than choreic, yet at times it closely simulates ordinary chorea. The difficulty in distinguishing between the two is often increased by the fact that the weakness of simple chorea may, if unilateral, closely simulate hemiplegia. The existence of rigidity, contractions, and increased reflexes belongs exclusively to hemiplegic cases, and these will usually suffice to clear up all doubt with reference to the diagnosis.

Prognosis.—As a rule, this is favorable, and complete recovery can usually be predicted, the exceptions being few in number. Parents should always be warned of the tendency of the disease to return in succeeding years, and the fact should be stated that in a certain proportion of cases the disease may be of exceptional duration. The prognosis of the cardiac murmurs occurring in chorea should always be guarded, although some of these are functional and disappear with recovery from the chorea; but the number of those which do not disappear is very large and sufficient to make one always apprehensive as to the ultimate result. Acute chorea may be fatal from the accompanying endocarditis and much more rarely from the severity of the disease itself.

Treatment.—The general management of the case is equally important with the administration of drugs. A child with chorea should at once be taken from school, and should never be subjected to punishment or to ridicule on account of the movements. Special attention should be given to the patient's diet and general nutrition. Tonics, especially iron, are indicated in most cases. The food should be simple and nutritious, and all stimulants, particularly tea and coffee, should be absolutely prohibited. While fresh air is desirable, exercise should be prescribed with great caution and its effect should be carefully watched. A certain

amount of moral restraint is indispensable; thus it often happens that choreic patients do very badly at home where they are indulged and receive sympathy, while in a hospital, where they are under restraint and made to control themselves, they begin to improve immediately. In all severe cases the rest treatment should be employed. It is equally beneficial in the milder ones; the patient is put to bed, and complete mental and physical rest secured. This may be combined with gentle massage for fifteen or twenty minutes a day. The daily use of prolonged warm baths, either alone or in conjunction with massage, is at times decidedly beneficial. In other cases the regular use of cold douches is of value.

In estimating the value of drugs in the treatment of chorea, the natural course of the disease should be kept in mind, since those drugs which are taken after the third or fourth week are much more likely to be thought beneficial than those used in the early period of the attack. On account of the close association of chorea with rheumatism, antirheumatic remedies (sodium salicylate, aspirin, etc.) have very frequently been tried, especially in cases with fever and endocarditis and when joint symptoms supervene in the course of an attack. Our experience has been that they rarely have very much effect upon the course of the disease. They may alleviate the pain of acute arthritis somewhat and in large doses may reduce the temperature, but they exert little influence upon the severity or duration of the symptoms of chorea.

Arsenic was long, and still is, regarded by some as a specific for the disease. The usual method of administration is to begin with four drops of Fowler's solution three times a day for a child of eight years, and to increase the daily quantity by one drop every two or three days until eight drops are given at each dose. One should stop short of this if digestion is disturbed, or there is puffiness of the face or albumin in the urine. Arsenic should always be given after meals, and largely diluted. The possibility of arsenical poisoning should be remembered, although it is rare. We have known of several cases in which multiple neuritis developed after a few weeks' administration of the drug. In our hands arsenic has not been very effective against chorea.

Severe chorea requires sedatives. Not only do they relieve the symptoms but in many instances apparently have a distinct influence in shortening the duration of an attack. They must be given in quantities sufficient to produce an effect and the amount required is often enormous. The bromids, chloral, opium or morphin and veronal will be found the most efficacious. The bromids not infrequently must be suspended on account of eruptions. Morphin, hypodermically, is at times the most satisfactory drug. Improvement is shown by a diminution of the amount required to produce quiet but the above drugs must sometimes be continued for many weeks.

Chorea has a strong tendency to recur, especially in the spring months. Children who have had one attack should be closely watched, particularly with reference to their work in school. They should not be crowded in their studies, they should have long vacations, and the nervous system should not be put upon any severe tension for a long time.

OTHER SPASMODIC AFFECTIONS

Habit Spasm.—This term is used to describe certain spasmodic muscular movements which at first are only occasionally noticed, but which may persist until they become habitual and almost entirely involuntary. The movements usually affect the muscles of the face, but they may be seen in almost any part of the body. The most frequent varieties consist of blinking or sudden frowning, raising the eyebrows, grinding of the teeth, or some peculiar grimace. At other times there is sudden twisting of the head, shrugging of the shoulders, or jerking of the hands. Habit spasm is not often seen in the lower extremities, but the muscles of respiration are quite frequently affected. There may be a half-sigh, a sort of sob, or a peculiar dry, pharyngeal cough.

These movements are at first infrequent; but as the habit becomes more firmly fixed the spasm recurs every few minutes, and in severe cases it may be almost continuous. The form of spasm is not always the same; one may disappear and another take its place. The condition may last for months or years, and it may even be permanent.

Habit spasm is really little more than exaggerated nervousness continuing in some definite form until by repetition a fixed habit is established. It is different in cause, course, prognosis, and treatment from chorea, with which, however, it is often confounded.

The causes are those of neuroses in general. In the beginning, at least, the general health is usually below the normal. The patients are nervous children of neurotic antecedents. There may be a history of some definite exciting cause, such as illness or overwork in school. There is frequently some local cause of which the spasm is merely a reflex.

Habit spasm is to be differentiated from chorea: this is usually easy, from the limitation of the movements to one part or group of muscles and from the duration of the disease.

Treatment is quite unsatisfactory after the habit has become fixed, hence it is of very great importance that it should be arrested at the earliest possible age. Punishments are of no avail, and usually aggravate the condition. Rewards are much more effectual. The child's surroundings, work and study should be carefully investigated. Any local cause

which can be discovered should be removed. Especially should the general health receive attention.

Athetosis and Athetoid Movements.—These terms, introduced by Hammond, are used to describe a chronic form of spasm usually seen in the hand, but sometimes also in the foot, and even the face. It may affect both sides, but in most cases it is unilateral. The movement is slow, irregular, and incoordinate—a sort of "mobile spasm," it has been called —and there may be associated a certain amount of muscular rigidity. Such movements rarely occur in persons apparently healthy, but are usually seen as a sequel of cerebral palsies, generally hemiplegia. Recovery from the paralysis may be so nearly complete that the athetoid movements are looked upon as primary. In some cases the movements are more rapid and somewhat resemble those of chorea, the condition being sometimes classed as *post-hemiplegic chorea*. Athetosis is not influenced by treatment.

Rotary and Nodding Spasm of the Head.—These are rare forms of irregular movements usually observed in infancy. The condition was described long ago by Henoch. The most frequent is the rotary spasm, which consists in a side-to-side oscillation of the head, which may be slow or rapid, and in some cases is almost continuous. Some children have at times the nodding spasm also, and in others this is the only movement seen. Nystagmus is frequently associated, and may affect one or both eyes. In a few of the reported cases convergent strabismus was present.

The causes of the condition are extremely obscure. It is usually seen in infancy between the third and eighteenth months. It is believed by Raudnitz to be often the result of living in poorly lighted rooms, it being necessary for the infant to assume an unnatural position of the head in order to see things held before him. The nystagmus is regarded as analogous to that which develops in miners. While this explanation is satisfactory for some cases that are cured by being placed in well-lighted rooms, it is not applicable to all.

As a rule, the condition lasts for several months and improves, recovery almost always taking place. The prognosis is therefore favorable.

Nystagmus.—This term is applied to rhythmical, involuntary, oscillatory movements usually of both eyes. They are caused by the alternate contraction of opposing muscles. Nystagmus may be either vertical or lateral. It is most often seen in infants a few months old. In some cases the movement is almost continuous, occurring even in sleep; in others, it is only noticed at times of special excitement.

The etiology of nystagmus is obscure, and it may occur in quite a variety of conditions—sometimes referable to the eye, at other times to

the central nervous system. On the part of the eye, nystagmus may be due to blindness from any cause, to congenital cataract, corneal opacity, disease of the choroid or retina, or to errors of refraction. It may be seen in almost any organic disease of the nervous system, both with focal and diffuse lesions, especially in chronic hydrocephalus, insular sclerosis, tuberculous meningitis, and in diseases in which sight is impaired. While it is of no importance as a localizing symptom, nystagmus often indicates something more than functional disturbance. An exception to this may perhaps be made when it follows cerebral concussion. In such cases it is usually temporary, disappearing in a few days or weeks. Under other conditions it may continue indefinitely.

The condition of the eyes should be investigated in every case of nystagmus; it is only when the cause is here, and can be removed, that habitual nystagmus is amenable to treatment.

Hiccough (Singultus).—This is a spasm of the diaphragm which is usually seen in young infants. In them it is in most cases due to some irritation in the stomach, but is found in perfectly healthy infants with no digestive disturbance. It is seen after eating, and may depend upon overfilling of the stomach with food, swallowing of air, etc. In other cases it has no relation to the taking of food. In cases like the above, hiccough, though sometimes annoying, is of little importance. It may be associated with indigestion, with intestinal flatulence or inflammation, with peritonitis or with intestinal obstruction. With the last two conditions it is always an unfavorable symptom. In older children hiccough sometimes occurs as a pure neurosis.

The object of treatment is to remove the cause. In infants this is to aid in the expulsion of the gas from the stomach by manipulation or position. When it is a nervous symptom only, it may be arrested in older children by holding the breath, or by prolonged forced expiration, as in blowing a trumpet.

Thomsen's Disease (Congenital Myotonia).—This rare disease is usually congenital. It may occur in several members of the same family, and is almost always hereditary. The characteristic symptoms are a peculiar rigidity of the muscles which is observed when they are first brought into action after repose. This rigidity is spasmodic, and usually continues but a few moments. It may recur when voluntary movements are again attempted. If, however, muscular effort is persisted in, it soon passes off. It is increased by apprehension, excitement, or cold, and by observation. The legs are most frequently affected, the condition being often noticed when the patient starts to walk; any of the voluntary muscles, however, may be involved, even the tongue. It may be greater upon one side of the body than upon the other. The tendon reflexes are not increased but there is a marked and very prolonged contraction of the

muscles as a result of direct mechanical stimulation. The electrical stimulation of the nerves causes generally normal or diminished contractions; that of the muscles directly, either with the faradic or galvanic current, causes a contraction that remains for from ten to twenty seconds. The disease may be noticed very early in life and it generally increases in severity about the time of puberty. Thereafter it remains stationary, or nearly so. It never causes death but is incurable, although the symptoms may be improved somewhat by active muscular exercise.

The muscle fibers are increased in size and the nuclei much increased in number. There are no evidences of degeneration, but in the sarcoplasm may be seen a large number of small, round, colorless or yellowish dots that seem to indicate actual disease of this substance. Something can be accomplished by massage and muscular exercises to diminish the tendency to muscular rigidity, but nothing approaching a normal condition can be brought about.

Torticollis—Wry-Neck.—Torticollis may be congenital or acquired. Regarding the cause of congenital torticollis there is some dispute. Such cases have often been attributed to the contraction resulting from hematoma of the sternomastoid. It is our belief that this is rarely, if ever, the case. While it is possible that the deformity is sometimes the consequence of injury received during delivery, the cause of most of the congenital cases goes back to conditions existing before birth. It may be compared to club-foot, and may be due to a faulty position of the child *in utero*. There may be a congenital shortening of the sternomastoid muscle alone or of several muscles, or of all the tissues on one side of the neck. Very rarely congenital torticollis is the result of anomalies of one or more cervical vertebrae. The most frequent cause in the acquired cases is inflammations of the neck, the result of tonsillitis and pharyngitis. Such is the usual etiology of torticollis following scarlet fever, measles, or diphtheria. The exciting cause of the spasm is irritation of the cervical nerves, usually the spinal accessory, though others also may be involved.

Torticollis is seen with cervical adenitis, acute or tuberculous, and with cellulitis of the neck. Indeed, it may be the result of anything causing irritation of the trunk or branches of the spinal accessory nerve, either in the spinal canal, the cranium, or along the course of the nerve trunk or of any of its peripheral fibers. Most of the cases that have been described as the result of rheumatism and cold are probably due to infections occurring through the tonsils and pharynx. A cause which the physician should always have in mind is cervical Pott's disease; torticollis may be the earliest, and for several weeks sometimes almost the only objective symptom of this disease. Infrequent causes of tor-

ticollis are acute inflammation of the suboccipital articulations, unilateral dislocation, osteo-arthritis of the cervical spine and cervical rib.

The onset may be acute and accompanied by fever, or what is more frequent is that the torticollis gradually develops, it being several days or weeks before it is marked and permanent. The deformity varies somewhat, according as the sternomastoid muscle is alone affected, or the posterior muscles also, and as to which predominates. In simple sternomastoid spasm the head is inclined to the affected side and rotated toward the opposite side; the chin is raised, and the ear approaches the clavicle. When other muscles are involved the deformity is modified. If the trapezius is affected there is less rotation of the head, but it is drawn to the affected side and somewhat backward, while the shoulder is raised and the spine curved. Both of these symptoms may be seen to a slight degree in almost any marked case of sternomastoid spasm. Sometimes the spasm of the posterior muscles affects both sides; the head is then drawn backward and held rigidly, but without rotation. In recent cases the deformity can be partially or entirely overcome by passive force; but after a time this is impossible, owing to muscular shortening. Atrophy may take place in the affected muscle. In recent cases localized pain and tenderness are also frequently present, and sometimes they are severe. Attempts to reduce the deformity may produce great pain.

Prognosis.—The result in a case of torticollis depends upon the cause, the severity and the duration of the deformity. Recovery in most of the acute cases is complete in the course of a few days or weeks. In others, after the subsidence of the symptoms of local inflammation there may be no tendency to a reduction of the deformity. This, if untreated, may be permanent, owing to shortening of the muscles and fascia. The congenital cases with slight deformity are usually amenable to mechanical or postural treatment if begun early. There is in most of the other varieties a disposition for the deformity, if untreated, to persist, and even increase. If it has lasted several months the probabilities of spontaneous recovery or even of improvement are small.

Treatment.—The first indication is to remove or treat the cause when one can be found. Acute cases are to be treated by rest in bed, hot applications, counterirritation and friction, unless the pain is too severe. Cases which have lasted a month usually require some orthopedic head-support, and those which have lasted six months or more are rarely cured without a surgical operation. This may be either a subcutaneous tenotomy or myotomy of the sternomastoid, or an open incision. An old case of torticollis is a serious matter and radical measures should be resorted to early in the disease.

HYSTERIA

This is not a disease of childhood, but one which is occasionally seen in early life. All that will be attempted in this chapter is to point out the most common manifestations of hysteria when it occurs in children. After puberty it is essentially the same as in adults.

Etiology.—Hysteria is very rare before the seventh or eighth year, and most cases seen in children occur after the tenth year. As to sex, there is no such predominance of females as in later life, although even in childhood they are more frequently affected than males. Hereditary influences play an important part in the production of this disease. It is seen in children who inherit a nervous constitution, or in whose parents nervous diseases, such as insanity, or hysteria, or neurasthenia, have been present. Of the other etiological factors the most important are a disordered nutrition, frequently with anemia or chlorosis, and overpressure in schools. Masturbation may act as an exciting cause, or, indeed, anything which leads to an exalted nervous irritability and depreciation of the general health. It may follow any of the acute infectious diseases; or it may be excited by injury, fright, or imitation.

Symptoms.—There is scarcely any disease in which the clinical picture presented is so varied as in hysteria. It may simulate almost any form of organic disease of the brain, lungs, digestive organs, bones, or joints. The symptoms are seen in almost every conceivable combination.

Psychical symptoms frequently predominate. There may be seen periods of mental depression of longer or shorter duration, a change in disposition, an indifference to surroundings, a capricious humor, or a nervous condition of extreme irritability with irregular paroxysms of laughter or weeping without cause. There may be great excitability of temper, and fits of passion almost maniacal in their severity. There may be various hallucinations. Sleep is frequently disturbed, sometimes by attacks resembling ordinary night-terrors; sometimes somnambulism is present. There is often a disposition to deception about the most trivial matters, which may last for weeks. There is a tendency to imitate the symptoms of various diseases, which the patients may have witnessed in others or about which they have read. Sometimes the special senses are affected, giving rise to hysterical blindness or deafness, usually of short duration.

Sensory symptoms are the most frequent manifestations of hysteria in early life. There is often general or local hyperesthesia, which may be so great as to simulate inflammation of the various internal organs. Anesthesia is much less common, although it may be seen in children as young as eight or nine. Anesthesia is very frequently associated with

paralyses. In such circumstances it is apt to involve the whole of one or more extremities and in such a way as to be inexplicable by any organic lesion. Paralysis is an infrequent but striking symptom. There may be monoplegia or paraplegia, more rarely hemiplegia or paralysis of all four extremities. There may even be edema and a certain degree of atrophy of the affected extremity from disuse. The inability to stand or walk, though the legs can be moved perfectly in the recumbent position, is observed at times. Headache is an occasional symptom, and is sometimes associated with great tenderness of the scalp. There may be neuralgias in the different parts of the body, or sharp pain, sometimes accompanied by vomiting.

Joint symptoms are really a variety of sensory disturbances. They are not uncommon, and are often most puzzling. All forms of organic disease of these joints may be simulated. Joint symptoms are usually seen between the ages of ten and fourteen years, and occur in both sexes. There may be lameness referred to one of the large joints, curvature of the spine, or torticollis. The symptoms are most frequently referred to the hip, and next to the knee, the ankle, or the spine. The pain is acute. It is increased by motion, and by attempts at overcoming the deformity, if any is present. There is a marked hyperesthesia of the whole limb, and sometimes of the body. The resistance and pain caused by passive motion are often greater than in most joints, which are the seat of organic disease. In nearly every case there is marked tenderness of the spine upon pressure, especially in the dorsal region. The deformity may be very slight from spasm of the flexors only, or it may be severe, and followed by contracture, so that the thighs may be flexed tightly against the abdomen with the heels against the buttocks. Such deformities may last for months. There may be considerable muscular atrophy, but only that which comes from disuse. A special difficulty in diagnosis arises from the circumstance that these symptoms occasionally follow an injury.

Organic disease of bones and joints may usually be excluded by attention to the following points: The mode of onset is more abrupt than is seen in bone disease, and the course of the disease is quite irregular. The degree of deformity is greater than is seen in bone disease of the same duration. There is general hyperesthesia of the limb, acute tenderness of the spine upon pressure, and undue sensitiveness to heat or cold. The deformity varies from time to time, being always more marked when examination is attempted. If the patients are closely watched, other evidences of hysteria may be seen. Under complete anesthesia the contractures disappear entirely. There is no enlargement of the articular ends of the bones, no swelling of the soft parts, and no evidence of active inflammation or of suppuration. All the symptoms

except the deformity are subjective. Under proper treatment there is in most cases perfect recovery, often in a surprisingly short time.

Digestive symptoms are quite frequent. There may be loss of appetite, at times so extreme as to lead to great emaciation. There may be dysphagia from spasm of the esophagus, or regurgitation of food on attempts at swallowing. There may be troublesome hiccough. Vomiting is a frequent symptom. It is seldom severe. A very frequent form met with is that which occurs in school children before starting for school. Throughout the rest of the day nothing is vomited and the appetite may be good. Persistent diarrhea, constipation, meteorism, and incontinence of feces may be met with.

In the milder forms of hysteria there are seen many varieties of tonic or clonic spasm. There may be local spasm of the eyes, face, or mouth, spasm of the muscles of the neck producing torticollis, of the muscles of respiration causing dyspnea, which may be constant or paroxysmal. Disturbances of speech are quite common especially in older children. There may be inability to speak above a whisper while the voice is retained in singing or after the application of the faradic current to the neck. Stuttering and stammering may be due to hysteria. Very rarely no attempt at phonation can be made. A very common symptom is hysterical cough, which may be so frequent and so severe that grave disease of the lungs is suspected; the chest, however, is free from the physical signs of disease. In more severe cases we may have the symptoms of chorea major and attacks of hystero-epilepsy. The latter are rare in children and do not differ essentially from such attacks in older patients. There are usually prodromal symptoms. The convulsive movements are exceedingly varied in type. There are painful sensations and sensitive areas, by pressure upon which hysterical symptoms may be increased or even convulsions excited. The respiration may be rapid or irregular. All variations in tonic and clonic spasms may be seen. Opisthotonus is frequent. Consciousness is not fully lost, but is disturbed, and hallucinations are present. The temperature is normal.

Other symptoms occasionally seen in hysteria are polyuria, very frequent urination, sometimes incontinence of urine, and disturbance of the secretion of saliva or perspiration.

The general condition of hysterical patients is usually below the normal. They are poorly nourished and anemic; they sleep badly; they have capricious appetites and feeble digestion.

Diagnosis.—Hysteria is apt to be overlooked because its occurrence in children is not considered as often as it should be. In most cases the diagnosis is easy if hysteria is suspected. A combination of vague disconnected symptoms is usually present which admits of no other ex-

planation. Organic disease can be excluded only by careful and repeated examinations. It is to be borne in mind, however, that hysteria not infrequently complicates organic or constitutional disease. Much importance is to be attached to a family history of hysteria or of other neuroses.

Prognosis.—This is better than in adults, especially if the cases are taken in hand early, before the disease has become deeply seated. Very much depends upon how well the directions for treatment can be carried out. The prognosis is less favorable when the hereditary tendency is strongly marked. In many cases there are relapses later in life.

Treatment.—Prophylaxis is of much importance. When an hereditary tendency to nervous diseases exists in a family, or whenever very nervous children are placed under the physician's care, every means should be taken to further muscular development, keeping the nervous system in the background. Such children should lead an outdoor life as much as possible, preferably in the country. They should keep early hours, have regular exercise, and their education should be directed with moderation and judgment, special attention being paid to regularity of work and the prevention of overpressure in schools. Theaters and exciting books should be avoided. All stimulants, including tea and coffee, should be absolutely forbidden. The diet should be plain and nutritious. It is highly important that such children should be removed from association with an hysterical mother, when this is possible. The best results are usually obtained when the child is taken from his home surroundings and placed in some quiet retreat in charge of an intelligent nurse. Isolation is absolutely essential in many cases.

In the general management of a case of hysteria, it is of the first importance that the child should be cared for by a person of firmness, who can exercise proper control. The general health should be carefully looked after, and arsenic, iron, cod-liver oil, and other tonics given according to indications. Outdoor sports should be encouraged, and every means taken to interest the child in something which requires physical exercise. In cases of simulated disease, the child should be put to bed, no books or toys allowed, and no effort made toward his amusement. No sympathy should be exhibited, but the child should be treated with kindness and firmness. This moral treatment is quite as important as any other part of the therapeutics. In cases with hysterical joint symptoms mild counterirritation to the spine, preferably by the Paquelin cautery, is sometimes of distinct benefit. In no circumstances should mechanical force be used to overcome deformity. Many cases of hysteria improve under hydrotherapy; the cold douche, the cold pack, or the shower bath may be used. This is valuable in conjunction with massage and the rest treatment.

HEADACHES

Headaches are not common in little children except in connection with disease of the brain or meninges; in older children they occur from causes similar to those seen in adult life. The most frequent headaches may be grouped in the following classes:

1. *Toxic Headaches.*—Such are the headaches resulting from uremia, from malaria, and those seen in many acute infectious diseases. But the largest number are associated with disturbances of digestion.

2. *Headaches from Anemia, Malnutrition, and Nervous Exhaustion.*—These are most frequently seen in girls from ten to fourteen years old. Some are intellectually bright, and have been crowded in their school work; others are dull and learn only with difficulty, and in consequence worry over their work until their health becomes undermined. They sleep badly, lose appetite, and often become choreic. The anemia may be either the cause or the result of these symptoms.

3. *Headaches of Nervous Origin.*—These may occur in children who are highly neurotic, either from their inheritance or surroundings, and in those who are the subjects of epilepsy or hysteria, and they may be symptomatic of organic disease of the brain, such as tumor or tuberculous or syphilitic meningitis. True facial neuralgia is rare in childhood except from carious teeth; from this cause, however, it is not infrequent.

4. *Headaches due to Disease of some of the Organs of Special Sense.*—In connection with the eyes there may be conjunctivitis, keratitis, iritis, errors of refraction, or strabismus; connected with the nose there may be polypi, hypertrophic rhinitis, or adenoid vegetations of the pharynx; connected with the ears there may be otitis or foreign bodies in the canal. Each one of these conditions requires special treatment.

5. *Headaches due to Inherited Gout or Rheumatism.*—These are not very frequent, but they may be severe, and may at times simulate the onset of meningitis. They are often accompanied by pains in the joints, muscles, or nerve trunks.

6. *Disturbances of the genital tract* are rarely a cause of headaches in children, although this may be the case in girls about the time of puberty, especially when menstruation is delayed or difficult.

Diagnosis.—The diagnosis of headaches includes the discovery of the cause, and this is often difficult. In an infant or a young child, organic disease of the nervous system should always be suspected as a cause of severe headaches. In older children the important things to be considered, because the most frequent, are digestive disturbances, nervous

exhaustion, malnutrition, and visual disorders. An absolute diagnosis in a case of persistent headache can be made only by a careful physical examination, not omitting a study of the urine; often there must be a close observation of the patient for some time.

Treatment.—The only successful treatment is that which is directed toward a removal of the cause. Each one of the different groups above mentioned is to be managed differently, according to the principles elsewhere laid down regarding the treatment of these conditions. For the relief of the symptoms, cold to the head, a hot foot-bath, and phenacetin in moderate doses are perhaps the most certain of all remedies.

DISORDERS OF SPEECH

In this chapter will be discussed only functional speech defects, those depending upon organic conditions being considered in connection with diseases of the brain. The most common varieties are stuttering, stammering, lisping, alalia, backwardness, and functional aphasia. All forms are much more frequent in boys than in girls, the proportion being more than four to one.

Stuttering.—This is the most common form of speech disturbance. Articulation is distinct and the separate sounds are properly produced, but there is a difficulty in connecting the consonant with the succeeding vowel; this seems like an obstacle to be overcome. Occasional stuttering is seen in very many children. It is more frequent in the third and fourth years, before speech is thoroughly mastered. At this age it is aggravated or produced by disturbances of nutrition, but is usually a temporary condition, lasting for a few weeks or months. Recently a little boy of four was under our care, who became very anemic, slept poorly, and suffered from malnutrition as a result of the confinement incident to a home in the city. He soon began to stutter, and in a short time it became painfully marked. After a few weeks in the country he improved very much in his general condition, gained four or five pounds in weight, and his stuttering completely disappeared. In other cases stuttering follows some acute illness, and under such conditions also it is usually of short duration.

Most children who become habitual stutterers do not begin until they are six or seven years old, and sometimes even later. Stuttering may arise from imitation, and inheritance is an important etiological factor. It is frequently a mark of degeneration.

It is important that all such cases receive early treatment before the habit becomes firmly fixed. The prognosis is good for spontaneous recovery in nearly all the cases seen in very young children, and also in

those coming on after acute illness. Other cases in which the condition has become habitual should have the benefit of systematic training under a competent teacher in breathing and vocal gymnastics.

Stammering.—This term is sometimes used synonymously with stuttering. Kussmaul makes the distinction between them that, in stammering, individual sounds are difficult of production, while in stuttering it is syllabic combinations. Stammering is often accompanied by some defect in the organs of articulation—the teeth, lips, tongue, or palate—which is not present in stuttering.

The treatment consists in careful training and in the correction of whatever abnormal local conditions may exist.

Lisping.—In this there is an imperfect production of certain sounds, owing usually to a faulty position of the organs of articulation. The sounds may be so indistinct that they can not be understood. In this condition also there may be defective formation of some of the organs of articulation, although in the milder forms this is not the case. The treatment is similar to that of stammering.

Alalia.—This consists in a total inability to articulate. It is seen in all young infants during their earliest attempts at talking. In older children it is not a very rare condition, being usually associated with some mental defect.

Backwardness.—Backwardness is carefully to be distinguished from a late development of speech due to mental defects. At two years old children not deaf are almost invariably able to speak. Speech may be late in consequence of prolonged or very severe illness, and when it has once been acquired it may be lost from similar causes.

Functional Aphasia.—The term has been applied to a temporary loss of speech which sometimes occurs in chorea, and sometimes from severe fright or anything else which has produced a marked nervous impression. West records an instance in a girl of eight years, who was suffering from an attack of chorea induced by fright. Speech first became difficult and then was lost altogether. For a month the child could say only "yes" and "no." The child improved very slowly, but at the end of nine weeks had recovered completely. Loss of speech sometimes follows the acute infectious diseases, especially typhoid fever.

In all disorders of speech, the functional cases are to be distinguished from those which depend upon deafness and mental deficiency. The frequency with which these disorders are due to disturbances of general nutrition, and to local causes in the mouth and throat, should be borne in mind, and these conditions should receive their appropriate treatment early, before the habit of defective speech becomes firmly established. For the latter class of unfortunates, special training at the hands of a competent teacher should be advised, preferably in an institution.

DISORDERS OF SLEEP

Disturbed Sleep, Sleeplessness.—Disturbed or restless sleep is much more common in infancy and childhood than is true insomnia, although the causes of the two conditions may be the same.

Etiology.—In infancy these symptoms are most frequently due to hunger or to indigestion resulting from overfeeding or improper feeding. Very often disturbed sleep is the result of bad habits, such as rocking during sleep or night-feeding. Sometimes it arises from the pain of colic or otitis, rarely from dentition; at other times it may be simply the expression of a condition of extreme nervous irritability, the result of inheritance or of the child's surroundings. It is often caused by the persistent activities of a fussy nurse or mother.

In later childhood the first thing to be suspected when sleep is much disturbed is some derangement of the digestive organs; in this will be found the explanation of fully half the cases. The most frequent type, when the symptom is of long duration, is chronic intestinal indigestion, often associated with distention, a condition in which formerly the usual diagnosis was intestinal worms. Other cases are due to obstructed respiration from adenoid growths of the pharynx or enlarged tonsils, sometimes to nocturnal attacks of asthma. A lack of fresh air in the sleeping room, excessive or insufficient bedclothing, and cold feet, are other frequent causes. Disturbed sleep with "starting pains" is one of the earliest symptoms of hip-joint disease. In the nervous exhaustion resulting from overpressure in schools, and in malnutrition and anemia, disturbances of sleep are well-nigh constant. They are also seen in organic cardiac disease and in all pulmonary conditions accompanied by dyspnea or cough. Sleep may be disturbed in consequence of bad dreams which have their origin in exciting stories heard or read just before bedtime, or in too violent or exciting play. To discover the cause in almost any case it is necessary to investigate carefully the whole routine of the child's life.

Symptoms.—The condition may be one of real insomnia which may last for weeks or months; or the sleep may be simply disturbed and restless, the child waking many times during the night, and when asleep will not lie quietly, but constantly changes his position. Sometimes children wake suddenly with a scream, but immediately drop off to sleep again.

Treatment.—The essential treatment consists in the discovery and removal of the cause of the disturbance. This will often involve a radical change in the manner of feeding, in the hygiene of the nursery, and in all the surroundings of the child. A change of nurses sometimes results

in a speedy cure. In no circumstances should the physician countenance the use of drugs to promote sleep in children, except in the case of severe acute disease. Soothing syrups and all nostrums for "teething" should be absolutely forbidden; also the sucking of "pacifiers." Many mothers and nurses fall into the habit of using them, because the injurious effects are not appreciated. When the cause of sleeplessness is found and removed the child will sleep, but compulsory sleep obtained under other conditions is usually productive of more harm than good. If food, diet, and all bad habits have been corrected, nervous causes should be investigated. When no cause can be discovered the treatment should consist in putting the child upon the simplest possible diet, and in attention to such general conditions as anemia, malnutrition, and neurasthenia, some of which are almost certain to be present. In many cases a warm bath at bedtime will be found beneficial. A quiet, darkened room, plenty of fresh air, and the stopping of both eating and drinking during the night, are essential to a cure in most cases. When the condition accompanies some acute disease, the drugs which are most useful are codein and trional. A child of two years may take gr. $\frac{1}{20}$ of codein or two grains of trional as an initial dose, to be increased if necessary.

Night Terrors—Pavor Nocturnus.—Two classes of cases have been grouped under this head, both having this in common, that sleep is disturbed by fright.

The condition in the first group partakes of the nature of nightmare. It may be due to partial asphyxia from adenoid growths of the pharynx, or to other causes mentioned under disturbed sleep, or it may be gastric or intestinal in its origin. These cases are quite frequent. Sleep may be disturbed from the outset, and the attack may be merely the culmination of such disturbance. The child wakes in a state of fright and excitement, and often says he has had a bad dream. His mind is clear, he recognizes those about him, but it may be a long time before he is sufficiently calm to sleep again. The attack may be remembered perfectly the next day. Cases like this are to be managed in the same general way as those of disturbed sleep above mentioned.

In the second group are the only cases to which the term "night terrors" should really be applied. These are relatively rare, but the condition is a much more serious one. The symptom is generally due to some disturbance of the central nervous system. It occurs especially in those of neurotic antecedents, or those who have previously suffered from infantile convulsions, and it is often the precursor of other nervous attacks—migraine, hysteria, epilepsy, and even insanity. The attack usually comes suddenly where a child has previously been sleeping quietly, and more frequently in the early part of the night than later. He is generally found sitting upright in his bed in a bewilderment of terror,

being "afraid of the dog," or "the bear," or there is some other vision or hallucination which has produced the fright. Often this is associated with something of a red color. The child does not recognize those about him, does not know where he is, and may go to sleep again without coming to full consciousness. The next day there is no recollection of what has happened. Usually no after-effects are seen, but sometimes a large amount of pale urine is passed. The attacks may be repeated at intervals of a few months, or they may occur every few nights; but whatever the peculiar nature of the vision, it is likely to be repeated in nearly the same form. Such attacks have something in common with epileptic seizures, and the diagnosis between them may at times be difficult. They are to be regarded seriously, not only on account of what they are in themselves, but on account of what may follow.

Treatment.—All mental and nervous strain should be most carefully avoided, and when the attacks are frequent the bromids should be given at bedtime. Some person should sleep in the same room with the child, or in an adjoining one with the door open.

Excessive Sleep.—It is rare that either infants or children sleep an unnatural amount of the time unless one of two causes is present—organic brain disease, most frequently tuberculous meningitis, or the use of drugs. The latter is always to be suspected if with the sleep there is associated obstinate constipation. Opium in the form of "soothing syrup" or paregoric is the drug which has usually been given.

INJURIOUS HABITS OF INFANCY AND CHILDHOOD

On account of the close connection of such habits with disturbances of the nervous system, they may be properly considered with the functional nervous diseases. Although some of these habits may not be of serious importance, yet as a group they usually receive too little attention at the hands of the physician. The list is very long, and only the most important ones will be discussed.

Sucking.—This is a very common habit in infants, and during the first few months it is seen to some degree in most of them. If they are carefully watched the habit is easily stopped; otherwise it may continue indefinitely. Young infants usually suck the fingers when hungry, and this can scarcely be considered abnormal, but an effort should always be made to stop it, lest the habit become fixed. Lindner distinguishes between simple sucking and sucking with combinations. In the former, the child sucks some part of the body, such as the thumb, fingers, toes, tongue, lips, back of the hand or arm, or it may be some foreign substance, such as part of the clothing, the blanket, a rubber nipple, or the

"pacifier." This is the most common form that is seen. In the second variety the sucking is accompanied by the rubbing of some other parts, which seems to afford a pleasurable excitement; this may be the ear, the genital organs, or any other portion of the body. Sometimes sucking is accompanied by some practice which produces actual pain, such as pulling of the hair or scratching the body. Habits of sucking often persist throughout infancy, and not infrequently throughout childhood; they have often been known to continue up to puberty. The longer the habit has lasted the more difficult is it to break.

The results of sucking may be serious. Deformities of the thumb or finger, of the lips and teeth, and even of the jaws, are sometimes produced. We knew a woman whose thumbs to advanced age showed a deformity resulting from the habit of thumb-sucking while a child. In her case the habit was not broken until she was eight or nine years old. Probably the most pernicious result of sucking is its tendency to develop the habit of masturbation. Habitual sucking of one hand or finger may lead to spinal curvature.

Treatment.—In the management of these cases the most important thing is to arrest the habit early, before it becomes fixed. Too often the habit of thumb-sucking, or of sucking a rubber nipple, is encouraged by mothers, nurses, and sometimes even by physicians because of the temporary quiet which is thereby produced. In no circumstances should it be resorted to as a means of putting children to sleep or otherwise quieting the nervous system. With infants, the only treatment which is at all successful is mechanical restraint. It is of no use to cover the part which is sucked with bitter solutions. The hands of young infants may be covered with mittens, or with the long sleeves of a night-gown which is pinned to the bed, so that it is impossible for the child to get the part to the mouth; or, still better, cuffs or splints of pasteboard may be applied at the elbow, so as to prevent flexion of the arms. In the milder cases the habit is often discontinued spontaneously; but when it has been indulged in until a child is four or five years old, it is broken only with the greatest difficulty. Punishments are of little avail, but rewards are often successful.

Masturbation.—This is not uncommon even in infancy. Many cases have been observed during the first year, and some as early as the seventh or eighth month. It is seen in children of all ages and in both sexes; but in infants and very young children it is, in our experience, much more common in girls than in boys.

Etiology.—Local causes are present in many cases; they are usually something which produces undue irritation. The most frequent are, long or adherent prepuce, phimosis, balanitis, vulvovaginitis, eczema of the labia, threadworms, and tight clothing. A urine which is

irritating because of excessive acidity or the presence of crystals of uric acid may be a cause. Any irritation may lead the child to rub the parts in some way, and a pleasurable sensation being excited, this action is repeated until a habit is formed. Other causes are exercises in which the legs are rubbed together, or the body against a pole, as in climbing. To these causes must be added, in infants at least, the habit of sucking. After infancy the habit of masturbation is usually acquired from other children, but sometimes taught by vicious nurses.

General causes are also important as predisposing factors. These are the same as underlie most of the neuroses of childhood—viz., anemia, general malnutrition, and a highly neurotic constitution or nervous instability, which is often an inheritance, and is always aggravated by surroundings which tend to unnatural stimulation of the nervous system. When masturbation develops in a young child without any local cause, it may be an early sign of either mental deficiency or moral delinquency; if looked for, other stigmata of degeneration will often be found, and in many cases other vicious traits will appear later.

Symptoms.—In infants and very young children masturbation is usually accomplished by thigh friction or by rubbing the body against a pillow, a chair, or some other object. The variety of ways is almost endless. Frequently the child will simply lie upon the floor with the thighs crossed and rigidly held, and sway the body backward and forward. This lasts for a few moments, is accompanied by flushing of the face and some appearance of excitement, followed by relaxation, and often by perspiration. It frequently happens with little children that these "queer tricks," as they are often regarded, have been continued for months before their true nature is suspected.

A consciousness that they are doing something wrong, early leads even young children to seek seclusion when they repeat the habit. It is especially likely to be practiced when children lie long awake alone after they go to bed, or if they wake early. The habit is always made worse by any deterioration of the general health. We have known many children, who were thought to be entirely cured, to relapse under such conditions.

It is somewhat difficult to separate the general symptoms with which masturbation is associated, and upon which it largely depends, from those which are the direct result of the habit. There are some children in whom the condition is chiefly or entirely dependent upon a local cause, or when it is only occasionally practiced, in whom no general symptoms are seen, or at most only an unnatural shyness and a disposition to seek seclusion. Others are precocious and excitable, with an excessive amount of nervous sensibility. There are others in whom more marked nervous symptoms are present; the most striking are absent-mindedness, loss of

power of concentration, loss of interest in all amusements, and mental depression. Some girls of only seven or eight years may have fairly regular periods in which masturbation is practiced. In one of our patients such periods for a considerable time occurred monthly. During them even very little girls may lose all sense of modesty or decency. Every particle of self-control is gone. They become passionate, excitable, apparently possessed by the one uncontrollable desire to practice the habit. In the intervals such children may be quiet, modest, sweet-tempered, and perfectly normal. In some older subjects nymphomania, or even insanity, may be the ultimate result. Epilepsy, chorea, or hysteria may develop, particularly where a strong predisposition to them already exists in the family. The effect of masturbation upon the physical and mental development of the child may be serious when it is begun at an early age or is frequently practiced. But more striking is the change sometimes brought about in a child's moral nature. Even little children of eight or nine years may become centers of moral infection, which may involve a group of playmates or even a whole school.

Local symptoms of masturbation are not always present; in the male there may be redness and slight swelling of the prepuce; the organs may be abnormally large or simply much relaxed. The frequent occurrence of erections in young boys is always a suspicious symptom. In the female there is sometimes seen an abnormal development of the genital organs for the age, with an early appearance of pubic hair. Little importance is to be attached to adhesions of the clitoris. Sometimes there is vaginitis.

Prognosis.—Masturbation in children is at all times a most difficult condition to deal with. The outlook is better in infants and young children than in those who are older, because the latter are more difficult to watch and control; besides, in them the habit has usually become more firmly fixed. In young children local causes are frequently found to be at the root of the trouble; in those who are older general causes are more often present, and these it may be impossible to remove. In almost any case in which the habit has become firmly developed, many months and usually several years are necessary for complete cure. The tendency to relapse is very strong. When masturbation is a symptom of degeneracy it is usually hopeless.

Treatment.—The most important thing is an early recognition of the condition. The physician should put parents and nurses on their guard, and the first suspicions should be reported and the child carefully watched until all doubt is removed. In young infants much may be accomplished by mechanical restraint. The kind of restraint which is necessary will depend upon the manner of masturbating. If by the hands, they should be tied during sleep, so that the child can not reach

the genitals; if by the thigh-friction, the thighs should be separated by tying one to either side of the crib. In inveterate cases, a double side-splint, such as is used in fracture of the femur, may be applied. In children that are over three years old, all such contrivances are almost invariably unsuccessful. It is of the utmost importance in every case to have the child under the close surveillance of a competent and trustworthy person. He should be especially watched just after being put to bed and immediately after waking. Corporal punishment is often useful in very young children, but of little or no benefit in those who are over three years old. In fact, in such cases it may do positive harm, for deception and lying are soon added to the previous vice. The mother should secure the child's confidence, and in every way possible seek to strengthen his will and stimulate his self-control, using her influence to help him break the habit. In fact, in older children this psychic treatment is much more important than all other measures. Often absence from home under the care of a trustworthy companion is essential to successful treatment. Local causes, too, must be sought and removed whenever found. Circumcision should be done if phimosis exists; and even when it does not, the moral effect of the operation is sometimes of very great benefit. In girls improvement sometimes follows a separation under anesthesia of the preputial hood from the clitoris. But unless this is frequently repeated, the adhesions soon recur. Complete circumcision is sometimes done with advantage, and in very obstinate cases the clitoris may be cauterized. Blistering the inside of the thighs, the vulva, or the prepuce is sometimes useful. But as a rule none of these measures accomplishes anything permanent. Care should be taken that the clothing does not irritate the parts. The child should be removed from all vicious companions; but it is quite as important that the greatest vigilance should be exercised in the home and at school, so that the child should have no opportunity to teach other children the habit. In the most serious cases the child should be sent away from home and kept from other children. The coöperation of a trustworthy nurse or companion is indispensable.

General treatment should be directed to the child's condition; it is required in most of the cases. A child suffering from malnutrition and anemia should be sent to the country, kept out of doors and away from books, studies, and from everything which stimulates or excites the nervous system. Almost all active exercises except horseback may be recommended. Every means should be employed to build up the general health. These cases are most difficult and most discouraging ones for the physician. A cure results only by using all these measures and for a long time.

Nail-biting and tongue-sucking are two forms of habit which are less

frequent and less important than those already mentioned. The former is best remedied by wearing gloves and by keeping the nails cut very short. Tongue-sucking seldom becomes a fixed habit, and the child usually ceases it of his own accord as he grows older.

Pica or perverted appetite is an inordinate desire to eat various substances, such as dirt, sand, mortar, coal, or hair. It is most frequently seen in infants but may occur in older children. This habit is met with in those who are mentally defective, but not rarely in other children. These patients are usually highly neurotic and exhibit some of the other habits common to this class. In some children gastric derangements seem to play the part of an exciting cause. Pica is a common symptom of infection with hook-worm. The habit may continue for years unless corrected. The general health often becomes seriously undermined as a consequence of the disturbed digestion resulting from the presence of abnormal substances in the stomach. Children in whom such a habit is present should in the first place be watched and prevented from indulging in their abnormal craving. Secondly, the digestion and general health should be improved according to indications afforded by the individual case.

Head-banging is an expression of extreme nervous irritability most frequently seen in infants or in very young children. It is not indicative of any special form of nervous derangement, but is caused by the same morbid impulse which leads other nervous children to scratch their faces, pull their hair, etc. While in some children head-banging occurs only occasionally, we have seen patients in whom it existed for a long time. It may be repeated almost every night, and continue at intervals for two or three hours, and that without temper or excitement, but with such force as to produce contusions of the scalp and necessitate padding the sides of the crib. It is rarely a symptom of organic brain disease. Rickets is often associated and the nutrition of most of the patients is much below the normal. The treatment is general.

CHAPTER III

DISEASES OF THE BRAIN AND MENINGES

MALFORMATIONS

The malformations of the brain are of great variety, and many of them are solely of anatomical interest, as the conditions are incompatible with life. Only the most frequent and the best-known types will be mentioned, and those which are of interest from a clinical point of view.

Meningocele, Encephalocele, and Hydrencephalocele.—These three conditions have in common a protrusion of some part of the cranial contents through an opening in the skull. In Meningocele (Figs. 87, 90) there is protrusion of the membranes alone. These form a sac, which

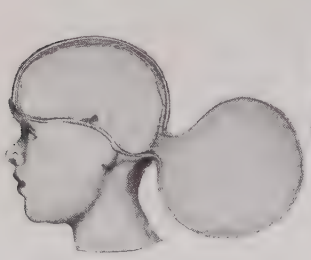


Fig. 87.—Meningocele.

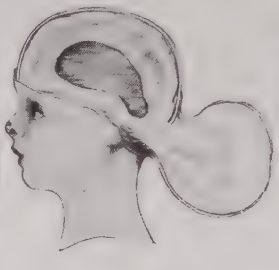


Fig. 88.—Encephalocele.

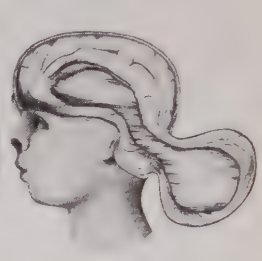


Fig. 89.—Hydrencephalocele.

is usually, but not invariably, distended by fluid. In encephalocele (Fig. 88) there is a protrusion of a portion of the brain substance; this is connected with the rest of the brain by a constricted neck or pedicle. The tumor may or may not contain fluid. In hydrencephalocele (Fig. 89) there is a protrusion of a portion of the brain substance which contains within it a cavity filled with fluid, this cavity communicating with the distended lateral ventricles.

Fig. 90.—Meningocele. Infant one Month old.

In all these conditions there is a tumor, usually pedunculated, of a round or pyriform shape, with a smooth or lobulated surface. The ordinary size is that of a mandarin orange; it may be as small as a walnut, or as large as the patient's head. It is generally covered by the scalp, which is often denuded of hair; but it may be covered only by granulation-tissue, or it may show a central cicatrix, like that of spina bifida. Other deformities, such as spina bifida, club-foot, and hare-lip are frequently present.

All these conditions are rare, but the most frequent and most serious one is hydrencephalocele, this being usually associated with hydrocephalus. The next in frequency is encephalocele, which has the best prognosis. This is frequently termed *hernia cerebri*. If fluid is present, it is external to the brain. In meningocele there is simply an accumula-

tion of fluid, which communicates by a small opening with the general arachnoid cavity of the brain.

Of 105 cases collected by Schatz, 59 occupied the occipital region and 46 were frontal. The aperture through which the occipital protrusion takes place is usually in the median line. It may communicate with the posterior fontanel, with the foramen magnum, or with the cleft of a spina bifida. The occipital bone may be divided in the median line, or rarely it may be absent.

FIG. 91.—FRONTAL MENINGOCELE. Infant Three Months Old.

In the nasofrontal form (Fig. 92) the tumor is usually at the root of the nose, a little to one side of the median line. The aperture is most frequently between the cribriform plate of the ethmoid and the frontal bones. It may be between the lateral halves of the frontal bone, causing a median tumor. The point of protrusion may also be the lateral region of the skull, generally about the lateral fontanel, or along the line of the sutures; it may project into the mouth or the pharynx. These anterior tumors are usually small, although large ones containing the anterior lobes of the brain have been seen.

The theory of the origin of these malformations which is most widely accepted is that they are primarily cases of intra-uterine hydrocephalus, and as the cranial cavity is gradually closed by the development of the bones, a certain portion of the brain is left outside.

FIG. 92.—NASOFRONTAL MENINGOCELE. Infant one week old.

Symptoms.—The tumor is always congenital, although after birth it frequently increases very much in size. A typical tumor is round and elastic, usually giving evidence of fluid; it usually pulsates synchronously with the heart; during screaming or forced inspiration, it increases in size; partial and in some cases complete reduction is possible, but this is usually followed by marked cerebral symptoms, even by convulsions. After partial reduction, an opening in the skull may often be made out. Microcephalus may be present, or there may be unequal development of the two sides of the head.

The following differential points indicate the most characteristic features of the three varieties: In meningocele, the tumor is at first small, but increases; it has a smooth surface; it is pedunculated; there is distinct fluctuation, perfect translucency, rarely pulsation; often it is completely reducible; compression of the tumor causes cerebral symp-

toms; the skull is normal. In encephalocele, the tumor is small and smooth; it is rarely pedunculated; fluctuation is absent; it is not translucent; there is distinct pulsation; it is usually reducible; pressure causes cerebral symptoms; the skull is normal. In hydrencephalocele, there is a large pendulous tumor with an irregular or lobulated surface; it is pedunculated; translucency is rarely complete; fluctuation is distinct; it is irreducible; pressure rarely causes symptoms; microcephalus and other deformities are often associated.

The occipital tumors are usually more serious than the frontal ones. The majority of cases die in the course of the first few weeks of life, death resulting from meningitis, convulsions, or rupture. In meningocele the tumor usually grows slowly, and ultimately may be shut off from the cranial cavity; but gradual thinning of the membrane may take place, and spontaneous or accidental rupture occur. In encephalocele the tumor grows slightly, or not at all. Most of these patients exhibit signs of mental impairment or other evidences of organic brain disease.

Treatment.—According to Treves, operation is justifiable only in case of impending rupture. The conditions present are essentially the same as in spina bifida. Meningocele may be aspirated or the sac may be laid open and a plastic operation performed for the closure of the communication with the cranial cavity; or the skin may be divided, and a ligature or clamp applied to shut off the communication with the brain. All these methods have been at times successful, but recovery in many instances is followed by the development of hydrocephalus. Encephalocele is to be treated by protection and compression. Aspiration may be resorted to if fluid is present. In hydrencephalocele the prognosis is absolutely bad under all circumstances. Schatz gives the following statistics, showing the results with and without operation, all varieties being included: Of twenty-four occipital tumors not operated on, three recovered; of thirty-five operated on by excision, ligation, or injection, six recovered. Of forty-six frontal tumors, there were six recoveries in thirty-two cases without operation, and two recoveries in fourteen cases with operation.

Microcephalus.—This is often regarded as due to premature ossification of the skull; but the hypothesis is certainly inadequate to explain most, if any, of the cases. In many children suffering from marasmus, the sutures ossify and the fontanels close much earlier than in healthy infants of the same age, chiefly because, with the rest of the body, the brain also has almost ceased to grow. In microcephalus the early ossification of the skull is usually due to arrested growth of the brain, and not the reverse. The reasons for the developmental arrest in the brain are for the most part unknown.

It is well known that there is not an invariable relation between the size of the head and the size of the brain, although generally the two correspond. If the circumference of the head is much below the average for the age (see introductory chapters), and relatively much less than the measurements of the rest of the body, microcephalus may be assumed to exist. Sachs calls attention to the fact that the circumference of the head may be nearly normal and yet the essential conditions of microcephalus exist, owing to imperfect development of the anterior part of the brain.

The symptoms of microcephalus are those of mental deficiency and cerebral paralysis, existing in all possible combinations and with variable degrees of severity.

The essential condition in microcephalus being an arrest in the development of the brain, it is not difficult to understand why the operation of craniectomy once thought promising has been generally abandoned. The results do not justify any operative measures yet proposed for the relief of these cases.

Congenital Hydrocephalus.—These cases may fairly be considered as belonging in this group, although they are discussed elsewhere.

Porencephalus (literally, a hole in the brain) is a condition in which there is a large depression in some part of the brain, but with surrounding parts well developed. Such depressions may involve a whole lobe, and they may be deep enough to reach the lateral ventricles.

Porencephalus is described as congenital or acquired. In the congenital form, the defect is usually found in the anterior or middle part of the brain. The origin of these conditions is still a disputed question. They are probably due to early vascular changes. Children sometimes live several years with very large defects, the symptoms depending upon the seat of the lesion. The acquired form of porencephalus is usually one of the late results of meningeal hemorrhage. It may affect one or both sides. Such cases present the symptoms of spastic paralysis—usually diplegia. In all cases with large brain defects, the space is filled with fluid.

PACHYMENINGITIS

Pachymeningitis, or inflammation of the dura mater, occurs both as an acute and a chronic disease.

Acute Pachymeningitis.—This is very rare in children. Only pachymeningitis externa is generally included under this term, as acute pachymeningitis interna does not occur alone, but usually with inflammation of the pia mater (leptomeningitis). It may be associated with disease or injury of the bones of the skull, but is most frequently seen in con-

nection with middle-ear disease. It generally begins as a localized process, but the inflammation may extend to the inner layer of the dura, and to the pia mater; or it may remain circumscribed, and terminate in the formation of an abscess between the dura mater and the bone.

The symptoms of acute pachymeningitis are distinctive only when the process is localized. They are then usually associated with middle-ear disease, and are indistinguishable from those of cerebral abscess. The treatment is surgical.

Chronic Pachymeningitis.—This, in children, almost invariably affects the inner layer of the dura mater (pachymeningitis interna); it is also known as *pseudo-membranous* and as *hemorrhagic pachymeningitis* or *hematoma of the dura mater*. Its causes are for the most part unknown. It is a rather rare condition, being usually discovered at autopsy in children, chiefly cachectic infants, who have died of other diseases.

Two classes of cases are to be distinguished—those with, and those without extensive hemorrhages. In the latter group there is found a thin, translucent, vascular membrane lining the inner surface of the dura. It may be only a delicate film which can be scraped off; it may be as thick as ordinary blotting-paper, or even twice that thickness. The membrane is often edematous; it is exceedingly vascular, and the vessels have very thin walls. There are usually scattered punctate hemorrhages, and there may be a few of larger size. This membrane may cover the whole inner surface of the dura, but in most cases it is principally over the convexity and may be found only here; it is apt to be more upon one side than upon the other. In cases of long standing there may be adhesions between the dura and the pia. When large hemorrhages have taken place, quite a different pathological appearance is presented. The lesions found in one of our cases are fairly typical: The infant was six months old, and the symptoms had existed for six days. The fontanel was bulging to a marked degree, and the sagittal and coronal sutures were separated. A thin recent clot from one-eighth to one-fourth of an inch in thickness covered nearly the whole of the right hemisphere and part of the convexity of the left. The entire dura was lined both at its convexity and base by a pseudo-membrane of grayish color, about one-sixteenth of an inch in thickness. The brain was anemic.

In cases of longer standing partial organization of the clot may be seen; in more recent ones the blood is partly or entirely fluid. We once saw acute leptomeningitis with a purulent exudation, associated with hemorrhagic pachymeningitis. In cases where life is prolonged for years, there may be partial or even complete absorption of the clot, followed by the formation of cysts, considerable inflammatory thickening

of the pia with deposits of blood pigment, and finally atrophy and sclerosis of the cortex. The source of the hemorrhage may be the rupture of a single large vessel, but more frequently the blood comes from many small vessels.

Symptoms.—These are due to the hemorrhage, and not to the inflammatory process. Until hemorrhage occurs there are no symptoms by which the disease can be recognized. Thus in many of the cases in which pachymeningitis is found at autopsy, its existence is not suspected during life. The occurrence of hemorrhage is sometimes marked by vomiting or convulsions, and usually there is loss of consciousness. It may be a question whether the convulsions are the cause or the result of the hemorrhage. In most cases they seem to be the result. They are usually general and repeated. If the hemorrhage occurs slowly, there may be stupor without convulsions until nearly the end. In the fatal cases the symptoms generally continue from two days to a week. There are dulness, stupor, and finally coma, death occurring in coma or convulsions. If the hemorrhage is diffuse—and this is apt to be the case—there is rigidity of all the extremities; if it is of one side only, the rigidity affects only one arm and leg. The pupils are more frequently contracted, but may be dilated or unequal. There is diplegia, hemiplegia, or monoplegia, according to the seat and extent of the hemorrhage. The respiration is slow and irregular and may be of the Cheyne-Stokes variety. The pulse is slow, irregular, and sometimes intermittent. The temperature is at first normal, but rises slowly until death occurs, when it is from 100° to 103° F. Generally the cranial nerves are not affected, and opisthotonus is absent. The knee-jerk is often exaggerated. In cases which do not prove fatal—these being chiefly in older children—we have a similar onset, but after a few days consciousness is regained, and only hemiplegia or monoplegia remains. The course of the paralysis is that seen after meningeal hemorrhage due to other causes. Wagner has reported a case in which recurring hemorrhages took place at intervals of several months, the autopsy showing distinct evidences of both old and recent lesions.

Pachymeningitis, we are inclined to believe, plays a more important rôle in the production of meningeal hemorrhages in children than has generally been accorded to it. From the frequency with which this lesion is found as a cause of sudden meningeal hemorrhages which are fatal, it is not unlikely that some of the cases which recover with hemiplegia or monoplegia, may be due to the same cause.

The prognosis depends upon the age of the patient and the extent of the hemorrhage. Extensive hemorrhages are usually fatal in infancy, but small ones are seldom so, for they are rarely at the base. The prognosis of the paralysis in cases not terminating fatally is the same as

after meningeal hemorrhage due to other causes, with perhaps an added liability to recurrent attacks.

Without large hemorrhages, pachymeningitis interna can not be diagnosticated; and it is impossible to differentiate the hemorrhagic cases from other varieties of meningeal hemorrhage. It is important to make a diagnosis between pachymeningitis with hemorrhage, and acute meningitis. In the former there is a sudden onset; stupor occurring early, usually on the first day, gradually diminishing in cases of recovery, or deepening into coma in fatal cases; localized or general paralysis, also occurring early; there is no fever in the beginning, and only moderate fever at the close. In acute meningitis there is usually a higher temperature, especially early in the disease; coma develops later, and rigidity of the extremities is less pronounced. However, when the hemorrhage occurs in the course of some other disease, a differential diagnosis may be impossible without lumbar puncture.

Treatment.—The treatment of hemorrhagic pachymeningitis is symptomatic. The indications are, to relieve cerebral congestion by applying ice to the head, to allay irritative symptoms by the use of bromids, and to keep the patient perfectly quiet.

ACUTE MENINGITIS

Several different varieties of acute meningitis are met with in children. Cerebrospinal meningitis is the only form which occurs epidemically; but this is also seen as a sporadic disease. It is due to a specific organism, the meningococcus. There are several other forms of acute meningitis which more or less closely resemble cerebrospinal meningitis clinically, and which were for a long time confounded with it. Pneumococcus and influenza meningitis are usually secondary inflammations, but sometimes are apparently primary. The typhoid bacillus and the gonococcus may cause acute meningitis, but very rarely in children. Acute meningitis may be due to any of the pyogenic organisms. This is sometimes spoken of as "septic" meningitis, and is almost invariably secondary. Finally, there is tuberculous meningitis, altogether the most common variety in young children except during epidemics of cerebrospinal meningitis.

Some idea of the relative frequency of the different forms of acute meningitis as seen apart from epidemics, may be gained from the following figures which give the number of cases occurring in the Babies' Hospital for a series of years, the diagnosis in every case being made by lumbar puncture or by autopsy. The patients were nearly all under three years of age. The organism found was as follows:

Tubercle bacillus	157	cases
Pneumococcus	23	"
Meningococcus (sporadic)	24	"
Staphylococcus or streptococcus	11	"
Influenza bacillus	5	"
Colon bacillus	1	"

CEREBROSPINAL MENINGITIS

(*Epidemic Meningitis; Cerebrospinal Fever*)

Epidemics of cerebrospinal meningitis are separated by quite long intervals and occur without any assignable cause. The following chart (Fig. 93) represents the prevalence of the disease in New York City during forty years. But little was seen of cerebrospinal meningitis until the epidemic of 1872. Since that time a certain number of deaths from this cause have occurred each year; but there have been seen about once

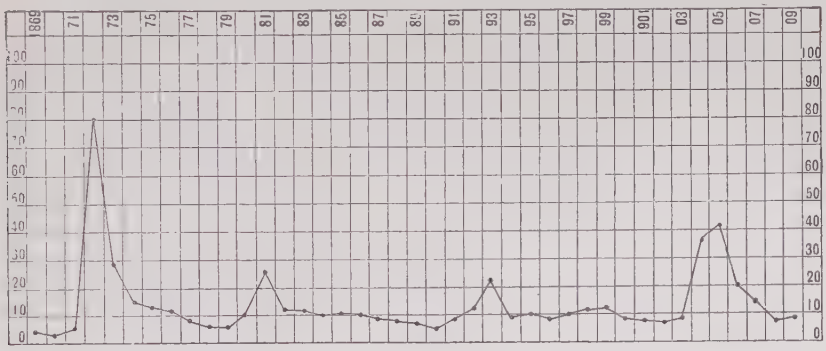


FIG. 93.—CHART SHOWING DEATHS FROM CEREBROSPINAL MENINGITIS IN NEW YORK CITY, FOR FORTY YEARS, PER 100,000 OF POPULATION.

in ten years epidemics of greater or less severity. The most important one was that of 1904-5. After each epidemic, for two or three years, the disease is prevalent, but it occurs with gradually lessening frequency until the average incidence is reached. What has been said of New York is true of almost every large city. In remote country towns, epidemics are occasionally witnessed, and after prevailing a few months the disease disappears as mysteriously as it came. Epidemics are usually seen in the winter and early spring, lasting for several months, generally reaching their height in March or April and slowly subsiding as warm weather approaches.

With reference to the cause of epidemics very little is known. When the disease prevails in cities it occurs especially in crowded tenements, being relatively infrequent in private houses.

Cerebrospinal meningitis has only recently been included among the communicable diseases. In a series of observations made by the New York Health Department the meningococcus was found in the nasal secretion of fifty per cent of the cases of meningitis examined during the first two weeks of the disease. It was found in the nasal mucus in ten per cent of the persons in close contact with cases. In Flexner's experiments upon monkeys he found the organism in the nasal mucus after animals had been inoculated by way of the spinal canal. These observations indicate that the nasal mucosa is a common avenue of infection and probably also a channel of elimination. The degree of communicability when compared with the common contagious diseases seems very slight. In fully seventy per cent of the cases investigated in the New York epidemic of 1904-5, but one person in a household was affected, although no effort at isolation was made. We have never known the disease to originate in a hospital patient, although in New York cases of cerebrospinal meningitis have been until recently received into the general wards with other patients. Sporadic cases of meningitis occur after epidemics, and quite apart from them without apparent cause, and it is very exceptional that any connection with a previous case can be established. About fifty per cent of the cases of cerebrospinal meningitis occur in children under five years, and about twelve per cent in those under one year. The youngest case we have seen was in an infant six weeks old.

The specific organism of cerebrospinal meningitis is the diplococcus intracellularis of Weichselbaum or, as it is now generally designated, the meningococcus. It is present in the meningeal exudate, in the cerebrospinal fluid obtained by lumbar puncture, and in some cases can be demonstrated in the blood, the lungs and other organs, sometimes in the large joints. It is almost invariably found in pairs or tetrads within the leucocytes. It is decolorized when stained by Gram's method. Outside the body the organism is unknown.

Lesions.—In epidemic meningitis death may take place so early that the changes found at autopsy are slight. There may be only a serous exudation and intense hyperemia, which is doubtless much less marked after death than during life. The cerebrospinal fluid is turbid and much increased in amount. The microscope, however, may show, even in these early cases, an abundant exudation of leucocytes in the pia mater. After the third day the lesions are quite uniform. The convolutions appear somewhat flattened from pressure due to distention of the ventricles. The inner surface of the dura is usually normal or only congested. There may be thrombi in any of the cerebral sinuses, or in the meningeal veins of the convexity. There is an exudation of greenish-yellow fibrin, which is sometimes very abundant. It is generally widely

distributed, but is usually most marked over the anterior half of the brain and at the base. In some cases it is limited to the base, but very rarely limited to the convexity. There is an increase in the quantity of cerebrospinal fluid. The ventricles are moderately distended with serum or sero-pus, and their walls may be slightly softened. The brain substance of the cortex may be reddened or may appear normal. In the meninges of the cord, lesions similar to those of the brain are usually seen. The exudation is principally upon the posterior surface, and may extend throughout the entire length of the cord, or be limited to its upper or to its lower portion.

Microscopical examination shows the exudation to consist of fibrin and pus cells, which infiltrate the pia mater. The superficial layers of the cortex in the inflamed areas often show minute hemorrhages and very marked cell-infiltration. Minute abscesses may be present. Very marked degenerative changes can usually be demonstrated in the nerve cells themselves. The cells of the neuroglia are also affected; they are swollen and increased in number; and there may be proliferation of the connective tissue about the blood vessels. Changes similar to those just described may be found in the cord, but these are less frequent and as a rule much less severe than those in the brain. Inflammatory products are sometimes present in the central canal of the cord and in the walls of the lateral ventricles of the brain. The inflammatory process frequently extends along the cranial nerves, especially the auditory and optic, and this may result in otitis or choroiditis; from the cord, it may extend along either the anterior or posterior nerve roots. Descending degeneration is found in the nerves both of the brain and the cord.

In patients that die after the disease has lasted two or three months, the later results of these lesions may be seen. There is usually present a chronic meningo-encephalitis, sometimes diffuse, sometimes localized. The pia mater is cloudy, thickened, and frequently adherent to the brain. Here and there are seen small, yellow, opaque patches which are the result of fatty changes in the cells and fibrin of the exudate, with some proliferation of connective tissue. The lesions are usually most marked at the base, where the thickening of the meninges and the adhesions may lead to the development of a secondary hydrocephalus.

In cases which have lasted a much longer time very marked changes are found in the brain substance. There may be generalized meningeal adhesions, with a diffuse cortical atrophy, but more frequently there are areas of sclerosis, especially over the frontal and temporosphenoidal lobes, with which there are almost always associated marked descending degenerative changes in the cord. Such lesions are, of course, permanent, and seriously interfere not only with the functions, but also with the growth and development of the brain.

The lesions and their effects are well illustrated by one of our patients who died six months after an attack. She was a bright little girl of four and a half years, and had a typical attack of meningitis of moderate severity. Convalescence was slow, but at the end of two months recovery was perfect in everything but her mental condition. She remembered nothing which she had previously learned in the kindergarten, where she had been an exceptionally bright pupil. Her mind was a blank. She was dull, listless, and her face had a vacant, idiotic expression. The special senses seemed unaffected, and her speech was retained. She died during an attack of convulsions. At the autopsy the pia was everywhere thickened and adherent, while in the cortex were present the earlier changes of a general encephalitis.

The visceral lesions most frequently found in epidemic meningitis are pulmonary. There may be lobar or bronchopneumonia, and in the lungs may be found the same organism as in the brain. Acute degeneration of the liver and kidneys is also frequent. The other viscera are seldom affected. Occasionally suppurative inflammation of the joints occurs.

Symptoms.—The symptoms of cerebrospinal meningitis do not differ essentially in the sporadic and epidemic cases, except that the most severe forms of the disease are seen in the latter. They may be divided into several quite distinct groups:

1. *Hyper-acute Form.*—Cases of this kind are rarely seen except in an epidemic, and usually occur at its height. The onset is very abrupt, the course short and intense, and death may take place in from twelve to thirty-six hours. The following case illustrates this type: A little girl of ten years was well enough at 2 P.M. to carry a bundle of clothes a dozen city blocks. Returning home, she complained of intense headache, vomited frequently, and was so weak that she was obliged to go to bed. In a few hours she passed into deep coma, with very high fever, and died at 11 P.M.

The earliest symptoms are usually intense headache, repeated attacks of vomiting, and very high fever. There is great prostration and the nervous symptoms increase so rapidly that in a few hours the patient may become comatose and death occur in a short period. The temperature rises rapidly to 103° or 104°, sometimes to 106° F. A few petechial spots may be discovered over the face, chest, or extremities. There is usually no rigidity, but rather general relaxation. The pulse is weak, in most cases rapid, but sometimes slow and irregular. The respiration is usually irregular both in frequency and depth.

The symptoms appear to be due to two factors: the intensity of the infection, and the rapid accumulation of cerebrospinal fluid, causing coma with eventual respiratory paralysis. Usually both these factors are present, but the second one seems the more important. In support

of this view is the striking infrequency of cases of this type in infants with an open fontanel. Should the patient survive the violence of the onset, a period of reaction occurs, and after a day or two the disease follows the regular course.

2. *Usual Form.*—In this also the onset is generally abrupt, but not so violent as in the cases just described. It may be marked by intense headache, vomiting, convulsions, delirium, chills, and fever with general hyperesthesia and rigidity. The initial temperature is from 101° to 104° F. Opisthotonus, with severe pains in the back of the neck and along the spine, and general muscular rigidity are usually present. There is often active delirium, but rarely stupor or coma. The pulse

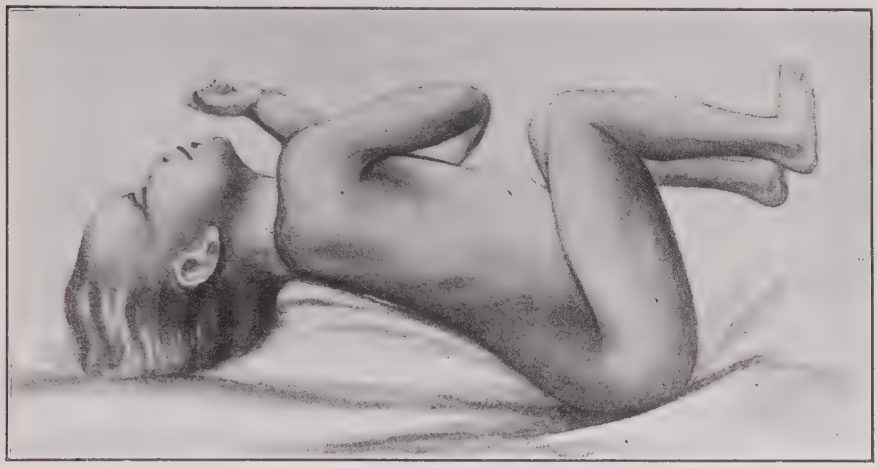


Fig. 94.—Posture in Cerebrospinal Meningitis. (Smith.)

is generally rapid, 120 to 150, and sometimes irregular. The respiration is often slightly irregular, and it may be rapid or slow. The eruption is not so frequently seen as in the very acute cases.

As the disease progresses, the nervous symptoms often change but little from day to day for two or three weeks. They are mainly of the irritative type—moderate delirium, extreme hyperesthesia, tremor and muscular rigidity. The posture is quite characteristic (Fig. 94). Owing to the opisthotonus the child can not lie upon the back, but rests upon the side, with arched spine and neck, and general flexion of the extremities. There is a rather rapid loss in weight, steadily increasing prostration, and a weak, rapid pulse. The bowels are usually constipated. From time to time attacks of vomiting occur. In many cases there is considerable difficulty in feeding. The duration of this form of the disease without specific treatment is from three to six weeks. The course is often marked by periods of remission and exacerbation. If recovery is

to take place, the temperature gradually falls to normal and often at times it is subnormal. The mind becomes clear, and one by one the nervous symptoms disappear, the muscular rigidity being usually the last to go. Convalescence is always protracted.

In cases ending fatally, the patient usually passes into a deep stupor or coma, with extreme prostration, a slow, weak, irregular pulse, shallow respiration of the Cheyne-Stokes variety, sunken abdomen, general relaxation, and death occurs from exhaustion or from bronchopneumonia.

Occasionally the attack is much prolonged, the fever and all the active symptoms continuing from eight to twelve weeks. Emaciation sometimes becomes extreme, and with a few nervous symptoms may continue long after the fever ceases. In infants, death is often due to marasmus. While a fatal outcome is more frequent in these prolonged cases, a few recover completely, even when marked symptoms have lasted for eight or ten weeks.

3. *Mild Form.*—Especially toward the end of an epidemic, and sometimes occurring sporadically, there are seen cases which in their onset and for the first two or three days resemble those just described; but instead of running the usual course, the fever and the nervous symptoms subside rapidly and convalescence is established early.

4. *Chronic Form.*—Owing sometimes to the extent, sometimes to the position of the lesions, the disease does not subside at the usual time, but nervous symptoms continue after the temperature and most of the other constitutional symptoms have passed away. These cases are chiefly of the basilar type, and often lead to the development of chronic basilar meningitis with secondary hydrocephalus. They are more fully considered in a later chapter.

Onset.—One of the most striking features of this disease is the abruptness with which it develops. Occasionally there are indefinite symptoms for a day or two before active symptoms begin; but in the great majority not only the day, but the hour of the onset is definitely marked. The most frequent initial symptoms are the simultaneous occurrence of severe headache and vomiting, followed by high fever and marked prostration. The vomiting is usually repeated, projectile, and has no relation to meals. Convulsions occurred in the beginning of thirty per cent of our cases. Occasionally a decided chill is seen. After twenty-four hours acute general pains and hyperesthesia are usually present, together with rigidity of the muscles of the neck and extremities, giving rise to opisthotonus and muscular contractions.

Skin.—Eruptions upon the skin vary much in frequency in different cases and in different epidemics. The most characteristic one is the appearance of small punctate hemorrhages, resembling flea bites; they are not numerous, but may be found on almost any part of the body,

most frequently upon the extremities, the upper part of the chest, and neck. In our experience they have been present in about fourteen per cent of the cases. Sometimes larger hemorrhages are present. We have twice seen a very extensive purpuric eruption with hemorrhagic areas from half an inch to three inches in diameter over the face, buttocks, and extremities. This eruption belongs to the early stage of the disease and is rarely visible after the third or fourth day unless unusually extensive. In some cases a general erythema is present; in others, an eruption closely resembling measles. Herpes upon the lips and face is common in older children, but is rare in infants. Bed-sores are very common in protracted cases. They are found over pressure points—the trochanter, the malleoli, and the side of the head; in several instances the ear has been the part affected.

Nervous System.—Headache is a frequent initial symptom and is usually severe; it is more often frontal than elsewhere, and may be associated with vertigo. There are acute pains in the back of the neck, along the spine, and marked general hyperesthesia, which is often so intense that any movement of the body causes agonizing cries. This is one of the most striking symptoms of the disease, and may continue throughout the acute stage. The mental state varies much in different cases. Delirium is frequent in the early stage of the severe form; it is usually active, sometimes maniacal. After delirium dulness or apathy ensues, giving place to great irritability when the patient is disturbed. Convulsions are not uncommon early, but are seldom repeated in the course of the disease or toward its close. There is rarely continuous stupor or deep coma except toward the end of fatal cases. In many cases with high temperature and quite severe symptoms, after the subsidence of a short early stage of excitement or delirium, the mind remains perfectly clear throughout the attack. In these circumstances an erroneous diagnosis is often made, particularly if the physician has not observed the case from the beginning.

Tonic spasm of the various muscular groups is one of the most characteristic features of this disease and is seldom absent. Like the hyperesthesia it is persistent. The rigidity and contraction of the muscles of the neck and back produce cervical or general opisthotonus; cervical opisthotonus is most marked with lesions chiefly at the base, and may be wanting in the rare cases when the lesion is almost entirely at the convexity. Tonic spasm of the extremities usually causes general flexion of the thighs, legs, and arms. Late in the disease this may be replaced by complete extension of the lower extremities with dropping of the feet. The tonic muscular spasm gives rise to Kernig's sign, viz., inability to extend the leg when the thigh is flexed upon the body. In young children one should not place too much dependence upon this sign.

While rarely wanting in cerebrospinal meningitis, it may be present in other conditions. Brudzinski's sign is frequently present, but not diagnostic. Muscular rigidity is one of the most constant symptoms of cerebrospinal meningitis and one of the last to disappear. It may be absent in the early stage of the hyper-acute cases, and very late in fatal cases, when there may be general relaxation. Other nervous symptoms frequently present are ankle clonus, muscular tremor, especially of the hands, and paralysis, which may be facial, monoplegic, or hemiplegic. Early in the disease the knee-jerks are usually increased; in the later stages they may be lost.

Eye and Ear.—The pupils in the early stage are generally contracted; toward the close they are usually widely dilated. Ocular paralyses are not so frequent or so marked as in tuberculous meningitis. The same is true of the changes in the optic disc, although these vary much in different epidemics. There may be congestion of the fundus, retinitis, or optic neuritis. In some epidemics such changes have been observed in fully half the cases. In that of 1904-5, in our hospital cases, they were rarely seen, and then were but slightly marked. Conjunctivitis is frequently present and may be severe. There may be choroiditis and sometimes complete destruction of the eye, but usually this is unilateral. In most epidemics the ears are more frequently affected than the eyes. Early deafness may be due to a lesion of the auditory nerve, is generally bilateral, and often permanent. Acute otitis media occurs as a complication, and the meningococcus is occasionally found in the discharge. Permanent deafness is sometimes due to changes in the auditory nerve or in the brain itself.

Fever.—This disease is usually attended by high fever, but the curve is apt to be an irregular one and show wide variations. The temperature is nearly always high at the onset; in the hyper-acute cases it may reach 106° F. or higher. The usual range during the disease is from 100° to 105° F. (Fig. 95). Sometimes it is steadily high; not infrequently a few days after a sharp acute onset it falls nearly or quite to normal and remains there for several days. Cases seen in this afebrile period are most difficult of diagnosis. This stage may be followed by another sharp rise, and afterward continuous fever. Periods of remission and exacerbation in the temperature are seen in a large proportion of the prolonged cases. Often it becomes subnormal. The temperature may bear no relation to the severity of the other symptoms. Its course is greatly modified by the serum treatment.

Respiration is disturbed very early in the disease, when it is often irregular and may be slow or rapid. Throughout the greater part of the attack it may be nearly normal. Occasionally it is of the typical Cheyne-Stokes variety.

Pulse.—Throughout the greater part of the disease the pulse is rapid. In the early stage it is often weak, and sometimes irregular. The average frequency in young children is from 130 to 150. A slow, irregular pulse is occasionally seen late in the disease in patients who are in deep coma.

Blood.—A leucocytosis is present in nearly all cases. The average is from 25,000 to 40,000. The increase is chiefly in the polymorphonuclear cells which usually form from 80 to 85 per cent. of the leucocytes. Blood cultures made early in the disease have in some cases shown the presence of the characteristic organism.

Digestive System.—Vomiting is one of the most frequent symptoms of onset but rarely persists throughout the attack. Late in the disease

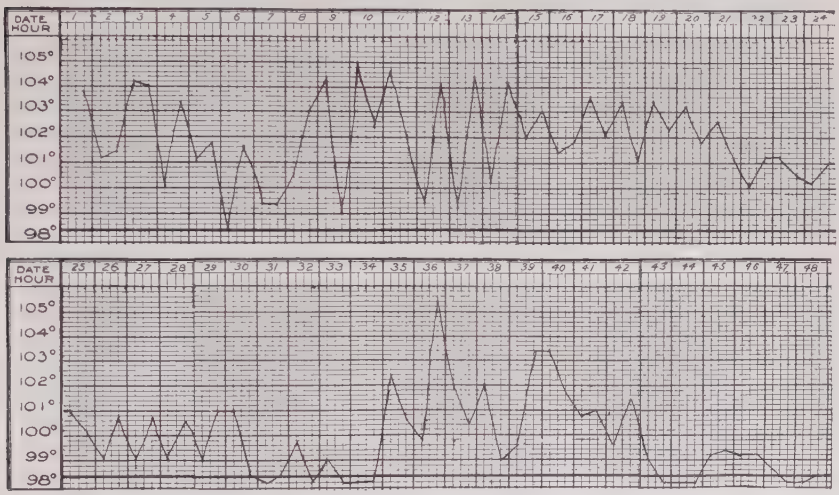


FIG. 95.—CEREBROSPINAL MENINGITIS. Recovery without serum treatment. Fairly typical chart of prolonged case, showing remissions and exacerbations. Patient 3½ years old; unconscious, blind, and deaf for 2½ months; complete recovery.

it may be most troublesome. As a rule constipation is present. The tongue is coated, dry, glazed, sometimes covered with sordes. In a small proportion of cases jaundice has been observed. On account of the loss of appetite, great irritability, delirium, and stupor, the greatest difficulty is often experienced in feeding these patients. In young children gavage is much more satisfactory than rectal feeding. Early in the disease the abdomen is natural. In the late stage it is often very much retracted.

General Nutrition.—This is impaired in nearly all cases. There is a progressive wasting, greater than would be explained by the disturbance of digestion. In the protracted cases it may be extreme. Infants and young children often die of inanition or marasmus long after the active symptoms of the disease have subsided.

Other symptoms of importance are the tense, bulging fontanel, in

infants rarely absent early in the attack, but often wanting in the late wasting stage; incontinence of urine and feces, and retention of urine, are very frequent and often overlooked; occasionally swelling of some one of the large joints is seen.

Course, Duration, and Termination.—Excluding the hyper-acute cases in which death occurs very early, the usual duration of active symptoms in cases not treated with serum is from three to six weeks. Of 350 cases recovering without serum, the disease lasted less than one week in three per cent; in fifty per cent it was five weeks or longer. Some very protracted cases terminate favorably. We have seen one child recover completely after 84 days of fever, and another after 102 days. Most of the prolonged cases are marked by periods of exacerbation and remission. Not until the temperature has been normal for several days, the mind has become clear, and the hyperesthesia and rigidity have entirely disappeared, can we consider convalescence as established. Recovery is slow, and it may be many months before the child is quite well. In 220 cases receiving serum treatment the average duration of active symptoms after the first injection was 11 days.

In fatal cases, death may come early from coma, convulsions, or heart failure. It may occur in the middle period from complications, most frequently pneumonia, or the terminal stage of the disease may be seen with extreme wasting, and finally death from exhaustion.

Complications and Sequelae.—The chief complications are pneumonia, otitis, conjunctivitis or choroiditis, and bed-sores; rarely, nephritis and arthritis. Sequelae are, unfortunately, very common. There may be perfect recovery so far as physical functions are concerned, but the child be left mentally deficient. In some cases the defect is so slight as not to be evident for several months or even years; in others the mental faculties are entirely lost. There may also be various types of paralysis—strabismus, facial paralysis, monoplegia, hemiplegia or diplegia, and often contractures, which are sometimes temporary, but apt to be permanent. The acute attack may be followed by chronic meningitis with hydrocephalus. Deafness is quite common, usually of both ears, and deaf-mutism is not an infrequent result in young children. Blindness is not so common and is usually unilateral. As a late result epilepsy may develop.

Prognosis.—The mortality is usually higher in epidemics than when the disease occurs sporadically. It is usually greater at the height of an epidemic and lower at its close. The average mortality before the serum treatment was about 70 per cent. We know of no recorded epidemic in which the mortality was less than 50 per cent. In the last year (1905) of the New York epidemic, of 1,780 cases tabulated by the Department of Health the mortality was 76 per cent. Of 59 cases treated

in our hospital wards in the same epidemic the mortality was 80 per cent, nearly all these patients being under three years of age. Of 24 cases under one year only one recovered. Of the cases seen in private practice, largely older children, the mortality was 50 per cent. None of these had serum treatment. Not all of those who do not die are to be classed as recoveries, for in fully 25 per cent serious sequelae remain. The results with serum are referred to under Treatment.

Diagnosis.—Lumbar puncture is the only accurate means of diagnosis we possess. By it we can not only differentiate meningitis from other diseases with nervous symptoms, but can distinguish this from other varieties of meningitis. Furthermore, this is possible very early in the disease. With proper precautions it is practically free from danger, and it should be employed whenever meningitis is suspected. The procedure is not difficult, but the technic is important.[1] The quantity of fluid which may be removed at one time varies from a few drops to three or four ounces. During the first day or two it is usually slightly cloudy; sometimes it is very turbid and it may be thick and purulent. As the disease progresses the pus cells gradually diminish, and in favorable cases disappear, but may reappear with an exacerbation of the symptoms. These changes are much modified by serum injections.

The presence of many leucocytes in the cerebrospinal fluid indicates meningitis, which may be due to the meningococcus, but also to the pneumococcus, the influenza bacillus, the staphylococcus, or the streptococcus. The variety can be determined only by microscopical examina-

[1] Puncture should not be attempted with an ordinary surgical exploring needle, but with the special lumbar needle devised by Quincke. This is merely a fine trocar and cannula and is made stronger than an exploring needle, which may break. The child is placed upon the right side with the thighs tightly flexed against the abdomen to separate the spines and laminae of the vertebrae as much as possible. The point chosen for puncture is in the median line between the third and fourth lumbar vertebrae. This is on a level with the highest part of the iliac crest. The strictest asepsis is required. The skin should be cleansed and painted with iodin and the needle boiled. The pain is no greater than from exploratory punctures elsewhere. No anesthetic is necessary for infants, but sometimes is required for older and especially sensitive or nervous children unless they are comatose. Local anesthesia may be employed or a few whiffs of chloroform given, but always with caution, for the combined shock of the puncture and the chloroform is sometimes considerable. The child should be closely watched for at least fifteen minutes after the puncture is made. The canal is reached at the depth of about one inch. The trocar is now withdrawn and the fluid usually flows freely through the cannula, sometimes spurting forth some distance, owing to high pressure. A dry puncture is generally due to the fact that the canal has not been entered; sometimes, because the exudate is too thick to flow through the small needle, or the needle may be plugged. Raising the patient to a sitting posture usually causes a freer flow, as does also flexing the head upon the chest if opisthotonus is extreme.

tion of stained smears from the sediment of the fluid obtained after standing or after centrifuging, and by cultures, which should be made immediately after the fluid is withdrawn. In cerebrospinal meningitis diplococci are found within the pus cells and some are also free in the fluid. The organisms are usually numerous.

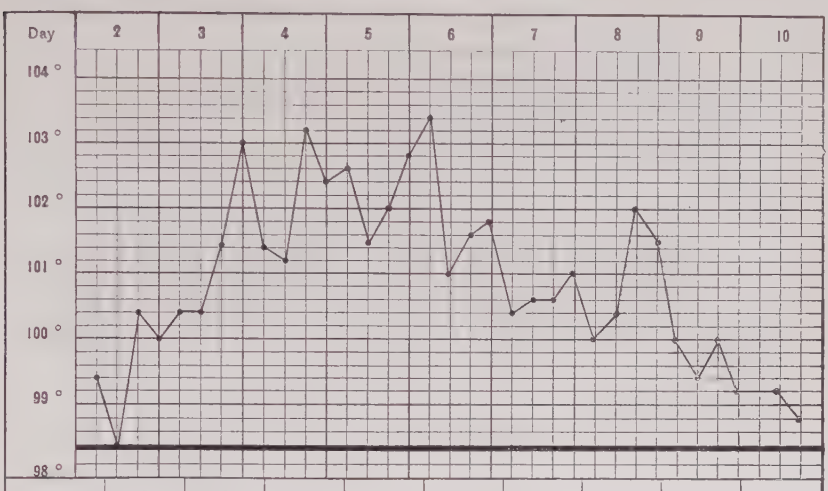
The diagnostic value of lumbar puncture, when properly performed, is very great; not only are positive findings conclusive, but early negative findings almost certainly exclude meningitis. Exceptional cases are occasionally met with in which early punctures give a clear fluid and no organisms are found; a few days later the fluid becomes turbid and organisms are abundant. The meningococcus may persist for a long time. In one of our cases not treated by serum it was present on the ninetieth day.

The diagnosis of cerebrospinal meningitis by symptoms alone presents peculiar difficulties at the beginning of the attack. The most valuable early symptoms for diagnosis are, a sudden onset with intense headache, vomiting, high temperature, prostration, the petechial eruption, marked rigidity of the neck and extremities, with hyperesthesia, great irritability or early stupor. Later, three symptoms are rarely wanting—persistent hyperesthesia, muscular rigidity of the neck and extremities, and fever. Kernig's sign is seen in other conditions and is not diagnostic. The spinal symptoms are more to be relied upon for diagnosis than are the cerebral symptoms. The mind in some cases remains perfectly clear; in others there is delirium, but seldom continuous, deep coma.

At its beginning, cerebrospinal meningitis may be confounded with pneumonia or other diseases with cerebral symptoms. It is differentiated with certainty only by lumbar puncture. It is sometimes difficult to distinguish cerebrospinal from tuberculous meningitis and from acute poliomyelitis with meningeal symptoms. Cerebrospinal meningitis is relatively infrequent except in epidemics. The fluid is usually turbid and contains many cells of the polymorphonuclear variety; in tuberculous meningitis the fluid is clear and the few cells found are nearly all lymphocytes. Tuberculous meningitis may occur anywhere or at any time. Its characteristics are a gradual onset with indefinite symptoms, low temperature, persistent drowsiness, irregularity of pulse and respiration, absence of active delirium, late coma, less marked hyperesthesia and rigidity, duration seldom over three weeks from the beginning of definite cerebral symptoms, termination invariably fatal. Cerebrospinal meningitis, however, frequently ends in recovery, and it is the only form of acute meningitis which does so. In poliomyelitis the spinal fluid resembles that of tuberculous meningitis.

Treatment.—Flexner's serum for the treatment of cerebrospinal meningitis is more effective in controlling the disease than any other measure thus far proposed. It is obtained by immunizing horses with

toxins and cultures obtained from many strains of the meningococcus. It acts chiefly on the bacteria themselves; i. e., it is a bacteriolytic serum. It is used as follows: After withdrawing by lumbar puncture all the fluid that will flow readily, under the strictest aseptic precautions, the serum, warmed to the body temperature, is injected without removing the needle. In some exceedingly sensitive patients the administration of



Day	2	3	4	5	6	7	8	9	10
Leucocytes		20,400	25,600	15,000	16,400	16,000	12,500	21,000	20,000
Serum Injected	40 c.c.	30	35	30					
Fluid Removed	80 c.c.	40	40	40			55		20
Nature of Fluid	Purulent	Slightly Turbid	Slightly Turbid	Almost Clear			Clear		Clear
Organisms	Many	Few	None	None			None		None

FIG. 96.—CEREBROSPINAL MENINGITIS TREATED BY SERUM. Infant, 7 months old, Babies' Hospital: 24 hours ill; intense prostration; respiration, 80; signs of pulmonary edema; general relaxation; stupor; profuse hemorrhagic eruption. First fluid, purulent; amount removed, amount of serum injected, and the changes in the fluid shown in the chart. Immediate improvement in symptoms after first injection. Subsequent symptoms typical. A rise in temperature on the 8th day and the increase in leucocytes on the 9th and 10th days suggested relapse; but as the fluid was clear and no organisms could be found in smears or by culture no more serum was given; complete recovery.

a few whiffs of chloroform may be necessary. The injection is made by gravity, using a rubber tube and small funnel. It should be made very slowly, occupying several minutes. Raising the hips facilitates the inflow of the serum. To be effective, it must be brought into contact with the organisms in the spinal canal in a considerable degree of concentration.

The initial dose of the serum now used is 10 to 15 c. c. for infants, and 15 to 25 c. c. for children from two to twelve years old. The dose is usually repeated in twenty-four hours (in very severe cases in twelve

hours) and a daily dose thereafter until four or five have been given. The indications for further injections are: continuance of marked nervous symptoms, persistence of temperature, persistence of leucocytosis and of great numbers of polymorphonuclear cells in the cerebrospinal fluid, even though no organisms are found in smears and there is no growth from cultures. To introduce more serum than the amount of fluid withdrawn is somewhat hazardous. In the milder cases it sometimes happens that a single dose may suffice for a cure; but even in such circumstances it is safer to give at least three doses on successive days. The serum arrests the inflammatory process by destroying the organisms which produce it. To accomplish this a sufficient dose must be given, and given early, before important inflammatory changes have taken place.

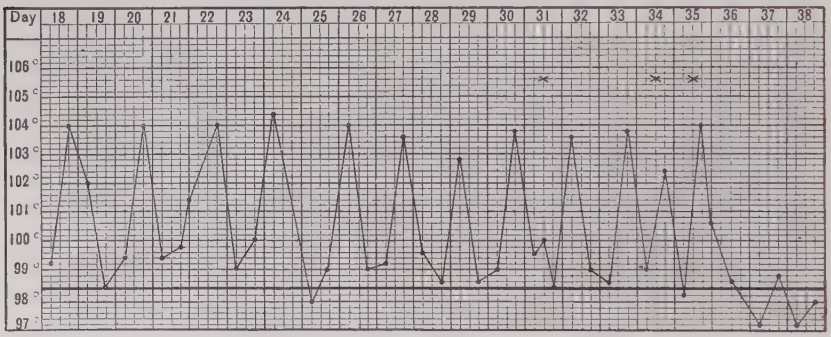


FIG. 97.—CEREBROSPINAL MENINGITIS. Late injection of the serum, prompt effect; complete recovery. Boy, 11 years, St. Vincent's Hospital, New York. Early symptoms obscure, and on account of swelling and pain in joints diagnosis of rheumatism made; cerebral symptoms not marked. First lumbar puncture made on 31st day and meningococcus found. Serum injected on the 34th and 35th days. Rapid fall in the temperature followed by cessation of all symptoms and complete recovery.

An immediate effect of the injection is seen in the cerebrospinal fluid. There is a marked reduction in the percentage of polymorphonuclear cells. The number of meningococci is greatly reduced and their vitality lessened. After the first injection they stain with difficulty, and after a second injection it is generally impossible to grow them, although they are usually present in small numbers (Fig. 96). The effect on the symptoms is often striking. There is a marked reduction in the temperature, which may amount to three or four degrees in twenty-four hours, and it may not rise again (Fig. 97). The stupor and delirium often diminish rapidly, and soon disappear. Improvement is also seen in the patient's general condition, pulse, and respiration. The last symptoms to be affected are usually the rigidity of the neck and extremities.

Intraspinal injections are not wholly devoid of danger. A moderate degree of shock following the procedure is quite common. The child's head should be lowered and he should be closely watched for half an hour

or more. In rare instances more serious symptoms are seen, usually in the nature of an acute failure of respiration. Alarming symptoms generally come on quite abruptly with little warning, and unless promptly recognized and energetically treated death may follow. A number of theories have been advanced in explanation of these phenomena, but it seems clear that they are due to the changes produced in the intracranial pressure. If the symptoms develop while serum is being injected, the funnel should be lowered and some of the fluid siphoned out of the canal. Atropin should be given hypodermically and artificial respiration employed energetically. We have seen but a single fatal result, but in several instances it was necessary to use artificial respiration for fifteen or twenty minutes before normal respiration was established. It is evident that the greatest care should be used in injecting serum and that the possibility of the development of serious symptoms should always be kept in mind. A close observation of the blood pressure during the injection has been advocated by Sophian; its fall furnishes a warning of the development of serious symptoms. Our own experience leads us to the belief that it is of some value, but that very careful watching of the child's pulse and respiration answers quite as well.

The results of this treatment show a much larger percentage of recoveries than has been obtained by any other method.[1] Of 1,500 cases of all types, in patients of all ages treated by this serum, the general mortality was about 25 per cent. The figures represent results obtained in many epidemics in all parts of the world. The statistics from this country are not so favorable as those from abroad with the same serum, for the reason that in the results here are included reports from many physicians who, without experience in the use of the serum, treated but one or two cases. The foreign statistics, however, are in larger groups, and the cases for the most part were under the care of men who had had experience with the serum. In the epidemic in France the mortality of the cases not treated by serum was about 70 per cent, while in those receiving serum it was but 15 per cent. This indicates what may be expected with serum treatment under favorable conditions. One of the most striking evidences of the value of this treatment is the results obtained in infants under one year. Without serum these cases have almost invariably terminated fatally; with serum over 50 per cent of them have recovered.

The results are much modified by the time of injection as shown by the following table:

[1] For details, see articles by Flexner and his associates in the Journal of Experimental Medicine, from September, 1908, to 1915. Reliable serum can be obtained from the New York Health Department.

Time of Injection	Flexner. (All sources, chiefly U. S.)	Netter. (France.)	Dopter. (France.)
1st to 3d day	14.9%	7.14%	8.2%
4th to 7th day	22.0%	11.1 %	14.4%
After the 7th day	36.4%	23.5 %	24.1%

In Netter's series Flexner's serum was used; Dopter used the serum prepared at the Pasteur Institute.

The effect on the course and duration of the disease is no less marked than that upon the mortality. The duration of acute symptoms is very much shortened, and in about one-fourth of the cases the disease terminated by crisis (Fig. 97). This is more often seen in cases injected early, although it is observed in some injected as late as the fourth week. The infrequency of complications and sequelae is also noteworthy. Not only do patients recover, but they recover quickly, and in most instances completely. The absence of complications and sequelae is, no doubt, to be explained partly by the effect of the serum in shortening the disease.

Relapses occur in a small proportion of the cases. They are due to the fact that the organisms have not been entirely destroyed by the serum. They are usually indicated by a rise in temperature, an increase in the leucocytosis, and an aggravation of the nervous symptoms. They are to be treated like a primary attack, daily injections being repeated so long as organisms and symptoms persist.

Very little improvement is to be expected in patients who have passed the febrile stage and who are suffering chiefly from the effects of distention of the ventricles due to a chronic basilar lesion. The most unpromising early cases are those of the fulminating type which have usually advanced so far before the serum is given that recovery is impossible. Unpromising also are cases in which a very thick purulent fluid is present which can hardly be withdrawn through the needle. The amount which can be removed is usually very small. The diffusion of the serum in the canal is difficult. In such cases Robb (Belfast), before injecting the serum, has used with success irrigation of the spinal canal with a warm sterile salt solution. In some cases, particularly in infants, when the withdrawal of fluid by lumbar puncture has been impossible owing to adhesions or other causes, fluid may be removed by puncturing the ventricles of the brain through the fontanel. The serum is then injected into the same cavity. The procedure is not difficult, and, if carefully done, attended by little risk. We have used it in several cases. The effect of the serum seemed quite as marked as when it was introduced in the usual manner.

In any case suspected to be cerebrospinal meningitis lumbar puncture should be made as early as possible. If the fluid obtained is purulent or only slightly turbid, the serum should be injected at once. If the fluid is clear, the disease is probably not cerebrospinal meningitis, and one may wait for a bacteriological report. Meningitis due to the pneumococcus, the bacillus of influenza, or to pyogenic organisms, may also give a purulent fluid, but no harm would result from using the serum in such a case, although no benefit should be expected.

The injection of various chemical agents (protargol, lysol, etc.) has from time to time been advocated; but the experimental work of Flexner and Amoss has shown that such substances are absolutely without value and may even diminish the chances of natural recovery.

Lumbar puncture *per se* has some slight therapeutic value. It relieves pressure and by reducing the number of microörganisms may have a slight effect upon the inflammatory process, especially when used early; but in most cases this is only temporary. An ice-cap should be applied to the head, and at times an ice-bag along the spine. The bowels should be kept freely open. Treatment otherwise is directed toward the symptoms of the disease. Severe pain requires morphin or codein sometimes in quite large doses. For other nervous symptoms—delirium, sleeplessness, etc.—the bromids and chloral, sulfonal, or trional may be given, or warm sponge or tub baths. Stimulants are indicated by a weak, rapid, and irregular pulse. Caffein and digitalis or strophanthus should be used, but not strychnin.

The nutrition of the patient is important. Feeding is often difficult, and gavage may be advantageously employed. Bed-sores should be prevented by cleanliness, frequently changing the patient's position, etc. Retention of urine may require the use of the catheter.

For the residual paralysis, massage, warm baths, and friction should be employed, but electricity only when all symptoms of central irritation have subsided. The prolonged use of iodid of potassium, especially in combination with mercury, seems to have some value.

ACUTE MENINGITIS DUE TO OTHER CAUSES

Besides the main varieties of acute meningitis, viz., that due to the meningococcus and that due to the tubercle bacillus, there are other forms differing in etiology, but closely related clinically, and therefore they may be advantageously considered together. It is only since the general adoption of lumbar puncture as a means of diagnosis that these forms of meningitis have been clinically differentiated. Formerly they were grouped under the somewhat indefinite heading of "simple menin-

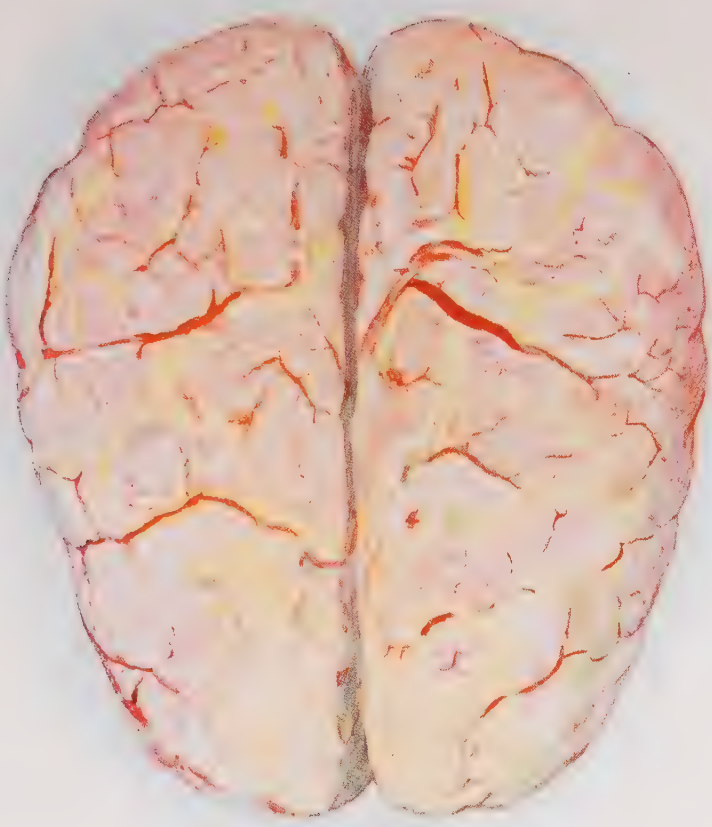
gitis." Three of these varieties, those due to the pneumococcus, the influenza bacillus, and pyogenic organisms, are sufficiently important to require separate description. Cases of meningitis due to the typhoid bacillus, the gonococcus, and the colon bacillus, have all been reported in children, but are so rare as only to deserve mention.

Pneumococcus Meningitis.—This is the most important variety included in this group and the one most frequently met with in young children. In our hospital patients about ten per cent of the cases of acute meningitis were of this form. Nearly all had pulmonary symptoms of greater or less severity, usually a definite pneumonia with consolidation; several had also empyema. Less frequently, pneumococcus pericarditis and peritonitis have been present. Occasionally pneumococcus meningitis is seen when there are no definite pulmonary symptoms or signs and when it is apparently a primary inflammation. However, in most cases pneumococcus meningitis is one of the results of a generalized pneumococcus infection. In every one of our cases of pneumococcus meningitis in which cultures of the heart's blood were made at autopsy, this organism was present. It was usually found in blood cultures made during life. This form of meningitis occurs in infants more frequently than in older children, and, in our experience, usually in very young infants: over half of the cases seen were in patients under six months old. While the disease usually develops at the height of an attack of pneumonia, it may precede the pulmonary symptoms and it may develop during convalescence. We once saw it as late as the fourth week.

Lesions.—In a general way the anatomical changes resemble those described in cerebrospinal meningitis, with the exception that the marked changes in the brain substance which are usually dependent upon the long course of that disease are wanting. As a rule, also, the lesions are limited to the brain. If the cord is involved, it is only to a slight degree.

Acute meningitis due to the pneumococcus is characterized by a more abundant exudation of fibrin and pus than is seen in any other variety of meningitis. The lesion may affect the entire brain, but it is especially marked at the convexity and over the anterior lobes. Sometimes it is limited to these regions, the meninges of the base escaping. The exudate may be so abundant as almost to conceal the convolutions. (See Plate XI.) There is usually less distention of the ventricles than in cerebrospinal meningitis.

In cases apparently primary, or when meningitis occurs very early in the course of a general pneumococcus infection, the symptoms are usually indistinguishable from those of ordinary cases of cerebrospinal meningitis. It is generally not until lumbar puncture is made that the variety of meningitis is suspected. When meningitis occurs as a secondary in-



ACUTE PNEUMOCOCCUS MENINGITIS, COMPLICATING PLEUROPNEUMONIA

Child twenty months old; on twenty-third day of a protracted attack of pneumonia, vomited six times, and the temperature, which had been nearly normal for four days, rose to 103° F. On the following day general convulsions, which were repeated frequently during the next few days; temperature, 101° to 104° F.; death in convulsions on twenty-eighth day.

Autopsy.—Pleuropneumonia of left side; lung resolving. Anterior portion of brain enveloped in lymph and pus, more marked at the convexity, but present also over the base.

flammation it is often latent, and not infrequently is found at autopsy when not suspected during life. Usually, however, the meningeal complication is indicated by the abrupt development, in the course of an attack of pneumonia, of vomiting or convulsions, followed by active delirium or stupor. Because the lesion is principally, sometimes only, at the convexity, many of the symptoms belonging to meningitis with basal lesions are absent. There is rarely cervical opisthotonus; the fontanel may not be bulging; pulse and respiration may not be disturbed, in fact, there are no cranial-nerve symptoms and the symptoms due to spinal involvement—hyperesthesia, rigidity, Kernig's sign, etc.—are usually wanting.

The course of pneumococcus meningitis is generally short and acute, death taking place within three or four days from the first symptoms. We have several times seen a prolonged type of the disease lasting many weeks; one case ended fatally near the end of the third month; another patient recovered from the acute symptoms, but remained partially paralyzed and mentally defective.

The diagnosis of pneumococcus meningitis can positively be made only by lumbar puncture. The cerebrospinal fluid in gross appearance does not differ from that seen in cases due to the meningococcus. The cells present are chiefly polymorphonuclear. Pneumococci are very abundant and are easily found in smears and grown readily in cultures. The existence of pneumococcus meningitis is not always shown by lumbar puncture. We have met with one case in which repeated punctures gave negative results, and yet the autopsy showed meningitis to be present, but only the convexity was affected. The organisms were readily found in the meningeal exudate.

Influenza Meningitis.—This form of meningitis in many respects resembles the form just described. According to Wollstein,[1] there had been recorded, up to 1911, 49 cases of pure, and 9 cases of mixed, influenza meningitis. Of these, 28 were in infants under one year old. Since then many additional cases have been reported. The disease is certainly not very rare. Of the cases which have come under our own observation, all but one have been in infants and all have ended fatally. In our experience, influenza meningitis has been secondary to other influenza infections, usually those of the rhinopharynx or bronchi. The organisms were found by culture from the secretions of these parts during life. One patient, an infant of eight months, was admitted to the hospital with an acute abscess of the elbow joint. Two days later symptoms of meningitis developed, and death occurred in three days. The autopsy showed an extensive purulent meningitis. Pure cultures of the

[1] American Journal of Diseases of Children, January, 1911.

influenza bacillus were obtained from the pus of the elbow, the fluid drawn by lumbar puncture, the meningeal exudate, and the heart's blood. The lungs showed influenza bacilli and streptococci.

The lesions of influenza meningitis, in the few cases in which autopsies have been made, have differed in no essential particular from those described in the pneumococcus variety. In the cases coming under our observation in which examinations were made, the influenza bacillus was obtained from the heart's blood as well as from the cerebrospinal fluid.

Clinically, influenza meningitis usually runs a short, very acute course. There are no features by which it can be distinguished from the pneumococcus or meningococcus form, except the findings of lumbar puncture. In gross appearance the fluid does not differ from that seen in the other forms. There is usually marked turbidity; the cells are abundant and of the polymorphonuclear variety. The organisms are generally not numerous in the smears, in marked contrast to the other forms of meningitis. They are readily grown upon blood agar, but not upon ordinary media. If, therefore, from a turbid cerebrospinal fluid no growth occurs, influenza meningitis should be suspected.

Meningitis Due to Pyogenic Organisms—Septic Meningitis.—Meningeal inflammations set up by the streptococcus or staphylococcus are not very common in young children. They are almost always secondary. In the newly born this form of meningitis is seen in general pyemia, usually from umbilical infection; it also follows infection of a spina bifida. In older children it follows injuries to the head, erysipelas of the scalp, operations upon the brain, and otitis media with mastoiditis or sinus thrombosis. Such a complication of otitis in infancy is, however, extremely rare. The lesions consist in a widespread general inflammation of the pia with an abundant exudate of pus, but with less fibrin than in the two varieties previously described.

The symptoms of septic meningitis are not distinctive. The course is usually a rapidly progressive one, and the termination almost invariably in death. The fluid drawn by lumbar puncture in most cases is markedly turbid, and shows great numbers of pus cells. The organisms are present in large numbers and are readily recognized both in smears and by cultures upon ordinary media.

Diagnosis.—The differential diagnosis of the different forms of meningitis from each other, and from other diseases with cerebral symptoms, is made with certainty only by means of lumbar puncture, which should be done in all cases of doubt. The appearance of the cerebrospinal fluid is essentially the same whether the inflammation is due to the meningococcus, the pneumococcus, the influenza bacillus, or to the staphylococcus or streptococcus. The symptoms of meningitis in general, de-

scribed in the chapter on Cerebrospinal Meningitis, are present in most of the cases.

Prognosis and Treatment.—The prognosis in all varieties of acute meningitis, except that due to the meningococcus, is very bad; almost every case of meningitis due to other causes is fatal. From what has been said, it would appear that treatment is as yet most unsatisfactory, and is only symptomatic. Wollstein's researches at the Rockefeller Institute, however, indicate that influenza meningitis may occasionally be controlled by serum treatment. A goat serum has been produced which regularly controls the experimental disease in monkeys. Its use in children has thus far been very seldom successful, since there is usually a general influenza septicemia and since the disease is so rapid in its course that an early diagnosis is rarely made.

TUBERCULOUS MENINGITIS

(*Acute Hydrocephalus; Basilar Meningitis*)

Tuberculous meningitis is a tuberculous inflammation of the pia mater of the brain, sometimes involving also that of the cord. It is by far the most frequent form of acute meningitis seen in young children. In our hospital experience, apart from epidemics of cerebrospinal meningitis, seventy per cent of the cases of acute meningitis have been tuberculous. It is more uniformly fatal than any other disease of early life. It is doubtful if it ever occurs as the only tuberculous lesion of the body. In infancy it is usually associated with general or pulmonary tuberculosis; in older children with tuberculosis of the bones, joints, or lymph nodes. Of our own cases, forty per cent of all deaths from tuberculosis in children have been due to meningitis.

Lesions.—The lesion consists in the production of miliary tubercles, with which are frequently found tuberculous nodules of variable size, and in almost every case there are also the products of ordinary inflammation of the pia mater—fibrin and pus—together with an accumulation of fluid in the lateral ventricles of the brain. Frequently there are tubercles in the pia mater of the upper portion of the cord. When few in number the tubercles are usually only at the base. When numerous they are seen scattered over the convexity. Tubercles are frequently found in the choroid coat of the eye. The amount of fibrin and pus in the exudate is usually small, and is much less than is seen in other forms of acute meningitis. The inflammatory products are most abundant at the base. In addition to the patches of greenish-yellow fibrin, there are adhesions between the lobes of the brain and thickening of the pia. In cases which

have lasted for several weeks, this thickening may be marked, owing to cell infiltration and the production of new connective tissue. The pia is studded with miliary tubercles, sometimes with small yellow tuberculous nodules; frequently there is arteritis, which is sometimes obliterating.

In the most acute cases the brain substance immediately beneath the pia is intensely congested, slightly softened, and shows under the microscope a superficial encephalitis. The lateral ventricles are usually distended with clear serum, sometimes with serum containing flocculi of fibrin or pus; the amount present varies from one to four ounces in each ventricle, being always greater in the subacute cases. The walls of the ventricles may be softened. The distention of the ventricles leads to flattening of the convolutions from pressure against the skull, to bulging of the fontanel, and sometimes to separation of the sutures.

Tuberculous nodules varying in size from a small pea to a walnut are frequently seen associated with meningitis in older children, but not often in infants. These nodules may be connected with the meninges, or they may be situated within the brain substance, usually in the cerebellum. The larger ones are classed as brain tumors. Inflammatory products are rarely found in the spinal canal.

Although it is not infrequent to see meningitis without symptoms of tuberculosis elsewhere, we have never failed at autopsy to find other tuberculous lesions in the body. In our experience the following are those most often met with, given in the order of frequency: (1) In infants, associated with general or pulmonary tuberculosis; (2) in children from three to twelve years of age, with tuberculosis of the vertebrae, hip, knee, or ankle; (3) at any age, with tuberculosis involving only the tracheal, bronchial, or mesenteric lymph nodes; (4) much less frequently with the pulmonary tuberculosis of older children.

Etiology.—Tuberculous meningitis is produced only by the transportation of the tubercle bacilli to the brain. They may find their way by the blood-vessels or by the lymphatics.

The following table shows the age at which the disease was observed in 410 cases of which we have notes:

Under one year	162
One to two years	149
Two to five years	76
Five to nine years	17
Nine to sixteen years	6
Total	410

In this series three cases were in children three months old or younger. Tuberculous meningitis in our experience occurs much more often in the winter and spring months than at other seasons (Fig. 98).

The most plausible explanation of this seems to be that these patients, infected some time previously, carry a latent focus of tuberculosis somewhere in the respiratory tract, usually in the bronchial glands. Under the influence of acute respiratory infections of the cold season, the latent tuberculous disease becomes active, and a rapidly spreading tuberculous process results. In infants and young children it rarely happens that pulmonary lesions are absent; but these patients are especially predisposed to early meningeal infection, and this often occurs before symptoms of tuberculosis elsewhere have manifested themselves. At the time of invasion, therefore, most of these children are apparently in the best of health. In older children there may have been previous evidence of tuber-

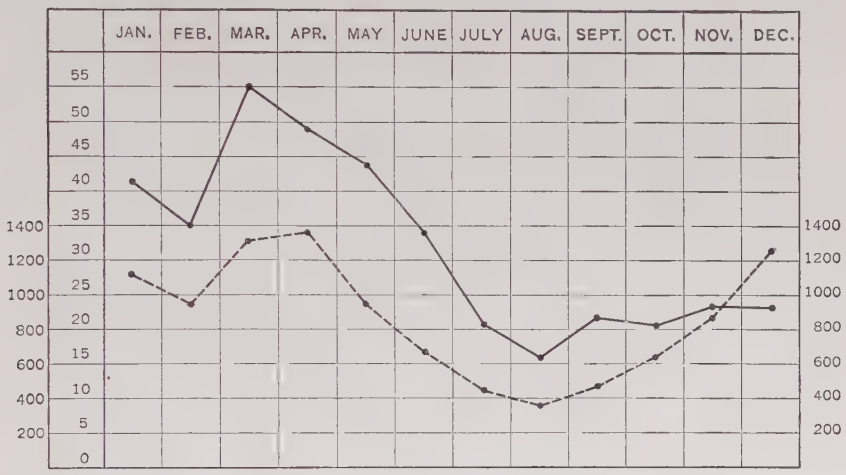


Fig. 98.—Seasonal Occurrence of 400 Cases of Tuberculous Meningitis. Lower Curve, Deaths from Pneumonia New York City, one year.

culosis in lungs, bones, or lymph nodes. The modes of acquiring tuberculosis are discussed in the general chapter on that disease. It is sufficient to say here that it is usually from some member of the family or household. This may be not only a person who is in the active stage of pulmonary tuberculosis, but one who is supposed to have been cured or one in whom the disease has not yet been suspected. Exposure may antedate symptoms by several weeks or months. Striking evidence in favor of the human origin of tuberculous meningitis is obtained from a study of the type of tubercle bacillus present in cases of meningitis. In thirty-two cases in our series, this was worked out by Park and Krumwiede in the Research Laboratory of the New York Health Department. In thirty the bacillus was of the human type; in one it was of the bovine type, and in one both types were present.

Symptoms.—In about two-thirds of the cases the onset is gradual;

but in a considerable number of those classed as abrupt, careful inquiry will elicit a history of previous indisposition. The most frequent early nervous symptoms are, disinclination to play, drowsiness, or sometimes constant fretfulness or irritability. Often there is a complete change in disposition. In a case under our observation this was most striking; a little girl previously devoted to her mother, could not endure her presence in the room. Sleep is restless and disturbed; there may be grinding of the teeth. Older children often complain of headache. At all ages, but particularly in infancy, early digestive symptoms are prominent. There are seen frequent attacks of vomiting without apparent cause; the bowels are generally constipated and the appetite is almost entirely lost. Usually there is also a slight but continuous elevation of temperature. Indefinite symptoms may last for four or five days, or they may be spread over two or three weeks without perhaps being sufficiently severe to attract much notice. Finally, unmistakable evidence of brain disease develops. The early disturbances are often ascribed to dentition, or to indigestion.

In most cases the first pronounced cerebral symptom is persistent and increasing drowsiness; exceptionally it is an attack of general convulsions, followed in a few hours by stupor. Often a period of irritative symptoms is present, lasting several days. There is headache, usually located in the frontal region, and occasionally photophobia; sometimes pain is indicated by the child's suddenly screaming out at night, which may be repeated many times without waking; sometimes during the greater part of the time for two or three days these frequent screaming attacks may be repeated. The skin is somewhat hyperesthetic; the reflexes are apt to be exaggerated; the muscles of the neck may be rigid and the head is drawn back, or there may be rigidity of the extremities. The pupils are normal or contracted; there may be nystagmus. The child is fretful, wishes to be left alone, and cries if disturbed. In some cases these symptoms are so marked as strongly to suggest cerebrospinal meningitis. They may alternate with periods of marked apathy and dulness. During this stage there is occasional vomiting, and the bowels are obstinately constipated. The pulse is usually somewhat accelerated, but may be slow and occasionally it is irregular. The respiration is of normal frequency, but a careful observation during sleep or perfect quiet will often show a distinct irregularity which is very significant. The temperature is usually elevated, ranging from 99° to 100.5° F. When a high temperature is seen, it is usually due to tuberculosis elsewhere than in the brain.

As the disease advances, the irritative symptoms subside, and the stupor becomes deeper and more continuous. If undisturbed, the child may sleep a great part of the time, but can be roused, and then appears

quite rational. Finally the stupor becomes so profound that the child can not be roused at all. Active delirium is rare. The pupils respond slowly to light or not at all; they may be unequal; occasionally there is seen strabismus, ptosis, or paralysis of the face. More often there is hemiplegia, or paralysis of one arm or leg. Such paralyses are often transient, disappearing after a day or two. Automatic movements of the extremities, particularly of the arms, are frequent. Muscular twitchings may be noticed. Opisthotonus is marked and well-nigh constant. In infants the fontanel is tense and bulging. In older children especially, the abdomen is retracted, giving the typical "boat-belly." After drawing the finger-nail along the skin of the abdomen, there appears a distinct red streak, which remains for several minutes. This is the *tâche cérébrale*, and it is almost always present. Other vasomotor disturbances may be seen. The reflexes are variable; in the early part of the disease they are usually increased, later they are diminished or abolished. The pulse now becomes slow and irregular, often intermittent. The respiration is almost always irregular; a very characteristic type consists in the movements becoming deeper and deeper until there is a sigh; followed by a complete arrest of respiration for several seconds. The phenomenon is then repeated. The accompanying tracing illustrates the type (Fig. 99). An examination with the ophthalmoscope usually shows the presence of choked discs, and in a very considerable number of the cases, if they are closely studied, tubercles may be seen in the choroid. Their presence is of much diagnostic importance. The blood picture in this disease is fairly characteristic. From 230 observations made in the Babies' Hospital, it was shown that early in the attack the total leucocytes are only slightly increased, they may be even below the normal. As the disease progresses they increase in number, the average during the last week of the disease being 29,600. The proportion of polymorphonuclears also shows a marked increase. The early range was 60 to 65 per cent; during the last week it was from 70 to 85 per cent.

FIG. 99.—TRACING OF RESPIRATION IN TUBERCULOUS MENINGITIS.

The progress of the disease is subject to great variations, especially in children over two years old. The advance of symptoms is slower and is interrupted by periods of remission which may continue two or three days. After being in quite deep stupor, a child may recover consciousness, and even sit up and play with toys, leading to the view that an error in diagnosis has been made. But this respite is only temporary; soon the child passes again into coma.

From this time the duration of the disease is from three to ten days. The child can not be roused at all. The pupils are widely dilated, and

do not respond to light. There is general muscular relaxation. There may be retention of the urine. Deglutition is difficult, often impossible. The respiration is more rapid, but still irregular. The pulse becomes very rapid and feeble, often 160 to 180 a minute. Toward the end the temperature often rises rapidly to 104° F., sometimes to 106° or 107° F. (Fig. 100). Death usually takes place from exhaustion in deep coma, or convulsions develop and continue from twelve to twenty-four hours until death. Sometimes a patient will live for days in a condition of prostration so extreme that death is hourly expected. A rapidly rising temperature or the occurrence of late convulsions usually indicates approaching death. Of fifty-seven cases, fifty died in coma, seven in con-

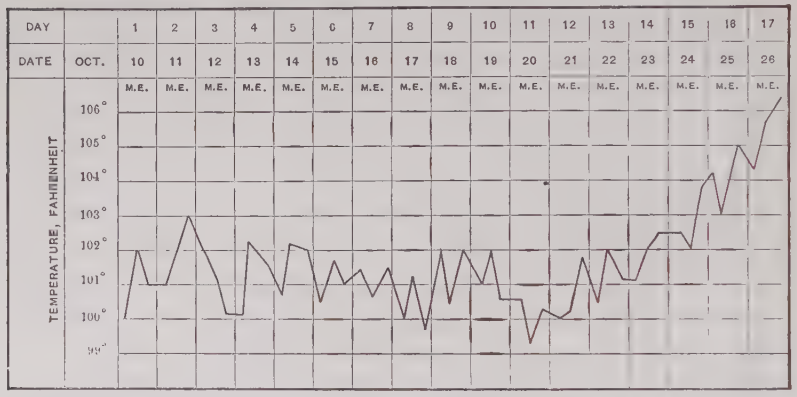


Fig. 100.—Fairly Typical Temperature Curve in Tuberculous Meningitis. Boy, twenty months old; death on seventeenth day.

vulsions. The entire duration of the disease from the beginning of definite nervous symptoms is rarely over three weeks, and in infants it is usually shorter than this.

Diagnosis.—Tuberculous meningitis is often overlooked because the patients do not give outward evidences of tuberculosis. Its frequency should always lead one to suspect it when protracted nervous symptoms are present in infants. There are no diagnostic symptoms in the early stage. The indefinite symptoms that belong to this stage of the disease are frequent in young children suffering from chronic indigestion associated with constipation. Cases of cyclic vomiting may present many of the symptoms of meningitis.

The most diagnostic symptoms of tuberculous meningitis enumerated in the order of their frequency are as follows: persistent drowsiness, obstinate constipation, vomiting without apparent cause, irregular respiration, irregular pulse, convulsions, opisthotonus, and fever which is usually slight. A positive diagnosis is made only by lumbar puncture; by this means this form is distinguished from other forms of acute

meningitis. The fluid drawn by lumbar puncture is usually perfectly clear, but sometimes after standing there is a slight deposit present. In rare cases the fluid may be turbid. As compared with the other forms of acute meningitis the cells are few in number. The usual cell count is from 100 to 250 per c. mm. Nearly all the cells, over 95 per cent in most cases, are mononuclear. Very exceptionally the polymorphonuclear cells are greatly in excess. The presence or absence of sugar has been in our experience of no diagnostic importance.

Tubercle bacilli are almost invariably present in the fluid, although in the early stage they are few in number and often difficult to find. But at the height of the disease by careful examination they can be found microscopically in nearly every case. They were found in 135 of 137 consecutive cases of tuberculous meningitis at the Babies' Hospital. They are more numerous late in the disease.

The technic is important. Fluid should be drawn into several tubes and the last one containing 15 to 20 c.cm. set aside for examination, as the bacilli are much more likely to be found in this. The tube should not be shaken, but should be allowed to stand for twelve hours, preferably in an incubator. A central fibrin coagulum generally forms in the fluid, and in this the bacilli are usually entangled. This should be spread out entire and carefully examined. In other cases the bacilli may be found after centrifuging. In most of the cases the number of bacilli present is not large and a search of half an hour to an hour is necessary; but not infrequently they are so numerous that they are discovered in a few minutes.

The Ross-Jones[1] and Noguchi globulin tests are useful in distinguishing inflammatory from normal cerebrospinal fluids. They are, however, of no value in distinguishing between the different forms of meningitis. A positive reaction is obtained with great uniformity in every variety of acute meningitis.

Bacilli have been found in the sputum, in our experience, in nearly one-half the cases in infants and young children with tuberculous meningitis, although in most of them there was little or no evidence of pulmonary disease.

The v. Pirquet cutaneous test gives reliable information except in moribund cases, in those excessively prostrated or with very poor circula-

---

[1] Lancet, May 8, 1909, p. 113.

A few cubic centimeters of a completely saturated solution of pure ammonium sulphate are placed in a test tube and 1 c.c. of cerebrospinal fluid is gently run on to the surface. A positive reaction is indicated by the formation of a ring at the point of contact of the two fluids. The ring is grayish white and sharp. It should form within three minutes. Indirect illumination should be used for its detection.

tion. A positive reaction was obtained in 161 of 194 cases tested. This test is of much assistance in early diagnosis.

If, then, a child with symptoms distinctly meningeal gives a positive reaction to the tuberculin test the probabilities of tuberculous meningitis are greatly strengthened, even though at the time bacilli may not have been found in the cerebrospinal fluid.

The cerebral symptoms of intestinal and many other acute diseases sometimes closely resemble those of tuberculous meningitis. From all such the diagnosis is made by lumbar puncture. In any case of meningitis in a young child the chances are greatly in favor of the tuberculous form, since it is much more frequent. The diagnosis from cerebrospinal meningitis and acute poliomyelitis is considered under those diseases. Differentiation from the meningeal form of poliomyelitis may be very difficult, owing to the similarity of the spinal fluid in the two diseases.

Prognosis.—Although there have been recorded a few instances of recovery after tubercle bacilli have been found in the fluid obtained by lumbar puncture, such an outcome is not to be expected. We have never seen such a case recover. The reported recoveries in which the diagnosis has rested upon clinical symptoms only, can not be accepted.

Treatment.—From what has been said regarding prognosis, it follows that if the diagnosis is correct the case is practically hopeless, no matter what treatment is employed; but as a positive diagnosis is not always possible, all cases should be treated like other forms of acute meningitis.

CHRONIC BASILAR MENINGITIS IN INFANTS

It was first pointed out in 1898 by Still that this disease is usually due to the diplococcus intracellularis; in other words, that it is a chronic form of cerebrospinal meningitis. Chronic basilar meningitis is most frequently seen after epidemics of cerebrospinal meningitis, but it is occasionally met with at other times as a sequel of a sporadic case. It occurs after an acute attack, when the basilar lesion persists, and becomes chronic. As acute cerebrospinal meningitis in infants is usually fatal if the attack is severe, it follows that the chronic form is seen only after the mild attacks. It is chiefly for this reason that the early symptoms often are not recognized as those of cerebrospinal meningitis. The patient frequently does not come under observation until all acute symptoms have passed away, the persistent opisthotonus being the chief feature of the case.

There is also seen in children, though very rarely, a chronic basilar

meningitis of syphilitic origin. Several such cases have come under our observation.

Lesions.—This process is usually limited to the base of the brain. The pia mater is thickened about the interpeduncular space, also over the medulla, pons, and cerebellum. It may be adherent to the inner surface of the dura. The foramina of Magendie and of Luschka are usually obliterated, and there results a distention of the lateral ventricles with clear serum, sometimes in sufficient amount for the case to be regarded as hydrocephalus. Rarely, pus may be found in the ventricles. There may be a cystic formation at the base of the brain due to the accumulation of fluid in one of the cisterns of the pia. In such circumstances

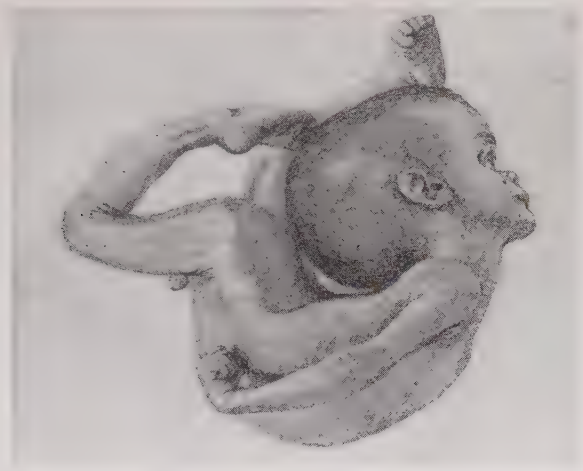


Fig. 101.—Chronic Basilar Meningitis—Extreme Deformity. Ill for five months; followed cerebrospinal meningitis; posture shown in the picture was maintained for the last six weeks; death at ten months. Autopsy showed typical lesions.

the cerebellum is often much compressed by the fluid. The cranial nerves may also be compressed.

Symptoms.—The onset is usually gradual, although in most cases there can be obtained a fairly distinct history of an early active period. The most prominent symptoms are cervical opisthotonus, moderate hydrocephalus, and usually general muscular rigidity. The opisthotonus is often extreme (Fig. 101) and is greater than is seen in any other disease. If placed upon his back the body of the child often touches the table only at the occiput and the sacrum (Fig. 102). The head is usually somewhat enlarged, but never to the degree seen in primary hydrocephalus; the fontanel bulges, and the sutures are separated. These symptoms are due to an accumulation of fluid in the lateral ventricles. The rigidity of the extremities is very great and in most cases constant; the legs and

feet are usually extended, while the forearms are flexed and the hands clenched. All the reflexes are greatly exaggerated. There is rarely coma, but mental dulness alternating with periods of great irritability in which general convulsions may occur. Vision may be impaired or wanting entirely. The fact that in most of the cases optic neuritis is absent is of some value in differentiating this disease from tumor. Nystagmus is often present and attacks of vomiting occur without evident cause. There is no fever except for a few days at a time during acute exacerbations. Fluid obtained by lumbar puncture is often clear but usually contains a slight excess of cells and the globulin reaction is positive. Occasionally turbid fluid may be obtained and there may be found

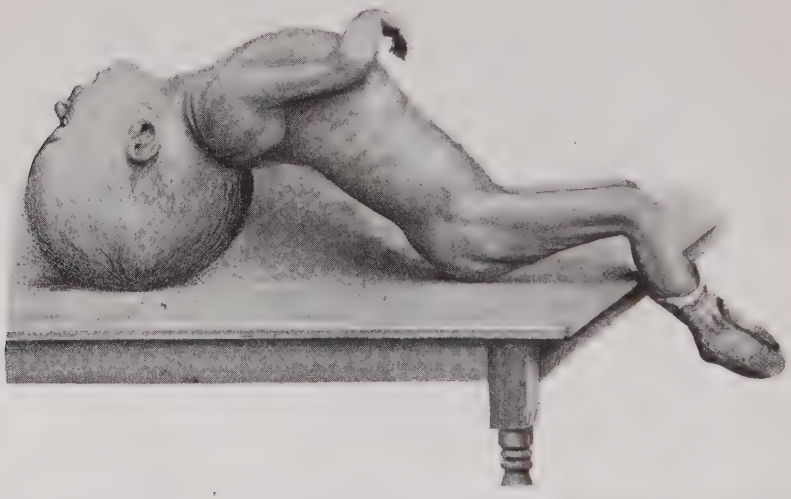


FIG. 102.—CHRONIC BASILAR MENINGITIS. A patient in the Babies' Hospital (diagnosis confirmed by autopsy).

a small number of meningococci, both intra- and extra-cellular. The usual duration of the disease is from two to five months; death may occur from convulsions, or from some intercurrent disease, such as pneumonia, but most frequently from marasmus. The prognosis is very bad except when the cause is syphilis, when recovery may take place.

Diagnosis.—The disease is to be distinguished from tuberculous meningitis, and from the opisthotonus of reflex origin which is occasionally seen in infants suffering from marasmus. It differs from tuberculous meningitis in its more protracted course, in the absence of fever and paralysis, and also in the greater prominence of the opisthotonus and hydrocephalus.

Treatment.—If meningococci are found, anti-meningococcus serum should be used. It will usually destroy the organisms, although it can-

not affect the pathological changes that have taken place as the result of their long activity. If there is any reason to suspect syphilis, salvarsan and the iodid of potassium and mercury should be administered. Operations for the relief of the hydrocephalus have, up to the present time, met with little measure of success.

THROMBOSIS OF THE SINUSES OF THE DURA MATER

This is not of very frequent occurrence. It may depend upon certain general conditions, when it is usually classed as *cachectic* or *marantic thrombosis:* it may be associated with local pathological processes, when it is known as *inflammatory* or *septic thrombosis.*

Cachectic Thrombosis.—This is seen in infants and young children, but is very rare after the age of five years. It occurs in the course of various diseases, the most frequent being pneumonia, pertussis, diphtheria, nephritis, tuberculosis, and the acute intestinal diseases. In connection with the last-mentioned group, altogether too much has been made of it, as it is really rare, and in only a very few cases does it explain the cerebral symptoms present. The actual cause of the thrombosis is the altered condition of the blood and the feeble circulation, as the walls of the sinuses are normal.

The most frequent seat of cachectic thrombosis is the superior longitudinal sinus. At autopsy one must be careful not to confound the soft, partly decolorized, non-adherent thrombi of post-mortem origin, with those of ante-mortem formation. The latter are firm, and when of long standing may be very hard and even show a laminated structure. They usually fill the sinus completely, and are adherent. The thrombus extends from the sinuses to the veins emptying into it, which stand out like dark worms upon the surface of the brain. The brain itself may be deeply congested, or it may be covered with a diffuse hemorrhage, but more frequently the brain and the membranes are simply edematous.

The symptoms of cachectic thrombosis are few and uncertain, and in a large number of cases the disease is latent. Very rarely is a positive diagnosis possible during life. When the thrombosis occurs just before death, its symptoms are so mingled with those of the original disease that they can not be separated. In some cases there may be localized or general convulsions, or paralysis, loss of consciousness, and strabismus.

The prognosis is bad, cases generally proving fatal in the course of a few days. The diagnosis is so uncertain and obscure that the treatment must be symptomatic, and directed toward the general rather than the local condition.

Inflammatory Thrombosis—Septic Thrombosis—Sinus-Phlebitis.—This condition is most frequently seen in children in connection with acute meningitis. It may exist either with the simple or the tuberculous variety. It also follows otitis—especially old and neglected cases—usually with necrosis of the petrous bone, but sometimes without it. It is much less frequently associated with disease of the ear in children than in adults. It may arise from traumatism, necrosis of the cranial bones, or from septic processes involving any of the cavities or any of the structures adjacent to the brain, such as the scalp, orbit, nasal fossa, mouth, or pharynx. Infection from the mouth or pharynx is most frequent in children in connection with scarlet fever or diphtheria; while usually secondary to otitis it may occur without it, the infection being carried by the blood-vessels. Infection from the nose may have its origin in ulceration from syphilis or tuberculosis. In the orbit, the source may be malignant disease.

The seat of the thrombosis will depend upon the original disease. If this affects the cranial bones or the scalp, it will be the longitudinal sinus; if the ear, the lateral sinus; if the base of the skull, the orbit, the mouth, the jaw, or the nose is affected, it will be the cavernous sinus. When thrombosis occurs with meningitis the lesions are much the same as in the cachectic form, with the exception that there are sometimes slight changes in the walls of the sinuses. If the patient has suffered from a local septic process, there may be puriform softening of the clot, and general pyemia, with the development of secondary abscesses in the brain, in the lungs, and in other organs. With such cases there may be associated a general or localized meningitis.

Symptoms.—The symptoms of septic thrombosis are more definite than those of the cachectic form. When occurring in the course of meningitis, it usually adds no new symptoms to those of the original disease. In the pyemic form the symptoms are more characteristic, particularly when associated with otitis. There are recurring chills with very high and widely fluctuating temperature. There is headache, and often localized tenderness of the scalp; the other symptoms which are present are usually the same as those of meningitis. If metastasis occurs, there may be evidences of abscesses in the brain or in other organs, and sometimes there are signs of suppuration in the jugular vein. A polymorphonuclear leucocytosis is usually present, and blood cultures in most cases show the presence of pyogenic organisms.

The local symptoms of the thrombosis differ somewhat according to the sinus affected: if its seat is the superior longitudinal sinus, there may be cyanosis of the face, dilatation of the temporal and frontal veins, and sometimes epistaxis; if the lateral sinus is involved, the process may extend to the jugular vein, which may be felt in the neck as a hard

cord, and there may be dilatation of the veins of the mastoid region, and even localized edema; when the cavernous sinus is affected, there may be protrusion of the eyeball of the affected side, edema of the lid, and with the ophthalmoscope the retinal veins appear enlarged and tortuous, sometimes being the seat of thrombosis. The process may affect either one or both sides. The course of septic thrombosis is rather irregular, varying from a few days to three weeks. In fatal cases death takes place from meningitis, cerebral abscess, or pyemia. The prognosis is very grave unless the disease is so situated that it is accessible to surgical operation.

Treatment.—The only successful treatment is surgical. Operation is easiest in thrombosis of the lateral sinus, being much more difficult if involving the superior longitudinal sinus. So many cases are now on record of successful operation upon septic thrombosis of the lateral sinus that it should always be urged when the diagnosis is clear.

CEREBRAL ABSCESS

Cerebral abscess is quite rare in children, decidedly more so than is cerebral tumor. In Gowers' collection of 223 cases, only twenty-four were under ten years of age. In infants, abscess is one of the least frequent diseases of the brain, and up to five years it is exceedingly rare.

Etiology.—By far the most frequent cause in children is otitis. This is the origin of the great majority of the cases. Abscess rarely complicates acute otitis, but is seen with the chronic form. Exactly how otitis causes cerebral abscess it is not always easy to determine. Usually there is caries of the petrous bone, but there may be none. The infection may extend through the small veins traversing this bone, or along the lateral sinuses to the cerebellum. Abscess is often attributed to the retention of pus in the ear, but it may occur when the discharge is free.

We have seen in a young infant abscess follow nasal infection, the process apparently extending through the cribriform plate of the ethmoid.

Traumatism is the second important etiological factor. Abscess may be associated with fracture of the skull, or follow simple concussion. The abscess is generally in the neighborhood of the injury, but occasionally is produced by *contre coup.* Abscess may be the result of infectious emboli, associated with general pyemia, though this is rare in early life; and finally it may occur without any assignable cause. The organisms usually present are streptococci, staphylococci, or pneumococci.

Lesions.—The most frequent seat of the abscess is, first, the tem-

porosphenoidal lobe; secondly, the cerebellum; thirdly, the frontal lobes. Other locations are very rare. Abscesses are usually single. In size they vary from that of a small cherry to an orange. We have seen a case in an infant in which one whole hemisphere was replaced by several large abscesses with thick walls, only a thin layer of cortex covering them. No cause for them could be found and the pus was sterile. The contents are usually thick greenish-yellow pus, which may be very fetid. When abscesses have lasted for some time they are usually surrounded by a dense pyogenic membrane, and may become encysted. The pathological process may be slow, and often is apparently stationary for a long period. Abscesses may rupture into the ventricles, less frequently upon the surface of the brain, causing meningitis, or the pus may even escape externally through the auditory meatus.

Symptoms.—These are general and local. The general symptoms are much the more important for diagnosis, and often are the only ones present. The local symptoms are those of a tumor. The clinical history of a case of abscess of the brain may be divided into three stages: First, the period of onset, or early acute inflammatory symptoms, fever, etc., which attend the formation of pus. Secondly, the latent period, or period of remission, in which very few symptoms are present; in many acute cases this stage is wanting altogether; in the chronic cases it may last for months, or even years. Thirdly, the final period, with recurrence of active cerebral symptoms, followed by death in a few days.

The onset may be accompanied by symptoms so slight as almost to escape notice. In most cases, however, headache and fever are present. The headache is usually severe, and often localized upon the affected side; in cerebellar abscess it may be occipital. The fever is moderate in intensity, and continuous. In addition there may be vertigo, vomiting, general convulsions, and cessation of the aural discharge, if one has been present. The duration of this stage is variable; it may be only a few days, or several weeks. It is shorter in traumatic cases, and in those which are due to pyemia.

The latent stage, or period of remission of symptoms may be quite short—only a few days' duration—and it is often absent. During this period the temperature may fall quite to the normal, and the headache disappear, or be only occasional and slight. However, if any focal symptoms have been present they remain unchanged.

The symptoms of the terminal stage are due to a rapid extension of the inflammatory process, with edema and softening about the abscess, sometimes to rupture into the ventricle, and sometimes to meningitis. The fever now returns, and may be high. There is headache, often very intense and continuous; there may be delirium and convulsions, and the gradual development of coma. In addition there may be vomiting,

paralysis, opisthotonus, retracted abdomen, and the other symptoms of meningitis. Occasionally all the earlier symptoms may be latent, and the terminal symptoms may be the only ones present. In infants, the fontanel is usually large and bulging; convulsions are rather more frequent than in older children.

The local symptoms of abscess are rather indefinite, owing to its usual situation. Abscesses of considerable size may exist in the temporosphenoidal lobe, in the central part of the frontal lobe, or in the cerebellum, without any definite local symptoms. If the abscess is near the motor area, there are the usual symptoms of disease in this location: spasm, or paralysis of the face, arm, or leg. A cortical or subcortical abscess is likely to cause convulsions. Cerebellar abscess may give rise to occipital headache, frequent vomiting, and when the abscess is large enough to press upon the middle lobe, there may be incoördination of the muscles of the extremities. Optic neuritis may be present, but other symptoms relating to the cranial nerves are rare. Localized tenderness over the scalp, when persistent, is a symptom of importance, and may serve to locate the abscess, if it is superficial.

Diagnosis.—Of the general symptoms, the most important for diagnosis are fever, headache, delirum, and terminal coma. These become particularly significant when following otitis or traumatism. The differential diagnosis of abscess is to be made principally from tumor and meningitis, and from these conditions more by the history and general course of the disease than by any special symptoms. The diagnosis of abscess from tumor is considered in connection with the latter disease. It is more difficult to distinguish between meningitis and abscess, since the two processes are often associated. With meningitis convulsions are more common, but they are rarely localized; rigidity and the inflammatory symptoms are more intense; the course is usually more rapid and more regular, being rarely interrupted, as is the course of abscess. Leucocytosis is more constant and usually more marked in meningitis. Lumbar puncture gives negative results in uncomplicated abscess while it gives positive definite information in meningitis.

Prognosis.—The prognosis in cerebral abscess is always grave, unless accessible to surgical operation. The progress may be slow, or rapid, but it is inevitably from bad to worse, and sooner or later the disease, if not interfered with, proves fatal.

Treatment.—The medical treatment of abscess in its active stage is that of any acute intracranial inflammation—ice to the head, absolute quiet, free catharsis, and full doses of the bromids or morphin, if pain is intense. The absolutely hopeless condition of these cases when left to themselves, and the recent brilliant results from surgical operations, should lead the physician to urge operation in every case.

CEREBRAL TUMOR

Tumor of the brain is not very infrequent, and may be seen even in infancy. From this time up to puberty there is no period of special susceptibility. In 269 of the cases in Starr's collection, in which the nature of the tumor was stated, the following were the varieties:

Tubercle	152 cases
Glioma	37 "
Sarcoma	34 "
Gliosarcoma	5 "
Cyst	30 "
Carcinoma	10 "
Gumma	1 "
	269 cases

Tuberculous tumors are more often multiple than are other varieties. Their most frequent seat is the cerebellum; next to this the pons and crura cerebri. They are occasionally cortical or central. Glioma is most often found in the cerebellum or in the pons, and next in the cortex; it is rarely central. Sarcoma is most frequently in the cerebellum; next to this, in the order of frequency, in the pons, the basal ganglia, and the cortex. Cystic tumors are either central or cerebellar. Taking the cases as a whole, the most frequent seat of tumors in children is: first, the cerebellum; second, the pons; third, the centrum ovale. They rarely spring from the cortex.

Tuberculous tumors are occasionally seen in infancy, but they occur most frequently between the ages of four and twelve years. They are always secondary to tuberculosis elsewhere, usually of the lungs and of the bronchial lymph nodes. They most frequently start from the membranes, rarely being centrally situated, and extend inward, infiltrating the superficial portion of the cerebellum or cerebrum. In more than half of the cases they are multiple. There is almost invariably localized meningitis at the site of the tumor; there may be adhesions between the dura and pia mater, and the disease may extend to the cranial bones. In size these tumors vary from a small pea to a child's fist. They may be softened and broken down at the center, or cheesy throughout. They are the result of a localized tuberculous inflammation, which does not differ essentially from that seen in other parts of the body. They rarely undergo calcification.

Glioma is not infrequent in infancy. It repeats the structure of the neuroglia, being composed of connective tissue and branching cells. It is an infiltrating tumor whose limits are difficult to determine even under the microscope.

Sarcomata may be of almost any variety. They grow much more

rapidly than gliomata. The two varieties are very occasionally combined in the same tumor—gliosarcoma.

Cystic tumors may be the result of porencephalic softening or of encapsulated hemorrhages in early life. Gliomata and sometimes sarcomata undergo cystic softening. Cysts may be parasitic in origin. The cause of many simple cysts is entirely obscure. They may be found in any part of the brain.

Carcinomata are always metastatic and are secondary to a primary growth elsewhere than in the brain. Gummata and vascular tumors are exceedingly rare until after puberty.

As the tumor grows, secondary lesions are produced in most of the cases. These are the result of pressure on contiguous parts of the brain interfering with their function, or of obstruction to the aqueduct of Sylvius or the fourth ventricle preventing the exit of fluid from the interior of the brain and thus causing hydrocephalus. Tumors in the posterior fossa are very frequently accompanied by hydrocephalus. Localized meningitis over tumors superficially situated is the rule, and this may be the cause of some of the symptoms. Rarely, cerebral hemorrhage may be associated.

Etiology.—The causes of cerebral tumors are for the most part unknown. In a few instances there is a history of definite traumatism. Sarcomata may be secondary, carcinomata and tuberculous tumors are probably always so.

Symptoms.—These may be divided into two groups: first, the general symptoms, which are common to tumors of all varieties, are chiefly due to pressure and are more or less independent of location; secondly, the local symptoms depending upon the situation of the growth.

Of the general symptoms one of the most frequent is headache. Though it varies much in its severity, character, and position, it is rarely absent. It is apt to be severe, and may continue for a long period, or it may be intermittent. The location of the pain has no definite relation to the situation of the tumor, nor is the intensity of the pain dependent upon the size of the tumor. It may be accompanied by sensations of tightness, compression, or tension in the head. It may be associated with localized tenderness of the scalp; when this is constant it is a valuable symptom for diagnosis, as it often occurs with tumors superficially located.

General convulsions are frequent in the early stage, but separated by quite long intervals; they become more frequent and more severe as the disease progresses. All degrees of severity are seen, from slight twitchings and temporary loss of consciousness to typical epileptiform seizures. They are most common when the growth is rapid and when complicating meningitis is present. Attacks of vomiting or of localized

spasm may for a considerable time precede general convulsions; and in a single attack there may be first localized and then general convulsions.

Mental symptoms are generally present in great variety and complexity. There may be only fretfulness and irritability, or a marked change in disposition. These symptoms are so frequent from other causes in children that they excite no apprehension, unless to them are added dulness, apathy, and somnolence. Later in the disease there may be attacks of melancholia or there may be periods of wild, almost maniacal, excitement; and, finally, the mental impairment may approach a condition of imbecility.

Disturbances of sleep are frequent. There is usually insomnia, but sleep may be broken by hallucinations, accompanied by attacks of screaming; rarely is there persistent drowsiness until toward the end of the disease.

Optic neuritis or papillo-edema (choked disc) is very frequent, occurring in 80 to 90 per cent of the cases. This is only recognized by the ophthalmoscope, as there may be no disturbance of vision. The choked disc is generally double. It is nearly constant with tumors of the posterior fossa, especially of the cerebellum. It is also very frequent with tumors of the corpora quadrigemina and of the parieto-occipital region. Papillo-edema is usually associated with tumors of the basal ganglia, but is late in appearance or frequently absent with tumors of the pons, corpus callosum or convexity.

Vomiting is a very frequent symptom, but diagnostic only when it occurs suddenly without assignable cause, and without nausea or other symptoms of indigestion. Usually attacks come several days apart, often occurring early in the morning. Vomiting is especially significant when frequently repeated, and of more importance in older children than in infants.

Vertigo is often associated with vomiting. At first it is occasional and seen upon changing position, but later it may be quite constant, especially with tumors in the posterior fossa.

A slow pulse is occasionally observed with brain tumors. It may be as low as 40 or 50 to the minute. This is the result of increased intracranial pressure, and is only found when the pressure is great. It is therefore usually a late symptom. Enlargement of the head, secondary to the hydrocephalus, at times occurs. It is more apt to be found before the fontanel has closed and the sutures are firmly ossified, but separation of the sutures and marked enlargement of the head may take place as late as the eighth or tenth year. Pressure of the tumor may cause erosion of the contiguous bone. The most frequent seat of this erosion is the sella turcica with tumors of the pituitary. This can often be made out by the X-ray, which also shows frequently separation of the

sutures and digital markings on the skull, the result of hydrocephalus. Very infrequently the shadow of a tumor is revealed. Diabetes insipidus is a symptom occasionally associated with tumors at the base, especially when the pituitary is involved.

Local Symptoms.—These depend upon the situation of the tumor, but not at all upon its character. They are the result of pressure or of destruction of brain tissue. They may therefore be irritative or paralytic symptoms. Local symptoms may be wanting entirely, and they may vary much in different cases even with tumors in the same situation. They are modified by the size and by the rapidity of growth, and by the existence of localized meningitis.

Tumors situated in the frontal lobe, as a rule, present few symptoms and may be entirely latent. Irritation of the frontal lobe may extend to the motor area and cause convulsions either local or general; but not often is there paralysis. Tumors of the left side (of the right side in left-handed persons) may cause apraxia, and when in the third frontal convolution, motor aphasia.

Tumors in the motor convolutions along the fissure of Rolando produce the most definite and uniform local symptoms. When situated at the upper portion the leg is affected, at the middle portion, the arm, and at the lower, the face. Irritative symptoms, such as rigidity or clonic spasm, commonly precede for some time the paralysis which results from pressure or destruction. These attacks of localized convulsions begin in the face, arm, or leg; but they usually extend more or less rapidly until all three are involved. They are often followed by slight transient paralysis. Consciousness is often retained and when lost is lost late in the attack. Such attacks are known as "Jacksonian epilepsy," and form one of the most diagnostic symptoms of cerebral tumor. Localized spasm may be associated with anesthesia or other disturbances of sensation. The paralysis generally first affects one extremity—the arm or leg, according to the location of the tumor—and afterward it may involve the entire side, including the face.

If the tumor is centrally located, or at the base, hemiplegia may be an early symptom from pressure on the motor tract. With cortical paralysis there may be associated ataxia and paresthesia or anesthesia.

Tumors of the parietal lobe may give no local symptoms. If the tumor is deeply situated there may be hemianopsia from pressure on part of the optic tract. If the inferior parietal lobule of the left side is affected, there may be word-blindness, or inability to understand written language.

Tumors of the occipital lobe produce, as the only constant local symptom, hemianopsia. This is usually bilateral, affecting the same side of both eyes, being on the side opposite to that of the lesion, i. e., a

tumor on the right side causes blindness in the left half of both eyes, so that the patient sees nothing to the left of a line directly in front of him. Instead of hemianopsia, there may be only irritation and various disturbances of sight.

Tumors of the temporosphenoidal lobe may be latent, or, if on the left side, may cause word-deafness, i. e., inability to understand the significance of spoken language.

Tumors in the island of Reil when situated upon the left side (right side in left-handed persons) may cause motor aphasia or disturbances of speech. If they are large they may produce symptoms by pressure upon the motor tract—hemiplegia or monoplegia.

Tumors of the basal ganglia cause marked general symptoms, but none of a definitely local character. The important symptoms relate to the various tracts or bundles of fibers which pass from the cortex through the internal capsule. These include the motor and the various sensory tracts, the olfactory, auditory, visual, and speech tracts. Any of these may be pressed upon, and the nature of the symptoms will depend upon the size of the tumor and the extent of the pressure. If only the anterior part of the capsule is affected there may be no symptoms; if the middle fibers, hemiplegia and disturbances of articulation; if the posterior fibers, hemianesthesia. All these may be associated, and any of them may be complete or partial. Tumors in this situation are apt to implicate the cranial nerves. Optic neuritis is quite constant, but may not appear early. Localized or general convulsions are rare.

The peculiar symptoms pointing to tumors of the crura cerebri are nystagmus, strabismus, and loss of pupillary reflex, sometimes with general muscular incoördination, and a staggering gait. There is usually third-nerve paralysis on the side of the tumor, and on the side opposite to the hemiplegia with which it is often associated. This variety of crossed paralysis is quite diagnostic. The symptoms of third-nerve paralysis are external strabismus, dilatation of the pupil, and ptosis. While hemiplegia is commonly present with large tumors, it may be absent with small ones, or may appear later than paralysis of the third nerve.

Tumors of the pons are quite common. The diagnostic symptoms consist in crossed paralysis, the cranial-nerve symptoms being on the side of the tumor, and the general motor and sensory symptoms on the opposite side. When the seat is the upper half of the pons, the third and fifth nerves are apt to be implicated, giving rise to ptosis, dilatation of the pupils, external strabismus, trophic disturbances such as ulceration of the cornea, and neuralgic pain in the face. Tumors in the lower half of the pons involve the sixth, seventh, and eighth nerves, causing internal strabismus, contracted pupils, facial paralysis, sometimes deaf-

ness, and auditory vertigo. Other symptoms associated with tumors of the pons are headache, vomiting and optic neuritis; convulsions being rare.

Tumors of the medulla are recognized by the involvement of the glossopharyngeal, pneumogastric, spinal accessory, and hypoglossal nerves. There is difficulty of deglutition, irregular respiration, irregular pulse, and vasomotor disturbances, such as flushing of the face and perspiration. There may be projectile vomiting, polyuria or glycosuria, opisthotonus, difficulty in articulation or in sucking, and in protrusion of the tongue. Hydrocephalus is often marked. These tumors may produce symptoms of pressure upon the motor or sensory tracts—paralysis, or partial anesthesia, with rigidity and exaggerated reflexes.

Tumors of the pituitary gland or in the immediate neighborhood may give characteristic symptoms. These are referred to hypopituitarism, a decrease in the function of the anterior lobe of the pituitary. There may be a marked deposition of subcutaneous fat with a tendency to mental dulness and with a retardation of sexual development at the time of puberty. This is frequently spoken of as "Fröhlich's syndrome." In some children with these symptoms there is an increased sugar tolerance so that as much as 150 grams of glucose may be taken at one time without glycosuria. These symptoms are usually found with benign growths. Malignant growths such as sarcomata are destructive and usually produce no such syndrome. Symptoms frequently associated with pituitary growths are bitemporal hemianopsia from pressure on the optic chiasm and later amblyopia. There may be paralysis of the extraocular muscles. Headache is not a striking symptom and hydrocephalus is inconstant. Acromegaly is rarely seen in children.

Tumors of the cerebellum are especially important, this being the most frequent location in childhood. When only one hemisphere is affected there may be no local symptoms. Tumors involving the middle lobe, or those large enough to produce pressure upon the middle lobe, give rise to vertigo and cerebellar ataxia. Vertigo is especially frequent; it may be associated with headache. Cerebellar ataxia is different from the ataxia due to a spinal-cord lesion, and strikingly resembles that of intoxication. It may increase until the patient is unable to walk, although there is no loss of muscular power. Vomiting is a frequent symptom, as are also optic neuritis and headache, which is usually occipital. When there is secondary hydrocephalus, as is usual, mental symptoms are present, and there may be enlargement of the head. Opisthotonus is occasionally seen, but general convulsions are rare.

Course.—This is usually progressive toward a fatal termination. The rapidity depends much upon the character of the growth. Malignant tumors, especially sarcomata, may cause death in a few weeks.

Tuberculomata may give symptoms for many months but are usually fatal before that time from general miliary tuberculosis or tuberculous meningitis. Occasionally symptoms of brain tumor may be present for several years without any distinct advancement and then with a sudden increase of symptoms death may take place in a few days.

Diagnosis.—Cerebral tumor may be confounded with abscess, chronic basilar meningitis, and chronic hydrocephalus. The symptoms distinguishing tumor from abscess are the following: Tumor may occur at any age; without definite etiology, excepting when tuberculous; the progress is steady, but generally slow, new symptoms being continually added; headache is more constant and more severe; optic neuritis more frequent; cranial nerves more often involved; mental disturbances more marked; focal symptoms are often definite; fever and leucocytosis are absent; duration, six months to two years. As compared with the above, abscess is not so frequent, being especially rare in infancy; there is a definite history of traumatism or ear disease; progress more irregular; symptoms often intermittent; headache less severe; mental symptoms less marked; optic neuritis and involvement of the cranial nerves less frequent; focal symptoms usually indefinite; fever and leucocytosis present except in the latent period; the most frequent complication is acute meningitis.

Chronic basilar meningitis may produce symptoms almost identical with those of tumor in the posterior fossa. It is, however, confined to infancy; hydrocephalus and opisthotonus are much more marked than are usually seen with tumor. An examination of the fluid obtained by lumbar puncture will assist much in the diagnosis.

Chronic hydrocephalus may resemble tumor; this occurs so frequently as a lesion secondary to tumor that the question often arises whether there is only hydrocephalus, or there is in addition a tumor. Hydrocephalus is often congenital, is usually encountered in the first year of life and commonly attains to a greater degree than is seen in secondary hydrocephalus. There is an entire absence of focal symptoms. Papillo-edema is rare but optic atrophy very common.

A diagnosis of brain tumor should not be made from the presence of Fröhlich's syndrome alone. The association of the general symptoms of tumor with hemianopsia or amblyopia or deformity of the sella turcica, is necessary. Many children show adiposity, sluggishness and a moderate delay in the development of the secondary sexual characteristics and eventually manifest nothing abnormal. A diagnosis as to the nature of a tumor is very difficult, but some information upon this point may be gained from the consideration of its etiology, the rapidity of its growth and the age of the patient.

Prognosis.—The prognosis of cerebral tumor is very bad. In the

overwhelming majority of cases the progress is steadily downward until death. Cases are occasionally seen which exhibit all the characteristic symptoms of tumor, even including optic neuritis, which recover perfectly. We have seen several such cases. They are probably not tumors but circumscribed areas of encephalitis that undergo complete resolution. An arrest of the growth very occasionally occurs in tumors of a tuberculous nature and recovery takes place with some function of the brain impaired. Such an outcome is distinctly unusual. The calcified tubercles that are sometimes found at autopsy have usually given no symptoms during life. Very little is to be expected from treatment unless the tumor is susceptible of operative interference.

Treatment.—If there is any reason to suspect syphilis, the iodid of potassium should be given in large doses and continued for a long period. Except for operative measures the treatment is entirely symptomatic. The possibility of total removal of a growth in childhood is very slight. The chief tumors are either infiltrating (gliomata, sarcomata) or part of a more or less generalized tuberculosis. The most favorable tumors for operative removal (endotheliomata) are very infrequent in childhood. The best outlook is probably with cysts. Without operation, however, the result is so nearly always in death that if there is any possibility of removal of the growth it should be attempted. If enucleation of the growth is not possible, cerebral decompression may preserve the sight for a long time and do much to diminish the pain and general discomfort.

HYDROCEPHALUS

Hydrocephalus, or "water on the brain," consists in an accumulation of serum in the cranial cavity. This may be between the dura mater and the pia (external hydrocephalus) or in the ventricles of the brain (internal hydrocephalus). The former is secondary and is quite rare, while the latter is not uncommon. Hydrocephalus may be acute or chronic.

Acute hydrocephalus is secondary to basilar meningitis, which is usually of tuberculous origin. The terms tuberculous meningitis and acute hydrocephalus are sometimes used synonymously. A moderate distention of the ventricles is frequent in all varieties of acute meningitis. The amount of fluid in acute hydrocephalus is not great, there being rarely more than three or four ounces present.

Chronic external hydrocephalus except in its mild form is extremely rare, and is nearly always a secondary lesion. It may follow meningeal hemorrhage, pachymeningitis, or any lesion causing cerebral atrophy. It is seen in its most marked form associated with congenital malforma-

tions of the brain, particularly imperfect development of the hemispheres. (See Fig. 103.) On incising the dura mater a few ounces, or sometimes even a pint, of fluid may escape. The convolutions are somewhat flattened, and may be greatly atrophied. Other lesions are

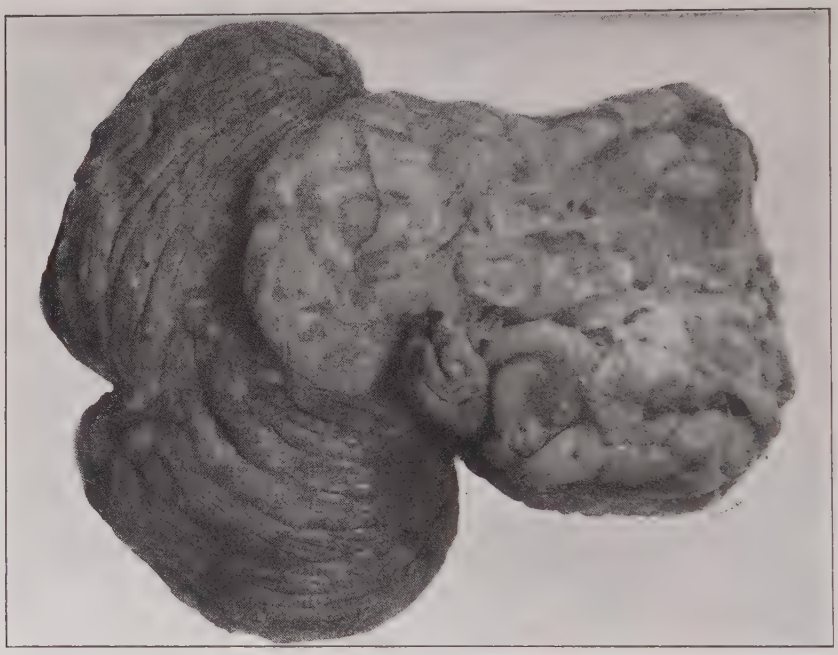


Fig. 103.—Brain in External Hydrocephalus, Showing Imperfect Development of the Hemispheres. Patient three and a half months old; head measured 20½ inches; increase in size, 2 inches in the six weeks before death; symptoms were typical of ordinary internal hydrocephalus. In the picture the small size of the cerebrum A is best judged by comparison with the cerebellum B, which is normal. The hemispheres were rudimentary; the basal ganglia were normal; the cranial cavity contained about one pint of fluid.

found either in the brain or in the dura mater. External hydrocephalus may cause enlargement of the head and separation of the sutures, and in fact most of the symptoms of the internal variety; but usually it is not severe enough to give rise to any decided symptoms.

CHRONIC INTERNAL HYDROCEPHALUS

This is the important variety, and when no qualifying term is mentioned this is the form of hydrocephalus which is always understood.

Internal hydrocephalus may result from many different diseases of the brain and meninges. In some the amount of fluid is moderate and

its presence adds little or nothing to the symptomatology of the condition. Tuberculous meningitis is an example. In others, such as tumors of the base of the brain, the collection of fluid may be considerable and cause definite symptoms but the primary condition and not the hydrocephalus is the important one.

Etiology.—The etiology of hydrocephalus in many instances has been obscure. This has been largely due to the difficulty of studying brains at autopsy on account of the injury that results from their

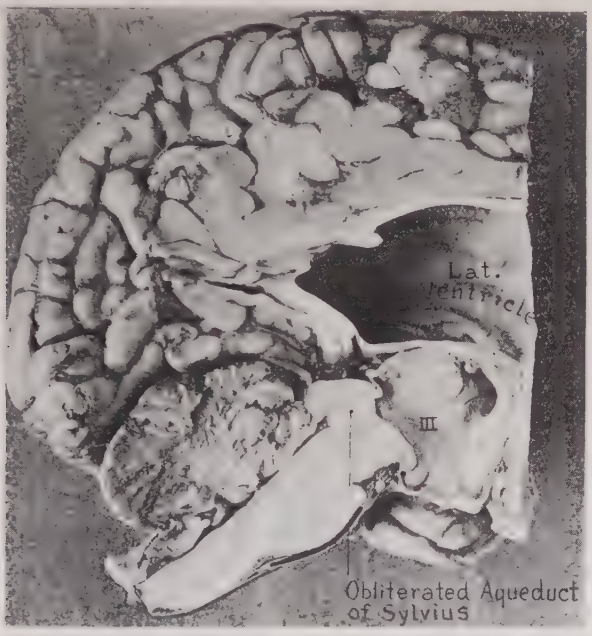


FIG. 104.—SAGITTAL SECTION OF 6 MOS. OLD CHILD, DYING OF HYDROCEPHALUS, SHOWING DILATED LATERAL AND THIRD VENTRICLES AND OBLITERATED AQUEDUCT OF SYLVIUS. (From Dandy and Blackfan.)

removal unless special precautions are taken. It has been customary to divide cases of hydrocephalus into the primary, when the cause was obscure, and secondary, when the cause such as tumor or abscess was readily apparent. There is no longer any justification for such a division. It seems now established that internal hydrocephalus is always a secondary condition depending upon mechanical causes. The recent studies of Dandy and Blackfan have shown that the cerebrospinal fluid is formed by the choroid plexus in the lateral, third and fourth ventricles —but that it is not absorbed there. It passes out of the brain through the aqueduct of Sylvius into the fourth ventricle and from there to the subarachnoid space by means of the foramina of Magendie and of Luschka. There is an automatic regulation of production and absorp-

tion by means of which the amount of fluid is maintained at the proper level. Hydrocephalus results when the aqueduct or the foramina are obstructed; or when in consequence of injury to the meninges as a result of inflammation, the cerebrospinal fluid can not be absorbed with sufficient rapidity from the subarachnoid space. In the latter instance the fluid is dammed back toward its source and the greatest pressure is thus exerted on the interior of the ventricles.

Obstruction to the flow from the ventricles is frequently brought about by a narrowing or complete absence of the aqueduct. (Figs.

Fig. 105.—Sagittal Section of Normal Brain of an 8 Months Old Child, showing Patent Aqueduct of Sylvius. (From Dandy and Blackfan.)

104, 105.) This condition must be considered a congenital abnormality. Obliteration of the foramina, however, is almost always the result of inflammation. This may occur in intra-uterine life or at any time after birth. Except for those cases plainly following upon meningococcus meningitis, the organism causing the inflammation is unknown. Interference with the absorption of cerebrospinal fluid is dependent upon some previous meningeal inflammation. It is probable that this in turn may be of intra-uterine or extra-uterine origin. No sufficient pathological examination of cases due to this cause has been made. It is the opinion of Dandy and Blackfan that the diminished absorption is due to adhesions limiting the size of the subarachnoid space.

In a large proportion of cases the disease is congenital, hydrocephalus beginning in the latter months of intra-uterine life. Syphilis is responsible for a certain proportion of cases. By some authors the proportion is considered a large one. Sufficient data have not been accumulated since the introduction of the Wassermann reaction to justify a conclusive statement upon this point. In our own experience the association is not frequent,—certainly fully four-fifths of the cases are not syphilitic. Heredity is a factor of some importance, as numerous instances are on record where two children in the same family have been affected. The most obvious explanation seems to be that the same meningeal inflammation or the same congenital abnormality has existed.

Hydrocephalus not infrequently develops after successful operations upon spina bifida or encephalocele. In such an event it is likely that an inadequate meningeal absorption was compensated for by the increased area afforded by the sac of the spina bifida. When the sac is removed the absorption of fluid is no longer adequate. There is no reason to believe that neuroses, alcoholism, tuberculosis or consanguinity in the parents is responsible for hydrocephalus. The rachitic head has been so often mistaken for hydrocephalus that an erroneous notion has arisen as to the association of the two diseases. There is no etiological connection between them.

Pathology.—Depending upon the cause and the duration of the condition the amount of fluid may be small or large. It may be only a few ounces or several pints. We have seen three pints in an infant two weeks old and five pints in one who died at four months. Much larger quantities than this have been reported, but in children living several years. In composition the fluid resembles normal cerebrospinal fluid. Minor changes have been reported but are not uniform. The fluid may be slightly yellow and there may be an excess of cells in cases following a recent meningitis. The effusion may become purulent from accidental infection resulting from operation, from rupture, or from infection through the sac of a complicating spina bifida.

A satisfactory examination of the brain can only be made if it is injected with formalin through the carotid arteries and two or three hours allowed to elapse before it is removed. The meninges may be normal. Frequently, however, they are thickened and there may be adhesions between them and the brain, especially at the base. The cisterna magna may, in this way, be greatly diminished in size or actually obliterated and adhesions may close the foramina of Magendie and of Luschka. The aqueduct of Sylvius may not be demonstrable. Ordinarily this is as large as a small quill. Microscopically, remains of it may be found in small islands of ependymal cells with or without a central opening. A gliosis has obliterated the aqueduct.

The chief changes in the brain result from the distention of the ventricles by fluid. This continues until the hemispheres are destroyed to a greater or less extent. The convexity of the brain thus suffers most. The basal ganglia and cerebellum are somewhat flattened but otherwise relatively normal. The progressive distention results in a gradual thinning of the brain substance which forms the ventricular walls; often these are found only one fourth of an inch in thickness or the cortex may be a mere shell (Fig. 106). The ependyma of the ventricle and the pia mater are at times actually in contact, all of the brain tissue having been absorbed. The brain in such instances resembles a large double cyst. In less marked cases there may be only a flattening of the convolutions. The foramen of Monro is dilated, and occasionally the foramen of Magendie also. The septum lucidum is greatly thinned or may have disappeared. The brain is anemic and the gray and white substance may be indistinguishable. The ependyma may be normal. It is usually somewhat thickened and pale, sometimes granular and may be infiltrated with new cells. The microscopical changes are inconstant and not marked. There is a tendency to atrophy and disappearance of the ganglion cells.

FIG. 106.—VERTICAL TRANSVERSE SECTION OF A BRAIN IN CONGENITAL HYDROCEPHALUS. From a child who died at the age of three weeks. *A*, distended lateral ventricle; *B*, its descending horn.

The cranium is markedly affected. The bones are often very thin; the fontanels are very large and the sutures, especially those of the vault, widely separated. There may be a formation of Wormian bones. After the removal of the fluid which alone gives it its configuration, the head may collapse. It should not be forgotten, however, that hydrocephalus may coexist with premature ossification, in which case the head may be small. Pressure of the fluid upon the roof of the orbit causes it to become less concave or even convex. When recovery occurs the sutures and fontanels may close with the help of the Wormian bones, and irregular thickening of the bones of the skull take place. The most frequent lesion associated with congenital hydrocephalus is spina bifida;

more rarely there is meningocele or encephalocele. Sometimes there are deformities in other parts of the body, such as club foot or hare-lip.

Symptoms.—Many cases of hydrocephalus are congenital and the child may die *in utero*. At other times the process may be so far advanced before birth that Cæsarian section or puncture of the head may be necessary before delivery is possible. In perhaps the majority of cases, no symptoms are observed at birth, or the head is only slightly larger than normal. Usually, nothing is noticed until the child is two or three months old, when it is discovered that the head is increasing in size at an abnormal rate. Instead of the usual half an inch a month it may be two or three times this. If the progress is rapid, other symptoms are soon evident—the infant cannot hold up his head, he is lethargic and all his perceptions are dulled. Only in rare instances is there blindness, but there is usually some interference with sight, which is, however, very difficult to make out with young infants. Very rarely there is deafness. The pupils are usually contracted and equal, though they may be dilated. Nystagmus and convergent strabismus are often present. In severe cases the eyes protrude slightly and are rotated downward, leaving some of the sclera visible. This gives a very characteristic expression and is due to the alteration of the roof of the orbit. If the hydrocephalus has developed very rapidly, a papillo-edema is sometimes seen. This is, however, exceptional and optic atrophy of greater or less extent is the rule.

There is usually rigidity of the muscles of the extremities, more marked in the legs, sometimes also in the arms; the hands being clenched with the thumbs adducted. The reflexes are exaggerated.

For a time the nutrition is well maintained, but when the head enlarges markedly, the body wastes and the disproportion between the two may seem greater than it really is. Convulsions are seldom seen. Cases which develop early and progress rapidly rarely live to the end of the first year, and are often fatal before six months. The causes of death are marasmus, convulsions, intercurrent disease, and rarely rupture of the head.

The cases which develop slowly are usually those that follow some meningeal inflammation. There may be a history of frank cerebrospinal meningitis. Sometimes there is only a history of unexplained fever without symptoms to draw attention to the meninges. When the symptoms develop slowly, the head may be but little larger than normal. The brain seems able to tolerate an almost indefinite amount of pressure if this develops gradually. The surprising thing about many of these cases is that the distinctly cerebral symptoms are so few. The more readily the bones of the skull yield to pressure, the fewer are the nervous symptoms, hence, other things being equal, they are less marked when

the disease begins before the sutures are firmly ossified than in the later cases. A comparatively small amount of effusion may cause very marked symptoms in a child two or three years old, while a much larger amount in an infant of a year may produce much less disturbance.

Even though the progress of the disease is slow the development of the children is greatly retarded. If the course is progressive, however, death eventually takes place, although it may be postponed for many months. The special senses are generally not noticeably affected; but intelligence in most cases is interfered with, in some only slightly; in others, very markedly, while some are idiotic. Contractions of the extremities are occasionally seen but usually more of the hands than of the legs. Sensation is not often affected. The course is a very chronic one and from time to time there may be exacerbation of the symptoms.

Spontaneous arrest may occur at almost any stage. There may remain only a moderate enlargement of the head and fair intelligence, or recovery may be delayed until the head has reached an enormous size, and the child, on account of this, quite unable to move. Such an outcome, however, is rare.

Dandy and Blackfan have shown that there are two distinct varieties of hydrocephalus, one due to obstruction and the other due to diminished absorption of the cerebrospinal fluid. When a solution of phenolsulphonephthalein is injected into the normal ventricle the dye appears in the cerebrospinal fluid within five minutes and is absorbed very rapidly, so that 15 to 20 per cent of it is excreted by the kidneys in the course of two hours. After its injection into the spinal subarachnoid space, its appearance in the urine is prompt and from 35 to 60 per cent is excreted in the course of two hours.

In the one variety of hydrocephalus, the 'phthalein, after injection into the ventricle, does not appear in the fluid obtained by lumbar puncture for a long time, and is excreted by the kidneys very gradually and during several days. If it is injected into the subarachnoid space, the excretion is as prompt as under normal circumstances. This demonstrates that there is an obstruction to the outflow of fluid from the ventricles into the subarachnoid space, the cause of which may be malformations or adhesions blocking the foramina of exit.

In the other variety, the 'phthalein injected into the ventricle appears promptly in the subarachnoid fluid but is excreted by the kidneys slowly and when it is injected into the lumbar region of the cord, it is also excreted slowly. This delayed absorption is the result of inflammation which has injured the meninges and diminished the area for absorption.

Prognosis.—Cases developing soon after birth and progressing rapidly are usually fatal before the end of the first year. It is very rare

that a hydrocephalic child reaches the age of seven years. The process may, however, go on up to a certain age and then cease spontaneously and the child may go through life with a head much larger than normal and usually with a somewhat impaired mental condition. In others the mentality is nearly or quite normal and yet some muscular weakness or even paralysis persists. This arrest of hydrocephalus is probably brought about by an adjustment which has taken place by which the meninges are able to absorb sufficiently to keep pace with the production of the cerebrospinal fluid.

Diagnosis.—The most important symptom is the enlargement of the head, and this can only be arrived at by careful measurement and comparison with the normal size. The rapidity of growth is quite as important for diagnosis as the fact of enlargement. If the head grows as much as an inch a month there can be little doubt. The enlargement most frequently confounded with hydrocephalus is that which occurs in rickets. In the latter disease it is almost invariably irregular; there are prominences over the two frontal eminences and over the parietal bones, often with furrows between them; the size of the head is chiefly due to thickening of the bones of the skull; the marked prominence of the forehead is not seen, and the increase in the biparietal diameter is not present; furthermore, there are other signs of rickets.

Pachymeningitis interna may be confounded with hydrocephalus. The fluid, however, is usually either reddish and reddish-yellow or is quite blood-stained and may contain red blood-cells. A differential diagnosis may be very difficult.

Treatment.—If syphilis is suspected, energetic treatment by mercury and salvarsan should be instituted. In our experience, benefit from these has not been very marked and little is to be expected unless they are employed very early. Repeated lumbar punctures have, in a small proportion of cases, apparently been of value in bringing about an arrest of the process. Since differentiation between the different varieties has been possible, we have seen benefit result in cases with a free communication between the ventricles and the subarachnoid space. On *a priori* grounds, this is the only variety in which lumbar puncture offers a possibility of benefit. Various operative measures have been proposed. Communications have been established between the lateral ventricles and the subarachnoid space. A number of cases have been treated in this way. The dangers of the operation are considerable; nearly half of the patients have died as a direct result. Of those who have survived, a few have shown striking improvement, but no complete cures have been reported.

Drainage into the jugular vein and into the subcutaneous tissues has also been employed. These operations offer but little possibility of cure.

Retrogression of the symptoms is not to be looked for. The most that can be hoped is to prevent any further injury to the brain. With the knowledge that has been recently acquired in regard to the cause of this disease, there is a much greater possibility of intelligently attacking the condition by surgical means.

Cranial Deformities Associated with Hydrocephalus.—Various cranial deformities may at times be associated with a considerable degree of hydrocephalus. The two most frequent of these are oxycephaly ("steeple-head" or *turmschadel*) and scaphocephaly. In oxycephaly (Fig. 107) the head is very high and short; in scaphocephaly (Fig. 108), it is narrow and elongated from before backwards. In addition to the change in the shape of the head, there may be with either form some degree of exophthalmus and optic atrophy which causes impairment of vision. This varies in severity from slight interference with sight to complete blindness. The intelligence is usually quite normal. Smell is often completely lost. Taste very rarely is affected. These cranial deformities seem to have no effect upon the duration of life. They are not amenable to treatment and the optic atrophy is usually progressive. It is possible that cerebral decompression may retard the optic changes but this has not yet been sufficiently employed to warrant a conclusion as to its influence.

FIG. 107.—OXYCEPHALY WITH EXOPHTHALMUS AND PARTIAL BLINDNESS, WITH OPTIC NERVE ATROPHY. Child 2 years old.

FIG. 108.—SCAPHOCEPHALY; in infant 17 months old.

INFANTILE CEREBRAL PARALYSIS

(Spastic Diplegia, Paraplegia, or Hemiplegia)

Under the term cerebral paralysis are included several groups of cases with causes quite dissimilar, but having certain definite clinical features in common. While the symptomatology is quite clear, there are many questions relating to the pathology that are not yet fully settled, although much has been added to our knowledge within the last few years. Paralysis depending upon cerebral tumor, abscess, or hydrocephalus is not included in this chapter.

FIG. 109.—EXTENSIVE ATROPHY AND SCLEROSIS OF THE RIGHT HEMISPHERE. From an infant seven and a half months old; probably the result of a meningeal hemorrhage at birth. *History.*—Twelve hours after birth was seized with general convulsions, which continued for three days. No other symptoms noticed till one month before death, when weakness of the left arm was observed. Never held head erect. Was plump and well nourished; died from erysipelas. *Autopsy.*—Pia not adherent; a large cyst occupied the region of the occipital and posterior part of the parietal lobes, showing in its floor discoloration and pigmentation, evidently from an old hemorrhage. Right optic nerve, tract, and crus much smaller than the left.

The cases of cerebral paralysis may be divided into three groups, according as the paralysis depends upon conditions existing prior to birth, upon those connected with birth, or upon those of subsequent development.

I. **Paralysis of Intra-Uterine Origin.**—This is the least frequent condition. In such cases there is some congenital defect in the brain, due sometimes to arrest of development, at others to such intra-uterine lesions as hemorrhage or thrombosis. There may be porencephalus, or cysts extending deeply into the substance of the brain, sometimes communicating with the ventricles. The origin of this condition is for the most part unknown. In rare cases the paralysis is due to cortical agenesis, a

condition in which the brain may seem normal to the naked eye, but the microscope shows a more or less complete arrest in the development of the cells of the cortex, usually affecting both hemispheres. In still other cases there are found gross defects in development in the motor centers of the cortex. Such a lesion is shown in Fig. 114. Cases in which there is conclusive evidence of intra-uterine hemorrhage are very rare.

In most of the paralyses due to intra-uterine lesions, loss of power is only one of the symptoms and usually not the most prominent. It is rare that there is not some mental impairment, and usually idiocy is present. The type of paralysis is nearly always diplegic or paraplegic. When this is due to arrested cortical development, a general flaccidity of the muscles may be seen instead of the rigidity so characteristic of the other forms of cerebral paralysis.

II. **Birth-Paralysis.**—Cerebral birth-paralysis is due in nearly all cases to meningeal hemorrhage. The primary lesions and the early symptoms have already been described in connection with the Diseases of the Newly Born. The secondary lesions present considerable variety. There may be found (1) meningo-encephalitis, (2) atrophy and sclerosis of the cortex, (3) cysts upon the surface, (4) secondary degenerations in the spinal cord.

1. *Meningo-encephalitis.*—This lesion is often quite diffuse. There is thickening of the pia mater, and it is usually adherent to the brain substance. The cortex is involved to a variable degree, depending somewhat upon the time which elapses between the initial lesion and the autopsy. The following were the microscopical changes found in the brain of a child in the Babies' Hospital, who died at the age of one year of measles[1]: The lesions were found everywhere in the cortex. The pia was universally adherent, and showed general cellular infiltration; its blood-vessels showed marked cell proliferation, and the veins in the sub-pial space were dilated and filled with blood. In the pia dipping in between the convolutions similar changes were present. In the cortex few, if any, normal pyramidal cells were found, but in the outer layers were an enormous number of small glia cells. Many of the blood-vessels showed a cell-proliferation of their walls. There was also degeneration in the pyramidal tracts of the lateral columns of the cord.

2. *Atrophy and Sclerosis.*—These changes vary much in extent and degree. There may be only a circumscribed area in which the convolu-

[1] The child was a first-born, delivered after a dry labor of forty-eight hours. He was asphyxiated, and from the first days of his life he had attacks of convulsions, usually repeated many times a day. (Photographed during one of these attacks. Fig. 110.) The child had the symptoms of typical spastic paraplegia—the arms being, however, slightly involved—retarded mental development, and convergent strabismus.

tions are small, firmer than usual, and covered with an adherent pia, or there may be an atrophy so extensive as to involve a large part of one hemisphere (Fig. 109), or sometimes of both hemispheres. Usually the lesion is somewhat diffuse over the convexity of both sides, and much more frequently of the anterior than of the posterior half of the brain. Where a depression of the brain exists the space is filled with cerebro-spinal fluid, and in many cases there is a deformity of the skull.

3. *Cysts upon the surface* may occur alone or in connection with the lesions just mentioned. These are usually small, about the size of a walnut, but they may cover a large part of a hemisphere. Such large cysts are sometimes classed as cases of external hydrocephalus.

Fig. 110.—Convulsions in Spastic Paraplegia.

4. *Secondary degenerations* of the internal capsule and the lateral columns of the cord are found in most of the cases associated with extensive atrophy and sclerosis, and in many of those in which only meningo-encephalitis is present.

Symptoms.—The type of paralysis will, of course, depend upon the extent and position of the original lesion. A diffuse lesion is followed by diplegia; one not quite so extensive by paraplegia; one affecting one side only, by hemiplegia, or even monoplegia, though this is very rare. The relative frequency of the different forms will vary according to the age at which the patients come under observation. According to our observations, which have been chiefly upon infants, the cases of diplegia and paraplegia have outnumbered those of hemiplegia more than four to one. The great majority of the congenital cases, or those due to hemorrhage occurring at birth, are without doubt diplegias or paraplegias, and very many of them succumb during the first two years; however, the cases of hemiplegia, because of the less serious lesion, live much longer. Diplegia and paraplegia will therefore be considered as

the characteristic types of cerebral birth-palsy, as the cases of hemiplegia do not differ from those due to later causes—i. e., the acquired form.

In the most severe cases that survive the symptoms of the early days of life there remains some rigidity of the extremities, chiefly of the legs, which is constant or intermittent, slight or well marked. There is often spasm of the muscles of the neck and trunk, giving rise to opisthotonus. In many cases there are frequent attacks of convulsions. The general physical development of the child is often interfered with, so that he remians small and delicate, or perhaps dies of some acute disease in early infancy, never having been able to sit erect, or even support his head. In other cases the general nutrition is not affected, and life may be prolonged indefinitely, but usually with some mental impairment. This is seen in all degrees; it may be so slight as not to be noticed until the child is two or three years old, or the child may be idiotic. Often these children are not able to stand until they are over three years old and do not walk alone until they are four or five years old, and then with a peculiar cross-legged gait, owing to spasm of the adductors of the thighs. This may be so great as entirely to prevent walking, and while sitting or lying the thighs may cross each other. These form the typical cases of spastic paraplegia, sometimes called "Little's disease" (Fig. 111). All the reflexes are greatly exaggerated. The arms are much less affected than the legs, and in about half the number they are not involved at all.

Fig. 111. — Spastic Paraplegia, Child two and one-half years old. New York Foundling Hospital, unable to walk or even to stand without assistance. The habitual position of the limbs, which is due to strong adductor spasm, is shown in the picture.

In the milder cases the early symptoms may be overlooked, and nothing excite suspicion until the infant is six or eight months old. There is then discovered unmistakable muscular weakness: the child does not sit up, or even hold up the head when the trunk is supported. Often there is observed before this time a tendency to stiffen the body and to throw the head backward, owing to spasm of the cervical or spinal mus-

cles. The muscular weakness is often mistaken for rickets, or regarded simply as backwardness. A closer examination usually discloses the presence of some rigidity of the extremities, particularly of the legs, and exaggeration of the knee-jerks. As the child grows older other symptoms of imperfect development become more and more evident.

There are changes in the shape of the skull, this being usually smaller than normal in all its diameters, or there may be asymmetry. There is an arrest of development in the paralyzed limbs. These are both smaller and shorter than normal. In many cases abnormal movements are seen, which may be of an irregular choreic type, or they may be athetoid. Epilepsy develops in from thirty-three to fifty per cent of all these patients.

III. **Acute Acquired Paralysis.**—This is usually of the hemiplegic type, although diplegia and paraplegia may in rare instances be met with. This group includes cases developing at any time after birth, but the great majority of those seen in childhood begin before the fifth year.

The etiology is often obscure. The paralysis sometimes follows traumatism. It is occasionally seen in the course of scarlet fever, measles, diphtheria, variola, pneumonia, or pertussis. The frequency with which these cases are ushered in with convulsions has led many to assign this as the cause of the paralysis. It is probable that the convulsions are more often the result than the cause of the lesion. In the acute inflammatory cases the cause is probably the same as in acute poliomyelitis.

Lesions.—The lesions of acute cerebral palsy may be grouped under three heads: (1) those of the blood-vessels: (2) those of the membranes; (3) those of the brain substance.

1. Lesions of the Blood-vessels.—There may be hemorrhage, embolism, or thrombosis. Hemorrhage is by far the most important. It is usually meningeal, rarely cerebral. It occurs more frequently at the convexity than at the base, and is often diffuse. Meningeal hemorrhage may result from pachymeningitis. It may be due to traumatism, when it is also from the dura mater; or from the acute hyperemia accompanying paroxysms of pertussis, when it may be from the dura or the pia; or it may be secondary to thrombosis of the superior longitudinal sinus. The association of hemorrhage with sinus-thrombosis is not very infrequent. It was found in two of our autopsies upon patients who died of pneumonia. Cerebral hemorrhage is extremely rare, but it occurs even in young infants.

Embolism is rarely found unless associated with acute rheumatic endocarditis, and then usually in children who are over seven years old. As in adults, the usual seat of the embolus is a branch of the

middle cerebral artery. Thrombosis has been met with in a small number of cases, but is extremely rare.

2. Lesions of the Membranes.—These are generally the result of an old cerebrospinal meningitis; sometimes they may be of syphilitic origin. In both, however, the process is rarely confined to the membranes; it is a meningo-encephalitis.

3. Lesions of the Brain Substance.—Atrophy and sclerosis are found in a large number of the autopsies made upon cases when the paralysis has been of long standing. They represent terminal conditions, however. They vary in severity and extent, and are followed by secondary degeneration in the cord, as in cases of birth paralysis. There may be the same development of cysts of the pia mater, or an accumulation of fluid in the arachnoid cavity, these taking the place of the atrophied convolutions. The nature of the primary lesion in these cases is not always clear. In a certain number of them it is an acute poliencephalitis, analogous to acute poliomyelitis, and probably due to the same cause. The cerebral lesion may be associated with cord lesions or it may occur alone. Their nature is considered in the chapter on Poliomyelitis. In still other cases a chronic diffuse encephalitis with atrophy is found at autopsy, closely resembling the conditions which follow a meningeal hemorrhage occurring at birth, yet the children were normal up to the second or third year, and there was no acute onset.

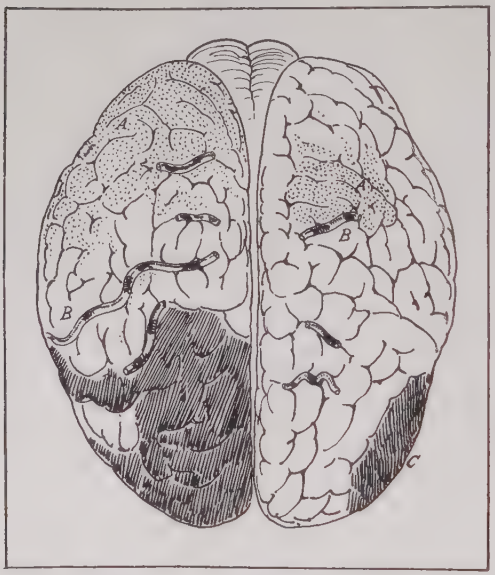


FIG. 112.—RECENT MENINGEAL HEMORRHAGE. Brain of an infant seven months old in the Babies' Hospital. *A*, punctate hemorrhages; *B*, thrombosed vessels; *C*, diffuse extravasation.

Acute paralysis sometimes occurs for which no explanation can be found at autopsy. An infant with pneumonia was admitted to the Babies' Hospital, who had developed, a few days before, typical right hemiplegia. It came on suddenly, with convulsions, and involved the face, arm, and leg. The arm and leg appeared to be completely paralyzed, but in the face the paralysis was incomplete. The paralysis

had begun to improve somewhat at the time of the child's death, which occurred a little over a week after the onset. At the autopsy no gross lesion could be discovered. A careful microscopical examination was made, and nothing abnormal was found except a slight increase of small spheroidal cells about some of the meningeal and cortical vessels of the motor area. Such cases are most likely a cerebral form of poliomyelitis.

Symptoms.—While diplegia and paraplegia are occasionally seen, the great majority of cases of acquired cerebral palsy are of the hemiplegic variety. When diplegia and paraplegia occur, it is usually in early infancy, and their symptoms and course differ in no wise from the birth palsies. We may therefore regard hemiplegia as the chief manifestation of acquired cerebral palsy.

The onset of the paralysis is almost invariably acute, with convulsions, which are usually repeated, and in severe cases followed by loss of consciousness. In the secondary cases these are generally the only symptoms. In one of our cases the patient went to bed apparently well, and awoke in the morning with hemiplegia. Such an onset, however, is very exceptional.

When the paralysis is due to acute poliencephalitis, the onset is usually with high fever, vomiting, often convulsions, followed by delirium or stupor. These general symptoms continue for a variable time, usually two or three days, before paralysis is seen. The temperature in most cases is from 101° to 103° F., and the fever sometimes follows, sometimes precedes, the convulsions. The loss of consciousness may last for several days, and the paralysis is frequently not discovered until consciousness is regained. If there is a very extensive lesion there may be diplegia, deep coma, and death, but this is very infrequent. Usually the lesion is more limited, and the symptoms are those of typical hemiplegia. The face sometimes escapes, and if involved it generally soon recovers. The paralysis of the arm and leg is at first complete, but may improve rapidly in the course of a few weeks. Disturbances of sensation may be present, but are usually of a transient character. After a variable period, from one to several weeks, the patient begins to use the paralyzed extremities, first the leg, afterward the arm, as in adult hemiplegia. The convulsions may be repeated for the first day or two, but prolonged or continuous convulsions are rare. They may be general or unilateral. With lesions of the left side of the brain, speech may be affected, and not infrequently in young children when the lesion is upon the right side. The reflexes are increased upon the affected side, and a slight ankle-clonus may be present.

After a few weeks the child may be able to walk, dragging the affected leg. The recovery in the leg is sometimes complete, but in most

cases a slight halt in the gait remains. The arm usually recovers more slowly than the leg, and contractures are likely to develop after a variable time, generally two or three years. In Fig. 113 is shown a characteristic deformity of the upper extremity. Contractures of the leg lead to various forms of talipes, generally equinus, from shortening of the tendo-Achillis. Sometimes the arm or the leg recovers so perfectly that the case may be regarded as one of monoplegia. In old cases the paralyzed limbs are atrophied; there is more or less rigidity, and the spastic condition may be quite marked. We have seen this limited to a single group of muscles in the leg. Aphasia is common in right hemiplegias, and it is not very rare in those of the left side, because infants appear to use both sides of the brain with nearly equal facility.

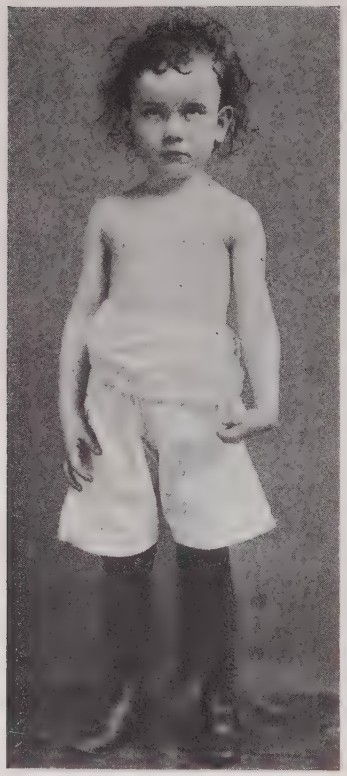


Fig. 113. — Deformity of Left Hand the Result of Contractures Following an Attack of Hemiplegia Four Years Before. Child seven years old.

The mental condition of these children is often normal, in striking contrast with the cases of congenital diplegia. The earlier the paralysis occurs the more likely are mental symptoms to be present, since we have here not only the direct effect of the lesion, but an arrested development of some part of the brain. Epilepsy is not an uncommon sequel; it may be of the Jacksonian type, or there may be attacks of general convulsions. In other cases there are post-hemiplegic movements of a choreic or athetoid character, or irregular incoordinate movements.

Prognosis of Infantile Cerebral Paralysis.—In diplegia and paraplegia the outlook is always unfavorable. A very large number of these cases which are due either to intra-uterine or birth lesions never reach the third year, but die in infancy from malnutrition or acute intercurrent disease. Those who survive usually show serious mental defects, and many are practically helpless on account of the extreme spastic condition of the muscles of the extremities.

In hemiplegia the prognosis is much more favorable. In most of these cases the paralysis is of the acute acquired variety, and the later

the period of onset, the less likely is the brain to be seriously damaged. In some of these patients complete recovery takes place; in others the residual paralysis is so slight as to be easily overlooked except on careful examination, the occurrence of epilepsy being perhaps the first thing which leads one to suspect that a previous paralysis has existed. The great majority of children who have suffered from infantile cerebral palsy have some degree of permanent paralysis and usually some deformities from contractures, the extent of both varying, of course, with the severity of the primary lesion. In all cases seen in young infants it is exceedingly difficult to give a prognosis in regard to future mental development. As a rule, the impairment is directly proportionate to the extent of the paralysis and its intensity.

Diagnosis.—The diagnosis between the congenital and acquired forms of cerebral palsy is of no great practical importance, and it may be impossible; for the symptoms in congenital cases are often not sufficiently marked to attract attention until children are old enough to sit alone or to walk.

It may be quite difficult to distinguish cerebral paralysis from infantile spinal paralysis. The history of an acute onset, the atrophied limbs, the deformities, and the absence of sensory disturbances, may be found in both conditions. Spinal paralysis is, as a rule, monoplegic, and often affects but a single group of muscles. Cerebral paralysis is either diplegic or hemiplegic in character, and even though only a leg or an arm may seem to be affected, a critical examination will usually reveal the fact that the other limb of the same side has also suffered. The presence of rigidity and exaggerated reflexes is quite as important evidence of this as loss of power. The electrical reactions, however, are usually conclusive; the reaction of degeneration is absent in cerebral paralysis, while it is usually present in spinal paralysis.

Simple as the differentiation may seem in most cases, the mistake is frequently made of confounding cerebral diplegia, particularly of the flaccid type, with rickets. Cases of acute acquired paralysis at the onset may be mistaken for acute meningitis, but early loss of consciousness, the early development of the paralysis, its permanent character, and the shorter duration of the acute symptoms, usually distinguish these cases from those of meningitis. The only definite means of differential diagnosis is by lumbar puncture; this gives negative results in cerebral paralysis and positive results in meningitis.

Treatment.—The course and the results of cerebral paralysis depend upon the extent of the injury to the brain, its nature, and the age at which it is inflicted—all these being conditions which are beyond the power of the physician to modify or control. The treatment of cerebral palsy is therefore extremely unsatisfactory. For the congenital cases

practically nothing can be done, except for the deformities and complications. The acquired cases during the acute onset are to be managed like all other cases of acute cerebral congestion or inflammation—absolute rest, ice to the head, and bromids. Electricity is not to be used in early cases, and little or nothing is to be expected from it in the late ones. Much can be accomplished in an educational way for the mental derangements resulting from cerebral palsy. An important part of the treatment relates to the deformities. Many of these may be prevented by the early use of orthopedic apparatus. Serious deformities in old cases may be greatly benefited by tenotomy or myotomy, followed by the use of suitable apparatus. The results of all other operative measures have been in our experience most unsatisfactory. Epilepsy is to be treated as when it depends on other causes.

AMAUROTIC FAMILY IDIOCY

Amaurotic family idiocy is a relatively rare disease. It is confined, almost, if not entirely, to the Jewish race. It shows strong familial tendencies—often two or three and sometimes even four or five children in the same family dying of the disease. There are no other known etiological influences.

The first symptoms are usually noticed between the sixth and the tenth months, up to which time the infant has generally appeared normal. At first it is only noticed that the child is making no progress in his development, or that his eyesight is not so good as formerly. He does not gain in ability to sit up or to use his muscles; he lies quietly, does not respond as he once did, and takes less interest in his surroundings. After a few weeks it is clear that the child, instead of advancing, is actually retrograding both physically and mentally. His muscles become so weak that he can no longer sit up or even hold up his head. Vision becomes less and less distinct; the child no longer recognizes the faces of friends or objects shown him. Finally, he becomes dull, apathetic and quite indifferent to his surroundings; then it is evident that he can not see at all. In the early stages the muscles are usually weak and flaccid; later there is rigidity with increased knee jerks and often marked spasticity. Children with amaurotic family idiocy are often fat and well nourished, but with the onset of weakness loss of weight occurs and eventually this may be so extreme that the emaciation may be a prominent factor. There may be general convulsions. The characteristic features of the disease are revealed by the ophthalmoscope. Occupying the place of the macula lutea there is a large, milky blue or white area with a bright cherry-red spot in its

center. With this there is also atrophy of the optic discs. The ocular changes are symmetrical.

The outlook is absolutely bad. The disease is progressive and usually fatal within a year from the time when the first symptoms are seen; but occasionally the blind, helpless child may live for several years if feeding with the stomach tube is resorted to, for swallowing eventually may become quite impossible.

There are characteristic pathological changes to be found in the cells of the central nervous system. The brain itself is not diminished in size, but is more firm and elastic than normal. The same is true of the cord. Microscopically, the ganglion cells show a marked and striking degeneration. They are swollen, their protoplasm is undifferentiated and the nucleus is excentrically situated and degenerating. There are oftentimes large, ovoid swellings upon the cell processes. Ultimately the nerve cells disappear and are replaced by neuroglia. These changes are very wide-spread and are found in the retina as well as in the brain and cord. In many cases hardly a normal ganglion cell may be found.

To be differentiated from amaurotic family idiocy is a less frequent form of degeneration, known as "familial maculo-cerebral degeneration." It attacks several children in a family and at about the age of six or seven years. These children become dull, stupid, lose their power of attention and eventually their ability to read, speak or even recognize people. With these symptoms there is a central scotoma which may be of high degree but does not produce complete blindness. The physical condition of the child may remain normal for a long time. The eyes show a combination of atrophy of the retina with pigmentation especially in the region of the macula. The condition is incurable. It is progressive, though the patients may live many years. Death occurs from intercurrent infection rather than from the disease itself.

MENTAL DEFICIENCY

(*Idiocy—Imbecility*)

By mental deficiency is meant any interference with intelligence or a limitation in the adaptation of the child to his environment. This interference with intelligence may occur in children as the result of various general diseases or those confined to the nervous system. In other chapters the mental deficiency occurring secondary to general diseases and also to organic disease of the nervous system, such as hydrocephalus, chronic meningitis, paresis, meningeal hemorrhage, etc., is

discussed. The present chapter will treat only of mental deficiency as an apparently primary condition.

Of all the factors that operate to produce mental deficiency, heredity is the most important. This statement does not require substantiation. It is generally recognized. The descendants of mental defectives may be normal, they may be so defective that it is readily appreciable in the first year or two, or the disturbance of mentality may be so slight that it can be recognized only after several years of life. The influence of parental alcoholism, especially chronic alcoholism, has been much discussed and there is a wide difference of opinion in regard to it. Some claim that it plays a distinct part in the production of feeble-mindedness; some, that it plays little or none. It seems to us that it is a factor of some importance. While it cannot be entirely ignored, it certainly does not have the influence that has been ascribed to it by many. Whether syphilitic infection *per se* tends to produce mental deficiency is open to question. It does not appear likely that its influence can be great unless it produces organic changes in the meninges or in the brain itself or in the blood vessels. Poverty, poor surroundings, bad atmosphere, etc., have been claimed to have an influence by affecting the health of the mother. Associated with these are almost always other factors such as heredity and alcoholism that probably have much more effect upon the offspring.

The changes to be found in the brains of defectives are of all degrees of severity. (Fig. 114.) There may be an atrophy of one or more portions of the brain, failure of development of one hemisphere, poorly developed convolutions and shallow sulci. In certain cases no changes are to be made out macroscopically. The position can be well maintained, however, that even in such cases, mental deficiency is dependent upon actual organic changes in the brain, for practically all observers have found, as did Hammaberg, that even when no gross alteration was apparent the ganglion cells were infrequent and poorly developed.

There may be all grades of mental deficiency. It is usual in this country to separate mentally defective children into three groups: (1) the *idiots*, those that never develop beyond the mental age of an average child of two years; (2) the *imbeciles*, those that never acquire a higher degree of mentality than the average child of seven, and (3) the *morons*, who do not acquire a higher degree of mentality than children of twelve.

It is frequently necessary for the physician to determine whether or not a child is mentally deficient. In doing so it should be remembered that normal mental development is very dependent upon physical development; but it does not necessarily go on with equal rapidity. If an infant has been premature or badly nourished for many months or

has suffered from some very severe illness, he may at the end of a year show no more mental development than an average child of six or eight months. Yet, with improvement in his physical condition his mental condition also improves so that eventually the normal is reached. There is a wide variation in the rapidity of development of normal children. Some are quite slow, especially in certain families. Proper atten-

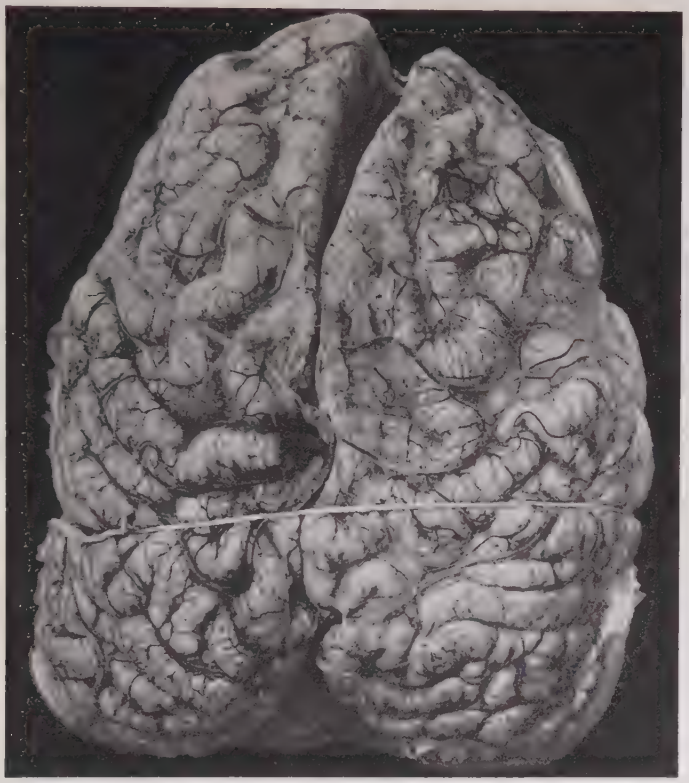


FIG. 114.—ARRESTED DEVELOPMENT OF THE FRONTAL LOBES OF THE BRAIN, PARTICULARLY OF THE RIGHT SIDE. From an idiotic child twelve months old.[1]

tion should be paid to this fact and too much emphasis should not be placed upon only slight deviations from the normal. The abnormal infant is distinguished not by slight, but by gross deviation from the normal. A high degree of mental deficiency can usually be recognized very early; the lesser degrees require longer observation. Even those children that are only slightly affected often give some definite evidence of it during infancy. Their mental development begins late and usually

[1] Microscopical Examination: Cortex in affected region one-third normal thickness; membranes and white substance normal; striking absence of characteristic nerve cells; very few large or small pyramidal cells present.

ends early. It is fair to assume that those whose mental development, in the absence of sufficient physical cause, is abnormally delayed, will suffer some permanent impairment of the mental faculties; but owing to the differences in the length of time that improvement may occur in different children, it is impossible to predict closely as to the final outcome.

To appreciate the abnormal, one must be familiar with the mental and physical development of normal children. Mental development shows itself in the early months of life chiefly by the acquisition of the ability to do certain physical things. The normal child about the third month begins to grasp objects—at the fourth month he recognizes people, between the third and fifth months he holds his head up firmly, at the fifth month he reaches for things, holds them in his hands and

Fig. 115.

Fig. 116.

Fig. 117.

Fig. 115.—Boy twelve years old; microcephalic; walked at about four years; can read and write; development like that of a normal child of eight years.

Fig. 116.—Microcephalic, seven years old; understands most of what is said; cannot talk intelligibly.

Fig. 117.—Girl of eight years; imbecile; cannot walk without help.

observes them. From seven to nine months, he sits alone, and laughs in play. From nine to ten months, many children stand. At a year they often begin to walk and to repeat single words. The mentally deficient child, on the other hand, may not even hold his head up at the end of a year. He makes no attempt to grasp objects, perhaps holds them for only a moment and then drops them. He cannot sit alone, he does not attempt to stand, and does not recognize people until perhaps the end of the second year or very much later.

Some mentally deficient children are exceedingly placid; others cry continually without apparent cause and are often exceedingly restless. The expression of the normal child is intelligent, bright and alert; the abnormal (Figs. 115, 116, 117) may betray his lack of mental capacity by his vacant, stupid expression, his open mouth, protruding tongue, drooling and his irregular, aimless movements of the hands. As time goes on, mentally deficient children not only remain backward in things that they should do, but they also do things that normal children do not

do. They develop screaming attacks, they throw their heads backward or frequently stiffen out. Strabismus is often present and there may be ill-defined attacks of a convulsive nature or typical convulsions.

It may be exceedingly difficult at times to differentiate between the merely backward child or the mentally deficient. The backward child is usually distinguished chiefly by the things which he does not do. He does not show an abnormal mentality. Children merely backward as the result of disease may not be able to talk until two years old or may not walk until after that time, yet may understand what is said and done for them; their expression is normal; they seem bright, and the development, although slow, is steady and progressive. Mentally deficient children, on the other hand, are not only very backward, but they usually reach the end of their development fairly early and it is not a complete development. As Scholz says, "the mentally deficient child of twelve is not a normal child of six; he is not merely a dwarf, but a cripple." This becomes increasingly evident as the defective child becomes older and his character and mental processes find better expression. He may be disobedient, unruly, untrustworthy, cruel to animals and playmates, not interested in the play of children, and may not conform to the ordinary standards of cleanliness and neatness. Most of the children are clumsy in their movements and especially not dextrous with their hands. There are many children, however, that are docile, kind and affectionate, but whose faculties are totally inadequate when compared with those of the average child. One with experience in testing mentally deficient children is able to tell with a considerable measure of accuracy what their mental capacity is. This is accomplished by observation and various tests, including the Binet-Simon test. This standardizing need not concern us here; but all physicians should be in a position to recognize the abnormal. The standardization of the abnormal and particularly the training should be in the hands of experts in that field.

MONGOLIAN IDIOCY

A form of mental deficiency that can be at once recognized by the physical characteristics of the child is the so-called Mongolian Idiocy, also known as "Kalmuck Idiocy." The cause of this is obscure. It cannot be shown that it is due in any way to syphilis or to the excessive use of alcohol in the parents. The condition appears with equal frequency in the sexes. It is found in the Caucasian race and we have seen several instances in the colored, but it has apparently not been reported among the Malay or Mongolian races. The factor of greatest importance is the age of the mother. The majority of Mongolian idiots are born to

women over 35 years of age. The number of pregnancies also appears to have an influence. These children are not infrequently the last after the birth of a number of healthy children. Much less frequently, they are the first, but the number of first or last children that are Mongols is greatly in excess of those in the middle of families. It is evident that the reproductive function has an important bearing upon their development. They are probably the result of incomplete or inhibited development, and have been called by Shuttleworth "exhaustion products."

This is one of the common forms of mental defect, apparently more frequent in England and in this country than elsewhere, perhaps on

Mongolian Types

Fig. 118.

Fig. 119.

Fig. 120.

Fig. 118.—Six months old; died at twenty-two months; could not hold up the head, or understand anything.

Fig. 119.—Boy, twenty-one months old; did not hold up his head until eighteen months; mental development that of a child of eleven or twelve months.

Fig. 120.—Girl four years old; mental development like that of a normal child of two and a half years; walks very awkwardly.

account of closer observation, as the result of the frequent attention that has been called to it.

Pathologically, the brains are, as a rule, small. The convolutions are poorly developed and there is apt to be an aplasia of some parts, such as the cerebellum, pons or medulla. The cortex is frequently thin and the ganglion cells few in number, with rather scanty cell processes.

The appearance of these children is very striking (Figs. 118, 119, 120) and it can at once be seen whence they have derived their name. There is a peculiar Mongolian type of countenance; the eyes are set closely together, they are slanting and the palpebral fissures narrow. There is frequently epicanthus. The head is brachycephalic and small. At twelve months it is often two inches below the average in circumference. The children are short for their age. Their hands are short and thick, especially the fingers; the little finger, not uncommonly, is so short that it does not reach to the last interphalangeal joint of the ring finger. The muscles are poorly developed, and there is a great relaxa-

tion of the ligaments, so that the strangest and most uncomfortable positions can be assumed at will and often by preference. The tongue is usually prominent, slightly protruding and deeply fissured. There is usually drooling from the mouth and often a nasal discharge, so that the lips may be greatly excoriated. Mouth-breathing is nearly always present. The nasopharynx is often small, sometimes owing to backward projection of the vomer, sometimes to a forward projection of the bodies of the cervical vertebrae. A very moderate amount of adenoid tissue may produce marked symptoms of nasal obstruction. The expression is often that of a child suffering from very large adenoid growths, and sometimes the early cases are passed over as simply "adenoids with mental dulness." Other defects are often associated. The ears are frequently misshapen; congenital malformations of the heart are quite common; in one of our cases there was absence of the patella.

Mongolian idiots are very backward in development. They frequently do not hold up their heads until a year of age, or later, and may not walk until the end of the second or third year. Speech is greatly delayed and seldom normal; although almost all, if they live sufficiently long, do eventually talk to a certain extent. These children have but little resistance to any acute disease. They are particularly susceptible to infection, and the majority die in infancy or early childhood. We see many of them as infants and few after the eighth or tenth year. They succumb chiefly to pulmonary infections or to tuberculosis. There is a certain degree of variation in their mental capacity, but it is singularly slight, and, as the majority of them look much alike, so also their mental processes are alike, and very few of them reach a higher mental development than that represented by a normal child of four or five years. They are restless, inattentive, and can be taught with great difficulty.

DEAF-MUTISM

Excluding the cases in which idiocy is present, which are not considered in this chapter, deaf-mutism may be due either to congenital or acquired conditions; the larger proportion of the cases belong in the latter class. When congenital, deaf-mutism may result from ostitis, or periostitis of the temporal bone, encroaching upon the cavity of the middle ear, from ankylosis of the ossicles, from absence of the internal ear or any of its parts. There may also be colloid degeneration of the labyrinth. It may result from atrophy of the auditory nerve, and it may be due to a lesion of the brain. These congenital conditions are often hereditary. An unusual form of congenital deafness is occasionally present with goiter. It is found especially in those regions in which

goiter is endemic. Its cause is unknown. Acquired deaf-mutism is most frequently the result of scarlet fever, and is due to otitis. The second important cause is cerebrospinal meningitis, where it may be due to a lesion of the brain, the auditory nerve, or the ear. It occasionally follows mumps, diphtheria, measles, and other infectious diseases. It may result from repeated attacks of acute otitis associated with adenoid growths or chronic rhinopharyngitis.

The younger the child at the time the deafness occurs the sooner the power of speech is lost. In most of the infectious diseases, if the attack occurs before the fifth year, speech is lost. According to Love, total deafness is rare among deaf-mutes; hearing for speech is present to a useful degree in about twenty-five per cent of the cases, while hearing by cranial conduction exists in nearly all cases. Deaf-mutism should be suspected if a child not idiotic shows at the end of two years no signs of beginning to talk. A careful distinction should be made between deaf-mutism and idiocy resulting either from congenital conditions or acquired disease.

It is necessary that this condition be recognized as early as possible, in order that the child may have the advantages of proper training during his early years. The physician should insist upon the child being sent as early as the third, and certainly by the fourth year to an institution where he may be taught to speak.

The treatment is mainly prophylactic. The most important relates to the care of the ears in scarlet fever, and the removal of adenoid vegetations of the pharynx and other causes which produce attacks of acute or chronic otitis. For the condition itself education is the only thing to be considered.

CHAPTER IV

DISEASES OF THE SPINAL CORD

MALFORMATIONS

Malformations of the cord are very frequently associated with those of the brain, and bear a certain degree of resemblance to them. (1) The cord may be absent (amyelia); this condition may exist alone or with absence of the brain. (2) The lack of development may be only partial (atelomyelia), as when some of the tracts are wanting. The most important one is defective development of the lateral tracts, which may be a cause of spastic paraplegia (Charcot). (3) There may be a malposition of some of the gray matter (heterotopia). (4) There may

be a double cord (diplomyelia); the division is generally incomplete, and is attributed to an abnormal development of the central canal; it is usually associated with other deformities. All of these malformations are extremely rare and of very little practical interest.

There remains to be mentioned the only one which is really important—spina bifida.

Spina Bifida.—This is a malformation of the vertebral canal with a protrusion of some part of its contents in the form of a fluid tumor. The tumor is elastic, compressible, usually increased by crying, and sometimes by pressure upon the anterior fontanel. The contained fluid is clear, resembling in all respects the cerebrospinal fluid. It is one of the most frequent congenital deformities.

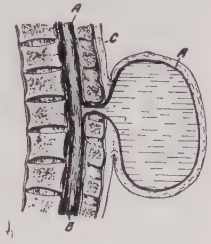


FIG. 121.—MENINGOCELE (partially diagrammatic). *A*, the membranes; *B*, the spinal cord; *C*, the integument. The accumulation of fluid is behind the cord, which does not enter the sac.

Spina bifida is due to an early failure in development—in most cases before the cord is segmentated from the epiblastic layer from which it is developed. Hence it remains adherent to the epiblastic covering, and the structures which should be formed between the cord and the skin are undeveloped. For this reason there is in the wall of the sac a fusion of the elements of the cord, nerves, meninges, vertebral arches, muscles, and integument. If the error in development occurs later, the cord and nerves may be attached to the sac, but not intimately fused with it; in still other cases the cord does not enter the sac at all. The malformation may occur before the central canal is closed; or, if closed, it may reopen from the accumulation of fluid. It is probable that the accumulation of fluid first occurs, and that this prevents the union of the parts of the vertebral arches.

Although the tumor is generally associated with a bifid spine, this is not necessarily the case. The protrusion may take place through the intervertebral notch or foramen, or there may be a fissure of the bodies of the vertebrae, and an anterior tumor projecting into the cavity of the thorax, abdomen, or pelvis; the tumor may be so small as not to be recognized externally—spina bifida occulta. The principal anatomical varieties are meningocele, meningomyelocele, and syringomyelocele.

Meningocele.—In this form there is a protrusion of the membranes only (Fig. 121). The accumulation of fluid is either in the arachnoid cavity or the subarachnoid space posterior to the cord. The opening of communication between the tumor and the spinal canal is small in this variety, usually being about one-twelfth to one-sixth of an inch in diameter. There may, however, be no communication. The skin is usually

fully developed (Fig. 122). The tumor is frequently globular, sometimes pedunculated, and may attain a very large size, being as much as five or six inches in diameter. This is because spontaneous rupture is not likely to occur, and the tumor does not become infected except by operative interference. With such tumors patients may live to adult life. This variety is most frequently seen in the cervical region. It has the best chance of natural recovery, and in it operation gives the best results.

FIG. 122.—MENINGOCELE, IN A CHILD ONE YEAR OLD.

Meningomyelocele.—This is by far the most frequent variety of spina bifida. It is the form usually seen in the sacrolumbar region. The accumulation of fluid takes place in the anterior subarachnoid space, less frequently in the anterior arachnoid cavity (Fig. 123). In this form the cord is contained in the sac, and usually forms a part of its wall. The tumor is smaller than the meningocele, the usual size being that of a mandarin orange. It is sessile, never pedunculated. As a rule it is only partly covered by skin, but has a central area, usually elliptical in shape, where there is only a thin, translucent membrane. This surface, which is known as the central cicatrix, is sometimes covered with granulations, and frequently ulcerates. The tumor often has a vertical furrow or a central umbilication, corresponding to the attachment of the cord on its inner surface. The usual relation of the parts is for the cord to run horizontally across the upper part of the tumor to the central cicatrix, with which it becomes blended, and from which again the nerves arise. These re-enter the canal at the lower part of the tumor, and are distributed below as usual. In other cases the cord joins the wall of the sac soon after its entrance, and its attenuated

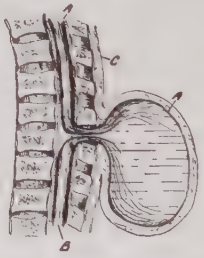


FIG. 123.—MENINGOMYELOCELE (partially diagrammatic). *A*, the membranes; *B*, the cord; *C*, the integument. The accumulation of fluid is in front of the cord, the filaments of which are spread out, forming a part of the wall of the sac.

fibers are found spread out all over the sac, coming together again below and entering the spinal canal.

The following case, upon which an autopsy was made, is a good example of the common variety: The child died on the third day after birth from rupture of the sac. The tumor occupied the sacral region. The first sacral vertebra was normal, and beneath this the cord passed out of the spinal canal, terminating in the cauda equina soon after entering the sac, and continued back to the central cicatrix. Here nerve filaments blended with the other tissues in an indefinite structure, from which again, with tolerable distinctness, the nerve structures could be seen to pass over the wall of the sac and return to the canal. The afferent and efferent nerves and the part of the membranes they carried with them formed several septa, making a smaller separate sac within the larger one. The large sac was clearly a dilatation of the anterior subarachnoid space, and communicated freely with the same space in the cord above.

Syringomyelocele.—In this variety the accumulation of fluid is in the central canal of the cord, the lining of the sac being here the attenuated and atrophied cord elements. This is the rarest form of tumor, but the one most frequently associated with hydrocephalus, and consequently has the worst prognosis. It may be found in the dorsal or dorsolumbar region as well as in the lumbosacral.

With spina bifida other deformities are frequently associated, the most common being club-foot, hydrocephalus, more rarely encephalocele or cerebral meningocele, and hare-lip. If hydrocephalus exists, there is in most cases a dilatation of the central canal of the cord and a direct communication between the tumor and the lateral ventricles of the brain. Pressure upon the anterior fontanel causes an increase in the size of the tumor, and conversely. Club-foot is usually double, most frequently talipes equinovarus. In a number of cases there is a history of some deformity in other members of the family. We have seen two successive children in the same family with spina bifida.

Symptoms.—The tumor in spina bifida is present at birth, and is most frequently lumbosacral. Paralysis is frequent in myelocele and syringomyelocele, but is not seen in meningocele; its degree and its location depend upon the situation of the tumor and the extent to which the cord is involved. It is rare in cervical tumors, and most marked in those situated in the lumbosacral region. In the worst cases there is complete paraplegia, with paralysis of the bladder and rectum. If the tumor is sacrolumbar or sacral, only the cauda equina is likely to be involved, and this but partially, so that the paralysis of the extremities is incomplete, and the bladder and rectum may escape. Spina bifida occulta is in rare instances the explanation of obsti-

nate incontinence of urine and sometimes of feces also. It may occur without paraplegia.

In Fig. 124 is shown a very remarkable case of sacral spina bifida in a boy of five years, who came under observation for incontinence of feces. The tumor was a little more to the left than to the right side, and had been overlooked. It had evidently pressed upon the lower branches of the sacral plexus, so as to affect the sphincter and the gluteal muscles of the left side. The atrophy was very marked, as shown in the illustration.

Fig. 124.—Sacral Spina Bifida.

The natural course of spina bifida is to increase steadily in size; and if the tumor is covered by skin, its growth may be almost unlimited. It has been known to attain a circumference of twenty-two inches. If the integument is wanting, and the sac wall is very thin, rupture is pretty certain to take place, either spontaneously or by some accident, in the course of the first few months; usually death then results from convulsions owing to the rapid draining away of the cerebrospinal fluid, or from infection. In a large number of cases death is due to marasmus dependent upon the associated conditions. Infection of the tumor may take place without rupture, the germs passing through the wall of the sac. If the opening communicating with the spinal canal is small, this infection may excite an inflammation limited to the wall of the sac, and result in a cure of the spina bifida, usually with sloughing. We have seen such a case in a girl ten years old in whom this occurred in infancy. The site of the former tumor was marked by a large dense cicatrix, and there still remained partial paralysis of the legs. If the opening into the spinal canal is large, inflammation of the sac is usually followed by spinal meningitis, which may extend upward and involve also the meninges of the brain.

Prognosis.—This depends chiefly upon the anatomical variety and the existence of complications. Simple meningocele, when covered by integument, gives the best prognosis, and complete recovery may occur. In meningomyelocele, especially if complete paralysis exists, the prog-

nosis is bad; and if there is also hydrocephalus, the case is hopeless. In many cases in which cure of the spina bifida has followed operation, hydrocephalus has subsequently developed. Of fifty-seven cases reported by Demme, twenty-five were operated upon, with seven recoveries and fifteen deaths, while three were unimproved; of the thirty-two cases not operated upon, twenty-eight died within the first month, and not one lived over two years—the causes of death being marasmus, rupture of the sac, and meningitis.

Diagnosis.—It is usually easy to recognize spina bifida, but it is often difficult to distinguish between the different varieties. The absence of a palpable fissure in the spine, perfect translucency, and a pedunculated tumor, all point strongly to meningocele. Paralysis of the sphincters and lower extremities, umbilication of the center of the tumor, a sessile tumor, a palpable bony fissure, and a large central cicatrix, point to meningomyelocele. The coexistence of hydrocephalus points to syringomyelocele.

Treatment.—In all cases the tumor should be protected from pressure, and when it is not covered by integument, care taken that the surface is kept absolutely clean and aseptic. It should be covered with some antiseptic powder and surrounded by a large pad of absorbent cotton, or a rubber ring-cushion. Complete paraplegia with involvement of the bladder and rectum, hydrocephalus, or extreme marasmus—all contraindicate operative interference. If these are absent, operation should be considered. The time of operation will depend somewhat upon the nature of the tumor. If it is covered by integument and growing slowly, it is well to wait until the child is at least six months old. In other cases delay is dangerous, because of the liability to spontaneous or accidental rupture.

The treatment by injection has now been entirely superseded by the operation of excision of the sac. For a description of this and the various plastic operations that have been proposed in connection with it the reader is referred to works upon operative surgery. In operating, it should not be forgotten that in the great proportion of the cases (ninety-five per cent, according to the London Clinical Society's Report, which, however, refers only to fatal cases) some part of the cord is in the sac. The cord is often present in tumors situated below the third lumbar vertebra, owing to its attachment to the sac.

Although recovery may follow operation, in a very large number of cases it is incomplete; some degree of paralysis, with atrophy, contractures and deformities, remaining because of the implication of cord elements in the sac. In a considerable proportion of cases, hydrocephalus subsequently develops, as after similar operations upon cerebral meningocele.

SPINAL MENINGITIS

In acute meningitis usually only the pia mater is involved. This rarely occurs alone, unless it is due to traumatism. It is most frequently associated with inflammation of the pia of the brain, and may occur either with the meningococcus or the tuberculous variety. A certain amount of acute inflammation of the pia mater accompanies most of the cases of acute myelitis.

Chronic spinal meningitis in children usually involves the dura only. Inflammation of the external layer (external pachymeningitis) is usually secondary to caries of the vertebrae. This is considered in the article on Compression-Myelitis.

Symptoms.—The symptoms of inflammation of the spinal membranes, no matter with what pathological condition it may be associated, are due to irritation of, or pressure upon, the cord or nerve roots. Those which are most common are: pain in the back, which is increased by movement, and usually by pressure upon the spinous processes; radiating pains following the course of the spinal nerves, felt in the extremities or in the trunk; rigidity of the spinal column due to spasm of the spinal muscles, or rigidity of the muscles of the extremities; and hyperesthesia along the spine, which may be quite acute. When pressure upon the cord is added, there is paralysis or paresis, sometimes muscular atrophy and anesthesia. Any of the above symptoms may be acute or chronic, according to the nature of the primary disease.

The diagnosis between spinal meningitis and myelitis is often not easy, for except in acute cases the two processes are usually associated; and in a given case it may be difficult to decide whether the lesion of the cord or of the membranes is the more important one. In meningitis, pain, tenderness, spasm, and irritative symptoms are generally more prominent, while loss of power and anesthesia are usually partial. In myelitis the pain, tenderness, and other irritative symptoms are less marked, while paralysis and anesthesia may be complete.

Treatment.—This relates first to the disease with which it is associated; in addition, counter-irritation by means of the Paquelin cautery, rest in bed, and in severe cases even immobilization of the spine by a mechanical support. Iodid of potassium is often useful.

MYELITIS

Myelitis is a rare disease in children, with the exception of two varieties which are discussed under separate heads, viz., compression-myelitis

and acute poliomyelitis. Otherwise myelitis usually results from injury, but it may occur as a complication of any of the acute infectious diseases, especially typhoid, scarlet fever, and diphtheria, and even as a primary disease, when it is attributed to exposure or cold, but when it is probably of infectious origin. We have seen it follow varicella. Chronic myelitis may be due to hereditary syphilis.

Myelitis usually occurs in children over ten years of age. In situation, it may be transverse, diffuse, or disseminated; the process may be acute, subacute, or chronic. The lesions and the symptoms are essentially the same as when the disease occurs in the adult.

Symptoms.—Myelitis usually comes on rather gradually, with only local symptoms; but the onset may be quite acute, with several general symptoms—fever, pain, prostration, and localized or general convulsions. The local symptoms vary with the seat and the extent of the disease.

In transverse myelitis loss of power and anesthesia are present below the level of the lesion; either of these may be partial or complete. At the level of the lesion there is a zone of hyperesthesia and "girdle-pains." All the reflexes below the seat of the lesion are exaggerated. Those at the level of the lesion are lost. There may be loss of control of the sphincters, bed-sores, degenerative changes in the paralyzed muscles, contractures, and vasomotor disturbances. The paralyzed muscles may be rigid or flaccid, according to the seat and extent of the lesion.

When transverse myelitis is situated in the cervical region there is paralysis and anesthesia of the arms, legs, and trunk. All the reflexes are exaggerated, and there is general rigidity of the paralyzed muscles. There is incontinence of feces and retention of urine, followed by incontinence from overflow. The pupils are frequently contracted, and there may be optic neuritis. Atrophy, when present, usually affects the muscles of the arms, and indicates that the cord to a considerable extent is involved. There is great danger to life, owing to paralysis of the muscles of respiration.

When the seat of disease is the dorsal region, the symptoms are similar to those above described, with the exception that the arms escape, and that the ocular symptoms are usually wanting. This is the most favorable seat of the disease.

When the disease is situated in the lumbar region, in addition to paraplegia and anesthesia of the legs, there is, from the beginning, incontinence of urine and feces. The knee reflexes are lost; the muscles atrophy, and usually give the reaction of degeneration. Bed-sores are frequent.

In diffuse myelitis the symptoms are a combination of the above groups. If a large part of the cord is involved, there are general paral-

ysis and anesthesia, loss of reflexes, marked trophic disturbances, bed-sores, etc.

The course of myelitis is slow, and it usually progresses steadily from bad to worse. Death is due to exhaustion or complications—cystitis, bed-sores, or hypostatic pneumonia—or to some intercurrent disease. In a small proportion of the cases there may be partial recovery, but very rarely is recovery complete. The diagnosis is to be made from spinal meningitis, tumors, and hemorrhage.

Treatment.—The treatment of the early stage consists in the use of ice to the spine, or counter-irritation by means of dry cups or mustard. Later, the iodid of potassium should be given in full doses; improvement may follow its use, even when there is no suspicion of syphilis. Electricity is contraindicated except in chronic cases, and then but little improvement is likely to result from its use. In these patients the most important thing is careful attention to cleanliness and to posture, in order to prevent bed-sores, cystitis, and pneumonia.

COMPRESSION-MYELITIS

(Pressure-paralysis of the Spinal Cord; Pott's Paraplegia)

Compression-myelitis is sometimes traumatic, but usually follows caries of the spine. It most frequently complicates this disease when the cervical or upper dorsal vertebrae are involved, rarely when the lower half of the spinal column is affected. This difference is probably due to the smaller size of the spinal canal in its upper portion. According to Gibney, paraplegia is seen in fifty per cent of the cases of caries of the upper half of the spine. Essentially the same condition, so far as the cord is concerned, may result from tumors of the spinal cord, or from anything else causing pachymeningitis. These, however, are exceedingly rare in childhood.

Lesions.—In spinal caries there occurs as a result of tuberculous disease a softening of the bodies of the vertebrae, which fall together from the pressure due to the superincumbent weight of the body. This causes a backward projection known as the kyphosis, or angular deformity. The spinal canal is encroached upon by the remains of the vertebral bodies whose ligamentous attachments have been loosened, and also by inflammatory products, the result of periostitis, and localized inflammation of the dura mater, chiefly of the external layer, but which sometimes affects the internal layer also. All these conditions lead to the production of a mass of inflammatory material, often containing tuberculous deposits, which is chiefly in front of the cord, but may surround it. The compres-

sion takes place slowly in most of the cases, from the gradual progress of the lesions mentioned. In a small number of cases there may be a sudden pressure from the slipping backward of one of the vertebral bodies.

In recent cases the cord at the seat of compression is a little smaller than normal. It is usually involved to the extent of from half an inch to two inches. Paraplegia may have existed when the changes found in the cord are very slight, and sometimes when no changes are visible to the naked eye. In more protracted and more severe cases, the cord is much smaller at the point of disease, and under the microscope shows the changes of interstitial myelitis (Gowers) with meningitis. In old cases there is degeneration of the nerve elements, atrophy, and sometimes disappearance of the ganglion cells, with more or less destruction of the nerve fibers; sometimes all distinction between the gray and white substance is lost. In addition to these marked changes at the point of pressure, there may be ascending or descending degeneration, as from other focal lesions. There is usually inflammation of the nerve roots, which have also suffered compression. It is in many cases surprising to see to what degree the cord may be compressed and still preserve its functions.

Symptoms.—In caries of the cervical region the symptoms of compression-myelitis not infrequently precede the deformity, and, in fact, the other objective symptoms of bone disease. The earliest symptoms of caries usually arise from irritation of the nerve roots, and consist of acute pains often not referred to the spine, but radiating to the different regions to which these nerves are distributed. They are felt in the neck, in the chest, in the epigastrium, and sometimes in the loins. Accompanying these pains, there is noticed a gradual weakness in the lower extremities, and sometimes also in the arms, according to the location of the disease. This may steadily increase for several weeks until there is complete paralysis. Other symptoms are then commonly present. There is usually some degree of anesthesia, and there may be numbness, tingling, formication, and pain. The sphincters are not often involved. When the disease is in the upper half of the cord, there is rigidity of the extremities and great exaggeration of all the reflexes, with marked ankle-clonus. In the rare cases in which the lumbar enlargement is involved, there may be loss of reflexes, paralysis of the sphincters, and bedsores.

The distribution of the paralysis will depend upon the point of compression. If this is in the cervical region, all four extremities will be paralyzed; if in the dorsal region, only the legs. According to the extent of the secondary lesions in the cord, there may occur muscular atrophy and contractures. With disease in the upper cervical region, death may

result from sudden pressure upon the cord, owing to a dislocation of the odontoid process; or there may be vomiting, pupillary symptoms, irritation of the phrenic nerve causing hiccough, or pressure causing paralysis of the diaphragm.

Course and Prognosis.—These depend much upon the treatment of the case. In many cases of paralysis occurring early in caries, complete recovery takes place in the course of a few weeks, sometimes in a few days, after the application of a proper mechanical support. In the cases which have been long neglected, or those in which the paralysis develops while proper mechanical treatment is being carried out, the chances are not so good. Gibney gives the following statistics of 133 cases under his personal observation: 31 proved fatal; 9 dying from myelitis, 14 from other diseases subsequent to recovery from the paralysis, and 6 from tuberculosis before complete recovery; 74 recovered from the paraplegia; 27 were recorded as improved or still under treatment. Relapses occurred in about fifteen per cent of the cases. The usual duration of the paralysis is from three months to two years. Recovery has often taken place in cases that have persisted for four or five years.

Diagnosis.—This is rarely difficult. Spinal caries should be suspected in every case when the symptoms point to transverse myelitis coming on without definite cause.

Treatment.—The indications are the removal of pressure and the fixation of the spine by a proper mechanical support. From his very extensive experience, Gibney has more confidence in the iodid of potassium than in all else except mechanical treatment. Large doses are required, often from sixty to ninety grains being given daily for months. The iodid should always be largely diluted. Patients should be kept scrupulously clean, and the position changed frequently to prevent the formation of bed-sores. Electricity is contraindicated. When the paralysis develops rapidly or occurs suddenly, relief may sometimes be obtained by the operation of laminectomy; but little is to be expected from this in the slow cases.

ACUTE POLIOMYELITIS

(*Epidemic Poliomyelitis; Acute Infantile Paralysis*)

There are few diseases regarding which our knowledge has increased so rapidly during recent years as acute poliomyelitis. The first great step in advance was made by Landsteiner and Popper, who, in the summer of 1909, succeeded in producing the disease in a monkey by intraperitoneal inoculation with the spinal cord of a patient dying of acute

poliomyelitis. They were not successful in carrying the transmission further. Shortly after this Flexner and Lewis, using the intracranial method of inoculation, had no difficulty in reproducing the disease and transmitting it through an indefinite series of monkeys. No other animal seems to be susceptible. These observations, now many times repeated, have not only definitely established the infectious character of poliomyelitis, but have cleared up many doubtful points in its pathology.

Acute poliomyelitis is now regarded as a communicable, infectious disease which prevails both epidemically and sporadically. Although possibly its most characteristic lesions are in the anterior horns of the cord, any part of the central nervous system may be affected. The changes in the cord substance are preceded by lesions of the meninges. Although the name poliomyelitis is still retained, the scope of the term has been greatly widened.

This disease is characterized by an acute onset, with fever and usually other marked constitutional and nervous symptoms, from which there may be rapid recovery; but generally there follows early and extensive loss of power. After this there is usually seen a gradual improvement, and sometimes complete recovery. More often, however, there is left some permanent paralysis in certain groups of muscles, which undergo rapid and marked atrophy. Formerly, poliomyelitis was seen chiefly as a sporadic disease; but since the year 1905 epidemics have occurred with increasing frequency in various parts of the world, and especially in the United States since 1907. As it is most frequently seen in very young children, and as it is altogether the most common form of paralysis at this period, the old term "acute infantile paralysis" is perhaps the most appropriate clinical designation.

Etiology.—Fully eighty per cent of the cases are seen in the first four years of life, the greatest incidence being in the second year. No age is exempt and in some epidemics the proportion of adult cases is quite large. Epidemics thus far observed have invariably occurred in the warm months; those in the United States, from July to October. Fully four-fifths of the sporadic cases also are seen during these same months.

The prevalence of poliomyelitis in an epidemic form began with the outbreaks in Sweden and Norway in 1905 and 1906. These were followed in 1907 by the epidemic occurring in New York City and vicinity in which there were observed nearly 3,000 cases. After that poliomyelitis gradually spread over the country, epidemics occurring during the next four years in nearly all parts of the United States. Large outbreaks were also reported in other parts of the world. The most extensive epidemic known was that of New York in 1916 in which over 4,000 cases were reported in a single month.

The simultaneous or successive occurrence of several cases in the same

family has long suggested that the disease was directly communicable. This has now been established by experimental evidence and is corroborated by clinical observations. The disease may be communicated: (1) by the typical acute paralytic cases; (2) by mild, ambulant or abortive cases; (3) by healthy carriers, i. e., persons who have been in close contact with one suffering from an acute attack; (4) by chronic carriers or those who have recovered from acute attacks. How long persons of the last two groups may convey the disease is not known. The virus has, however, been demonstrated on the mucous membranes of the mouth and nose after several months have passed. The disease, in most circumstances, is feebly contagious, and only a small proportion of those exposed contract it. As in the case of cerebrospinal meningitis, it is much more contagious when prevailing epidemically. The transmission by healthy carriers, though very exceptional, is undoubtedly the explanation of the occurrence of some of the widely separated cases seen in a community; others of obscure origin may be traced to abortive cases. That the virus of poliomyelitis is carried by insects has not been established. At present we know of no other way of acquiring the disease than by contact with affected persons or with those who serve as carriers.

The period of incubation of the experimental disease in monkeys varies from four to thirty-three days, the average being nine or ten days. In man, also, it is variable, but in most instances the second case in a family has followed the first one within ten days.

The specific organism of this disease belongs to the class of filtrable viruses. It passes through the pores of the finest porcelain filter. It has been isolated and cultivated outside the body by Flexner and Noguchi, and with a high-power microscope it can be seen as very minute globular bodies. It is present in largest quantity in the diseased nerve structures, particularly the spinal cord. In the earliest stages of the attack it is also found in the cerebrospinal fluid, but disappears at about the time paralysis occurs. It exists to some extent in other tissues of the body, particularly the lymph nodes. The disease can be transmitted to animals regularly and with certainty only by inoculation with an affected spinal cord, in which the virus persists for months after the acute attack. Experiments and clinical evidence indicate that the usual path of entrance is the nasal mucous membrane. Osgood and Lucas have shown that the virus persisted in the nasal mucous membrane of monkeys, in one instance for five months, in another for one and a half months, after the acute attack; which suggests that this may not only be an avenue of entrance, but possibly a mode of elimination of the infection, and indicates that the duration of the infective period may at times be a very long one.

Lesions.—As a result of the investigations, particularly of Flexner

and Lewis upon animals, and those of Harbitz and Scheel, Strauss, and others upon the disease in man, the pathology of acute poliomyelitis is now well known. This knowledge has greatly aided our clinical understanding of the disease.

The lesions found in this disease show in the cases severe enough to be fatal the effects of a widespread generalized infection. Not only are the nervous tissues involved, but also the parenchymatous organs and lymphoid structures. In the nervous system the virus first attacks the meninges, especially of the cord and medulla, setting up a cellular inflammation of the pia, which becomes infiltrated with small, round cells. These changes are most marked about the blood-vessels. Besides this the walls of the vessels themselves are infiltrated and their lumen narrowed. The lesion also affects the vessels entering the nerve structures. As a result of the vascular lesions anemia, edema, and hemorrhages are present, sometimes small and circumscribed, sometimes quite diffuse and extensive. Thrombosis seldom occurs. But more important still are the secondary degenerative changes in the nerve cells, the site and extent of which are determined by the vessels involved and the intensity of the changes in them. The lesions in the pons, medulla, and cerebrum, like those in the cord, are secondary to the vascular lesions.

The transient paralysis in cases that recover may be due to edema or to temporary vascular obstruction from pressure outside the vessels causing a local anemia. Permanent paralysis depends upon severe degeneration and actual destruction of ganglion cells; its extent, therefore, will vary with the number of the ganglion cells affected. Any part of the central nervous system may be affected, and the lesions are generally more extensive than the symptoms would lead one to expect. The gross appearances give but little idea of their severity. The process often involves nearly the whole length of the cord, being, however, generally most marked in the cervical and lumbar enlargements. The changes are chiefly in the gray matter of the anterior horns, and consist in acute degeneration of ganglion cells, usually marked and extensive. These cells in certain parts may disappear altogether, being replaced by leucocytes. The entire cord, however, may be involved. There is seen, but to a much less degree, infiltration with small round cells of the posterior horns, the columns of Clarke, and the white matter of the cord, everywhere closely related to the blood-vessels. There are regularly found changes in the spinal ganglia of a similar character to those described in the cord.

Lesions like those of the cord, though generally less marked, are seen in the pons, the medulla, the cerebellum, and even in the cerebral hemispheres. They are, as in the cord, especially related to the pia and the blood-vessels. There is seen acute destruction of ganglion cells

and areas of cell infiltration with lymphocytes. The changes are especially marked about the nuclei of the cranial nerves, and in the gray matter about the fourth ventricle. In some cases the basal ganglia are also involved. Areas of infiltration, sometimes quite diffuse, may be seen in the cortex, with also some slight degeneration of ganglion cells.

Thus, in the severe and fatal cases there is present a diffuse inflammation of the entire cord and its membranes, also of the medulla, pons, and basal ganglia, with less marked changes in the cerebrum, always accompanied by changes in the pia. In the milder cases it is probable that the inflammatory changes are limited to the cord, though in some patients dying later from other causes Harbitz and Scheel discovered changes in the upper centers, though no symptoms pointing to them had been present. From this account of the lesions it would appear that we can no longer distinguish between the lesions of acute poliomyelitis, acute bulbar paralysis and acute poliencephalitis inferior. They represent varying phases of one and the same disease. In recent acute cases no changes are usually found in the nerves except degeneration of bundles, corresponding to the degenerated areas in the cord, and probably secondary to them. Lesions in other organs are often present, the most frequent being bronchopneumonia and acute parenchymatous degeneration of the liver and kidneys, similar to what is seen in other severe general infections. The thymus, the solitary follicles of the intestine, and the mesenteric glands may be much swollen.

In autopsies made upon cases of long standing the affected part of the cord, which is often only one lateral half, is smaller than normal. The general changes are those of a sclerotic character. The ganglion cells of the affected anterior horn have either disappeared altogether, or they are few in number and so shrunken as to be hardly recognizable. The white matter also is smaller than in the sound part of the cord. The anterior nerve roots are degenerated quite to the muscles. The affected muscles are atrophied, and in extreme cases there may be a complete disappearance of muscle fibers, their place being taken by adipose and fibrous tissue. In places where the lesion is less severe the fibers are small. The affected limb is shorter and the bones smaller than upon the sound side.

Symptoms.—Cases of acute poliomyelitis present a wide variety of clinical symptoms depending upon the virulence of the infection, the age of the person attacked, but principally upon the part of the nervous system chiefly involved in the pathological process. They may be broadly divided into four general groups: (1) the cerebral cases; (2) the spinal; (3) the bulbospinal; (4) the non-paralytic or so-called abortive cases.

The Cerebral Type.—It is only very recently that acute poliencepha-

litis, fully described by Strümpell in 1885, has been regarded as a manifestation of this disease. Although some experimental evidence is still wanting, the identity of the histological changes and its association with acute poliomyelitis in epidemics leave little doubt that acute polioencephalitis is often only the cerebral form of acute poliomyelitis. Such cases are not to be confounded with the common forms of acute poliomyelitis with cerebral symptoms.

The onset is generally abrupt, with convulsions which are often repeated over a period of a day or two. There is usually vomiting and fever, which may be high. The paralysis which follows after one to three days of general symptoms is usually of the hemiplegic type, the face, arm and leg being involved. The reflexes are increased and the paralysis is of the spastic type. The face improves and usually recovers completely; the leg, next; while the arm is generally most affected and the paralysis is likely to be permanent and be followed by contractures. In its late results it resembles other forms of acute cerebral paralysis in early life.

The Spinal Type.—This group includes the most characteristic form of the disease and is numerically the largest. In the cases of moderate severity, the onset is abrupt and the symptoms may differ little from those seen in other acute infections. There is usually vomiting, which is not repeated, more frequently constipation than diarrhea, and fever which is generally not over 103° F. Drowsiness, irritability, headache and prostration are seen in most cases. After the first day more definite symptoms, indicating involvement of the nervous system, are present—general hyperesthesia, shooting pains in the legs, stiffness of the neck or extremities, pain on motion, etc. The blood shows a moderate polymorphonuclear leucocytosis and the cerebrospinal fluid is generally clear, but may be slightly opalescent. It shows a greatly increased number of cells, which at first may be chiefly polymorphonuclear, but very soon are nearly all lymphocytes. There is an increase of globulin. After the febrile symptoms have lasted for from twenty-four hours to three days, the paralysis is seen. Exceptionally, the early stage is very short, and the paralysis is noticed almost at the onset. In the lighter cases, the fever may not be over 100° or 101° F., and may last only a day, with all the general and local symptoms correspondingly mild, though the resulting paralysis may be extensive.

In the paralytic stage the loss of power sometimes comes on quickly in a few hours; but more often, rather gradually, and extends for from two to three days before it is fully developed. The other nervous symptoms usually continue. The posture is in most cases dorsal, with limbs semi-flexed, but in some cases with marked meningeal irritation there may be a general flexion of the body with opisthotonus, exactly as in

cerebrospinal meningitis. The same rigidity of the neck and extremities may also be seen. The knee jerks are not uniform, at first may be increased, but are soon lost on the paralyzed side and sometimes also on the sound side. Pain is present on motion, on pressure over nerve trunks and sometimes complained of when the patient is quiet. Retention of urine may be so complete as to require the use of the catheter, but in most cases the child is able to void, however, with considerable difficulty. The bowels in most of the cases are constipated. The mind is usually clear, though the child is very sensitive to handling, and there may be general hyperesthesia. The duration of the fever is on the average three or four days; it is rare for it to continue longer than a week. The temperature range is generally between 101° and 103°, and the fall to normal is gradual. Usually the height of the temperature is in proportion to the severity of the infection, but it does not measure the danger of the attack, which depends rather upon what part of the nervous system is involved most seriously.

The description above given is that of the type most frequently met with, but many other forms of the disease are seen which add much to the difficulty of diagnosis. Certain cases present marked cerebral symptoms, chiefly stupor, with very few spinal symptoms. After the usual onset, the drowsiness soon develops into deep stupor, which may last for a week or more. These symptoms, with the continuance of the fever, the stiffness of the neck and irregularity of the knee jerks, form a picture which is almost indistinguishable from tuberculous meningitis. These cases belong to quite a different group from those described above as the cerebral type. The paralysis, when it occurs, indicates an involvement of the cord at a high level and affects, besides other parts, one or both arms. Though the symptoms in such cases are most disturbing, the cerebral condition often clears up rapidly and completely.

Other types which may be seen in epidemics are: (1) those in which the symptoms of meningeal irritation are especially marked, extreme muscular and nervous irritability, hyperesthesia, rigidity, etc., a group of symptoms strongly suggesting cerebrospinal meningitis; (2) cases in which, with many of the above symptoms, pain is especially prominent; (3) cases in which gastro-intestinal symptoms are particularly marked; both vomiting and diarrhea may last for several days and their prominence may obscure the nervous symptoms.

The Bulbospinal Type.—The onset and general symptoms differ in no way from the severe cases of the spinal type. It is only after paralysis develops that the characteristic symptoms are seen. This group forms, according to Wickman, about 6 per cent of the epidemic cases. The lesions of the bulb are generally more extensive than one would expect from the symptoms. The symptoms of bulbar paralysis are nearly

always limited to one side, whether they occur alone or with paralysis of the arms and legs. Almost any of the cranial nerves may be involved, altogether the most frequent being the facial. The whole nerve is not always affected. The facial paralysis is usually transient, but may be permanent. Ocular paralyses are next in frequency, the external rectus being oftenest affected. Disturbances of speech are not infrequent, but rarely persist. They are often associated with disturbances of deglutition, which, while not common, may be so severe as to necessitate feeding through a tube. With these bulbar symptoms are often associated others, indicating involvement of the upper part of the cord, such as paralysis of the diaphragm, the intercostals, the neck, or the upper extremities. These cases form the most severe and fatal type of acute poliomyelitis met with, and it is the type that furnishes most of the deaths. The fatal result is nearly always from respiratory paralysis or bronchopneumonia.

Acute bulbar paralysis with lesions limited to this part, though formerly described as a separate and distinct disease, is probably only a form of acute poliomyelitis.

Another rare clinical type is an acute ascending paralysis with symptoms described as Landry's disease. After the usual onset, paralysis affects first the legs, then the arms, the neck and finally the diaphragm and intercostals, with death from respiratory paralysis. This extension of the paralysis usually occupies three or four days, though it is sometimes very rapid, and death may take place on the second or third day from the beginning of the attack.

Extent and Distribution of the Paralysis.—Wickman gives the following grouping of 868 epidemic cases in 1905:

One or both legs	353;	one or both arms	75
Combinations of arms and legs	152;	legs and trunk	85
Arms and trunk	10;	trunk alone	9
Ascending paralysis	32;	descending paralysis	13
Spinal and cranial nerves	34;	cranial nerves alone	22
Whole body	23;	not given	60

A comparison between this and 560 sporadic cases we have collected from various authors is interesting:

One leg	229;	both legs	176
Combinations of arms and legs	42;	one arm only	14
All extremities and trunk	79;	all others	10

In both series the large proportion of cases in which the legs are involved is striking; also the infrequency with which the arms alone are affected, and finally that in the epidemic cases there is a much larger

number with widespread paralysis and with cranial nerve involvement. The latter, when occurring sporadically, are generally classed under some other heading than acute poliomyelitis.

The paralysis, when limited to the leg, most frequently affects the anterior tibial group; next, the peroneal, and third, the quadriceps extensor femoris. The paralysis of the upper extremities most often involves the shoulder group, the deltoid being the muscle which usually suffers most severely. Paralysis of the sphincters is very rare, though bladder disturbances are quite common.

The most serious paralysis is that of the diaphragm and the intercostals; either may be involved alone and the patient recover, but when both are affected death follows. Diaphragmatic paralysis occurs when the lesion affects the third to the fifth cervical segments of the cord. It seldom occurs early and may develop quite late in the disease. Though this is always a serious symptom, it may last several days and yet recovery take place. When the diaphragm is paralyzed, all the accessory muscles of respiration are called into action; the respiration is wholly thoracic and the abdominal wall, instead of protruding, is retracted on inspiration.

Paralysis of the intercostals is rare, except in very severe cases, and is usually, but not invariably, fatal. It is seen in association with widespread paralysis of arms and legs, and in the rapidly spreading cases of ascending paralysis, and in the most severe infections. The respiration in intercostal paralysis is purely diaphragmatic, which is not always easy to recognize, as it is an exaggeration of the normal infantile type. When both intercostals and diaphragm are involved, we see one of the most distressing conditions seen in the disease, i.e., death by respiratory paralysis. A remarkably vivid picture of this is given in the monograph of Peabody, Draper and Dochez. The mind is usually clear, alert and full of apprehension. Every breath drawn is with severe effort. Sweating is profuse. Cyanosis is usually absent. The struggle may last for several hours before death takes place. Although life may sometimes be prolonged for a considerable time by artificial respiration, there is practically no hope of recovery.

Paralysis of the abdominal muscles is not common, is usually of one side, but may affect both. It is evident by a great bulging or "ballooning" of part of the abdominal wall, in coughing, sneezing, or any forced expiration. It may remain as a permanent paralysis.

Course of the Disease.—In those who survive the acute stage, there is a period of a few weeks' duration in which little change is seen. This is followed by spontaneous improvement, which usually begins in the muscles last affected, and reaches its limit in from three to six months. The paralysis remaining after this time is likely to be permanent. By

the end of six or eight weeks atrophy is present in the paralyzed muscles. The affected limb is distinctly smaller than its fellow, this being quite apparent even in infants. Except in the early stage, sensory disturbances are absent; the knee-jerk is lost in paraplegic cases, and in those in which the extensors of the thigh are paralyzed. There is arrested growth in the whole limb (Fig. 125). It becomes much smaller and shorter than its fellow. From paralysis of the shoulder and thoracic muscles various chest deformities may result (Fig. 126). The great relaxation of the ligaments at the joints may allow subluxation, especially at the knee and at the shoulder. The circulation in the affected limb is poor; it is often blue and cold.

FIG. 125.—AN OLD CASE OF INFANTILE SPINAL PARALYSIS OF THE ENTIRE LEFT LOWER EXTREMITY. Showing extreme atrophy of the thigh and leg, and a very characteristic deformity of the foot.

Very early in the disease the atrophied muscles begin to lose their power to respond to faradism. In the muscular groups which are to be permanently paralyzed, the faradic response may be lost in a week. The muscles in which recovery is to take place often preserve a certain degree of contractility. The response to the galvanic current may be increased for a few months, and then slowly fail as the muscular fibers themselves degenerate, and finally it may disappear altogether. The reaction of degeneration is present in the atrophied muscles, but in them alone.

Non-Paralytic Cases.—The terms "abortive" or "ambulant" are sometimes used to designate cases of acute poliomyelitis in which all the usual early symptoms of the disease are present, yet which recover without definite paralysis having developed. In some of these cases there is, however, a general muscular weakness. These represent instances of infection in which the nervous system either escapes altogether, or is so slightly involved as to give no definite symptoms. That such cases exist there can be no doubt. It is believed by many writers that in number

they equal or possibly exceed the paralytic cases. The evidence that they are genuine cases of acute poliomyelitis is not only their frequent clinical association in epidemics with frank cases, but has now, according to Flexner, been definitely established by laboratory findings, viz.: (1) there are certain characteristic changes in the cerebrospinal fluid—increased number of cells chiefly lymphocytes and the presence of globulin; (2) there has been demonstrated in the blood neutralizing immunity principles, such as are found in persons suffering from typical attacks, but not present in normal blood; (3) the virus has been detected on the nasal and buccal mucous membranes in such quantities as to make possible the communication of the disease to monkeys.

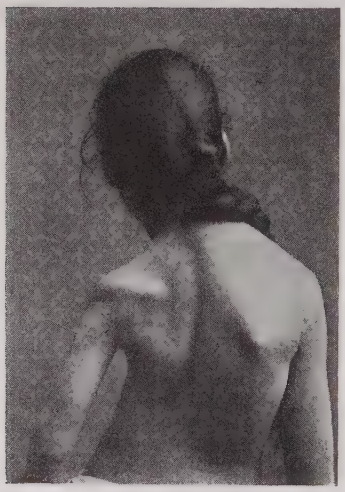


Fig. 126.—An Old Case of Infantile Spinal Paralysis of the Left Arm and Shoulder Muscles, with Resulting Lateral Curvature.

The recognition of non-paralytic cases of acute poliomyelitis has clarified many points in the spread of the disease. Poliomyelitis may be suspected by the fact of the attack occurring in close association with other typical paralytic cases; but there is nothing diagnostic in the clinical symptoms; the absolute diagnosis rests upon the laboratory evidence above cited.

Diagnosis.—The recognition of acute poliomyelitis before the occurrence of paralysis is impossible except by lumbar puncture. If this is performed early, the cerebrospinal fluid is found to be clear or slightly opalescent. The number of cells may be as many as 1,000 per c. mm. At first these may be nearly all polymorphonuclear; but soon they are replaced by lymphocytes, which generally form over 90 per cent of the cells seen. The test for globulin gives a positive reaction. By the time paralysis appears the cells have diminished greatly in number and soon the fluid may show no change by which it can be distinguished from the normal except an increased number of cells and an increased globulin reaction. The usual fluid found in acute poliomyelitis resembles that of tuberculous meningitis, which some cases closely simulate in their clinical symptoms. The v. Pirquet skin test is often a great aid in diagnosis; but in many instances one must wait two or three days for the course of the disease to declare itself, or until tubercle bacilli can be found in the cerebrospinal fluid. Cases with muscular pains, general hyperesthesia, rigidity and high fever may easily be confounded with cerebrospinal meningitis. It can be excluded only by lumbar puncture.

The later manifestations of the spinal type of poliomyelitis are a flaccid type of paralysis with marked atrophy and characteristic electrical reactions, but without sensory symptoms. It may be confounded with multiple neuritis, or the pseudo-paralysis of rickets. Multiple neuritis is rare in children except after diphtheria, and is more gradual in its onset. The type of paralysis and the electrical reactions may be the same as in poliomyelitis. Certain birth palsies, resulting from injuries received during delivery, may resemble poliomyelitis when the deltoid or shoulder group of muscles is involved. Without a clear history a differential diagnosis may be impossible. The muscular weakness of rickets is general; there is no reaction of degeneration and no history of acute onset. Scurvy is distinguished by the very acute hyperesthesia, by the swellings, and by hemorrhages from the gums or other mucous membranes together with a history of improper feeding. The child refuses to move his legs only because of pain. The cerebral form of poliomyelitis gives a spastic paralysis, usually hemiplegic in type, which may be indistinguishable from other forms of acquired cerebral paralysis.

Prognosis.—The dangers from poliomyelitis are twofold: that to life during the acute stage, and that to muscles in the form of permanent paralysis and disability. The mortality is much higher in epidemic than in sporadic cases. The death rate in the various large epidemics has ranged between 10 and 20 per cent. The danger to life is least in infants and very young children. In cases terminating fatally death usually occurs between the fourth and seventh days of the disease. The cause of death is generally respiratory paralysis or bronchopneumonia.

It is impossible to say in any case of advancing paralysis when it will be arrested. It rarely spreads after the seventh day. An important question in prognosis is whether paralysis will be permanent or not. Wickman reports recovery from paralysis in 44 per cent of 530 epidemic cases. This is a larger proportion than most writers give, and much larger than we have ourselves observed. Complete recovery from paralysis in 20 to 25 per cent of the cases is much nearer the average result.

Significant symptoms in any given case are the amount of wasting and electrical reactions. Muscles which soon lose completely their faradic contractility are almost certain to waste rapidly and severely. The best indication of coming improvement is the return of faradic contractility. If this is completely lost for six months, recovery is very doubtful; if faradic contractility is not lost, great and early improvement in the paralyzed muscles may be confidently predicted. After six months but little spontaneous improvement is to be looked for, and after two years none at all.

Treatment.—The communicable character of the disease being now established, it follows that all cases of acute poliomyelitis should be isolated; when the disease is epidemic this is imperative. It is not now known how long a given case may be infectious. A month's quarantine may be considered a minimum; but during epidemics a longer time should pass before an affected person should be allowed to mingle with other children. All discharges, especially those from the mouth and nose, should be disinfected and destroyed. Persons in contact with active cases should use some cleansing nasal spray or mouth wash as the only means now known for preventing infection. The same cleansing and disinfection of apartments should be practiced as after other infectious diseases.

Since we have as yet no specific remedy for poliomyelitis, the treatment during the acute stage is symptomatic and to be conducted along the same general lines as other acute infections. Hexamethylenamin (urotropin) has been extensively used in this disease, in doses of five to ten grains four times a day to a child of three or four years, but there is no convincing proof that any drugs are effective in aborting the disease or preventing or arresting paralysis. Absolute rest is essential, even in the mildest cases, and should be continued for an average period of two weeks; longer when irritative symptoms are protracted. Pains in the affected limbs during the acute stage may be lessened by the application of splints to insure immobilization and also at times by wrapping limbs in cotton. There should be as little handling as possible. It is important to support the limbs, so as to lessen the chances of deformity. There should be placed at the feet pads or sand-bags, to prevent foot-drop, which otherwise is almost certain to occur in cases of anterior tibial paralysis. Severe pain may require the administration of morphin or codein. Paralysis of respiration in the acute stage is practically beyond help. Feeding through the tube is sometimes necessary in bulbar cases for a considerable time, owing to paralysis of the muscles of deglutition.

When all acute symptoms have subsided, which is generally in three or four weeks, measures should be begun for the development of the paralyzed muscles. The beneficial effects of electricity have been greatly overestimated. In many cases, however, it is useful, but should never be alone relied upon. Faradism may be used three times a week for such muscles as respond to it; for other muscles galvanism should be employed. The pain and terror which the use of electricity excites in most small children makes its continuance a practical impossibility. It is far better under such circumstances to rely on other measures.

Massage and passive movements may be begun as soon as hyperesthesia has gone, and may be used at first daily and soon twice a day to

all affected parts. They should be continued for years. But still more important are active voluntary movements carried out by the patient himself, which should be developed with great care and systematically carried out for an indefinite period. It is really surprising what such measures when intelligently used can accomplish.

Mechanical Treatment.—Mechanical appliances are useful to prevent deformity, also to furnish support to the limb in order to enable the child to walk. By such means many get about with tolerable comfort for whom locomotion without apparatus is impossible except with crutches. To overcome existing deformities in neglected cases, braces are employed in conjunction with myotomy or tenotomy of the various shortened tendons, excision of portions of elongated tendons, and the production of artificial anchylosis in cases of "flail joints." By these means the orthopedic surgeon is able to give a great deal of relief to these unfortunate and sometimes helpless patients.

TUMORS OF THE SPINAL CORD

Tumors of the cord are exceedingly rare in children and almost unknown in infancy. They spring from the bone, from the meninges, or from the cord itself. The most common meningeal tumors are sarcomata, fibromata and lipomata, the last named being found in association with spina bifida. In the cord gliomata, sarcomata, solitary tubercles and gummata may be encountered.

The first and most important symptom is pain. This may be in the extremities or in a girdle form around the body. Associated with the pain may be a zone of hyperesthesia. Eventually there may be anesthesia. Motor symptoms are manifested sooner or later. There may be contractures or tonic spasm and finally spastic paralysis with exaggerated reflexes and ankle clonus. The arms are seldom involved. Especially characteristic is the Brown Séquard paralysis—a unilateral paralysis with a zone of hyperesthesia upon the paralyzed side and with anesthesia upon the opposite side. Paralysis of the bladder and rectum is present, but is not always an early symptom.

The diagnosis of tumor is to be made from these general symptoms, in the absence of injury or of caries of the spine, which is the most common cause of transverse lesions of the cord in childhood. The localization of the growth is to be made according to the rules of general neurology. This is difficult in childhood, because the tumors are apt to be diffuse (gliomata, sarcomata, tuberculomata) and because of the frequent inability to obtain the necessary coöperation from the child. The general symptoms are also very uncertain. We have seen a fibroma of

the meninges in a five-year-old child successfully removed by operation, which caused no pain at any time.

The prognosis is bad. Few cord tumors in childhood are of such a character or in such a situation that they can be removed. Unless they are malignant, or can be removed, death results from intercurrent disease, from bed-sores or from ascending inflammation of the urinary tract. The treatment is surgical. If operative removal is impossible, nothing can be done except to make the patient comfortable.

HEREDITARY ATAXIA—FRIEDREICH'S ATAXIA

While cases of Friedreich's ataxia are encountered with no history of a like disease in relatives, the disease is especially likely to attack several members of a family in one or more generations. As many as eight sufferers from the disease in one generation have been reported, and it has been traced through three generations. Friedreich's ataxia is for this reason to be classed among the hereditary degenerative diseases of the nervous system. Except for this hereditary influence, there is no etiological factor known.

Friedreich's ataxia is an infrequent disease, consisting of two fairly distinct types. The type of interest to pediatrists occurs early in childhood, usually between the fourth and seventh year. The other type, often known as the Marie type, is seldom found before the twentieth year and need not, therefore, concern us here.

The pathological changes are chiefly in the cord. These consist in a diminution in the circumference of the cord throughout its entire extent and in a degeneration of various tracts, chiefly the posterior columns. The column of Goll is affected throughout, the column of Burdach to a greater or less degree, and the crossed pyramidal tract to a slight extent. In addition, the cells of Clarke's column are degenerated and there is a consequent degeneration of the direct cerebellar tract and the bundle of Gowers. There is no degeneration in the cells of the anterior horns.

Symptoms.—Ataxia is the most striking and usually the earliest symptom. It is first noticeable and is always most marked in the legs. There is difficulty in walking and even in standing, but the ataxia of the legs is noticeable in any position, even when lying down. The children stand with their legs wide apart. In some instances there may be a distinct Romberg symptom, it being impossible for them to stand at all with the eyes closed. The gait is ataxic, much like that of locomotor ataxia at first, but later it may be so disturbed that the patient reels from side to side as if intoxicated. Eventually locomotion is impossible especially when the muscular weakness, which is regularly present, be-

comes extreme with atrophy. Early in the disease muscular weakness is slight. There may be wobbling of the head and there is usually a coarse tremor of the arms and hands. Sensation is well retained and control over the bladder and rectum is normal. Exceptionally there are sharp, lancinating pains in the legs. The knee jerks are commonly absent. Slow, scanning, sometimes explosive speech is very frequent and late in the disease speech may be nearly impossible. There is often a marked nystagmus. A striking symptom is the common deformity of the foot. This may be one of the first symptoms to be noticed. The foot appears shortened, it is markedly arched and is held in a position of slight equino-varus. The great toe is hyper-extended and sometimes the terminal phalanx is flexed. Kypho-scoliosis develops with the advent of muscular weakness. The intelligence is well retained for a time but suffers deterioration in the course of the disease. In the form described by Marie, there are often exaggerated reflexes, optic nerve atrophy and paralysis of the extra-ocular muscles; but these symptoms are seen in children with the greatest infrequency.

The course of the disease is progressively downward, the ataxia becoming more marked and that and the muscular weakness make walking impossible. The patient eventually becomes bed-ridden, in a condition of dementia. But the progress of the disease is very slow. It may last twenty or thirty years or more. Death is usually due to some intercurrent disease and is rarely the result of asthenia. Friedreich's ataxia is incurable. It can only be treated symptomatically.

DISEASES ASSOCIATED WITH PROGRESSIVE MUSCULAR WASTING

A number of diseases in infancy and childhood are accompanied by muscular wasting. This may be secondary to disturbances of nutrition, to some chronic infection or it may result from disuse. Wasting is also present with organic diseases of the nervous system, particularly as the result of some acute lesion such as poliomyelitis, and also with chronic crippling diseases such as spastic paraplegia, chronic meningitis, etc. But there is a group of diseases in infancy and childhood that is characterized chiefly by progressive muscular wasting with great weakness. They develop insidiously and with but few exceptions progress uninterruptedly to a fatal termination. They are of great chronicity and are practically incurable. Many show a marked hereditary tendency. Of these diseases, there are a number of more or less clear cut types that may be recognized clinically and pathologically. There are, however, very many cases that pathologically, as well as clinically, have the characteristics of two or even more types. For this reason, it has

been hard to classify these diseases. There has been much difference of opinion in regard to them and there are obvious objections to all methods of classification. The lesions in some of these cases are chiefly in the cord; in others, in the nerves, and, in the largest group, in the muscles. We shall therefore group them as the spinal, the neural and the muscular forms.

Spinal Muscular Atrophy.—The spinal forms are unusual in childhood. Chronic bulbar paralysis and the *Aran-Duchenne* type of spinal atrophy are so rare at this age as to be of little importance to pediatrists. The other type of central atrophy that has been described, though infrequently, is

The Werdnig-Hoffmann Type.—This disease is markedly hereditary; several children in a family may be affected and the disease has been traced through two or three generations. It is not a common disease—only twenty or thirty cases in all have been reported. The onset is early, usually toward the end of the first year. A weakness in the thighs and back develops in a child that up to that time has been entirely normal. This weakness extends so as to involve the shoulders, the neck, and, eventually, the arms and thighs. The legs and lower arms are only involved late in the disease, and the hands and feet rarely at all. There is marked atrophy of the muscles, particularly those of the pelvis and shoulders. The muscles show at times fibrillary contractions and there is always loss of deep and generally of superficial reflexes. There is a great diminution in response to both faradic and galvanic currents. The muscles of the face usually escape entirely. Bulbar symptoms are very unusual. Speech is normal and the mentality remains unaffected to the end. There is no interference with sensation. The progress of the disease is quite rapid. Death usually results in two or three years, from respiratory involvement or from pneumonia. The localization of the chief muscular paresis and atrophy in the pelvic and shoulder girdles, the progressive character of the disease and the retention of a clear mentality, distinguish it from the other diseases with which it is likely to be confounded which are chiefly: amyatonia congenita, poliomyelitis, progressive neural and muscular atrophy and amaurotic idiocy.

The pathological changes are clearly marked. There is an atrophy of the spinal cord, with degeneration of the cells in the anterior horns throughout its whole extent from the medulla to the cauda equina. Secondary to this is a degeneration of the anterior roots of the cord and of the motor nerves, with great atrophy of the muscles. There are no changes in the pyramidal tracts.

The progress of the disease is rapid. It is unbroken by periods of remission and the outlook is hopeless. No treatment has any effect.

Neural Muscular Atrophy.—The existence of a form of muscular atrophy dependent upon primary changes in the peripheral nerves is denied by many authors. We have retained such a classification for the reasons that in the peroneal type of muscular atrophy there are frequently severe lesions in the nerves, that the type is generally clearly marked, and that the disease runs a much more benign course than any of the other forms of muscular atrophy.

Peroneal Type. (Charcot, Marie, Tooth.)—This form of muscular atrophy exhibits as marked familial tendencies as any other known disease. Examples of it have been met with in five generations and it is seldom confined to one member of a generation. Herringham has recorded a family in which 26 members had been afflicted with the disease. The onset is generally after the sixth year. It begins slowly and symmetrically in the distal parts of the extremities, usually the legs. The extensor longus hallucis and the extensor longus digitorum and the tibialis anticus are usually the first muscles to waste; afterward the peroneal group. The localization of the muscular weakness causes inability to flex the foot, which hangs down, causing an impediment to walking. To overcome the impediment the knees must be markedly flexed, which causes the "stepping" gait. Double club foot in the position of equino-varus, often results from unopposed muscular action and from attempts to walk. On this account many of the cases first come to the attention of orthopedic surgeons. It is uncommon for the disease to begin in the hands, but instances of such a mode of onset have been reported. The atrophy then affects the small muscles of the hands. As the disease progresses the legs and forearms gradually become involved but the thighs and upper arms remain free. There is no hypertrophy of muscles or pseudo-hypertrophy. There may or may not be fibrillary twitching of the muscles. Sensation may be normal or there may be complaint of paresthesia, or of feelings of heat and cold. Shooting pains may be felt but the pain is never very severe and is frequently entirely absent. Control of the bladder and rectum is complete. There is a diminution of response to the faradic and also galvanic currents in the affected muscles and in certain instances this may obtain in muscles which are apparently normal.

The course is an exceedingly slow one and usually not continuously progressive. In this regard it differs greatly from the allied conditions. There may be remissions of such length that cure may be said to have occurred. The disease seldom results in death and many patients live an active, self-supporting life for years. A return of the affected parts to a normal condition is impossible even though complete arrest may take place.

The nerves in the peroneal type of muscular atrophy are almost

always the seat of a marked interstitial growth. Associated with the neural change is a degeneration of the posterior columns of the cord and a marked atrophy of the muscles involved. Here, as in all these allied diseases, exceptions may be found in a preponderant alteration in the cord and muscles and an almost complete escape of the nerves.

No known treatment arrests the prognosis of the disease. Electricity, massage and baths may assist in retaining muscular function. Orthopedic treatment (tenotomy, braces, etc.) is of marked aid in preserving the ability to walk.

Muscular Dystrophies.—There are certain well established facts in regard to the muscular dystrophies. The changes are primarily in the muscles. They are not dependent upon lesions of the nerves or the cord even though secondary degenerations may be present in those situations. While isolated cases are here and there encountered, muscular dystrophies are family diseases. They affect boys rather more often than girls. What it is that determines the progressive wasting of the muscles is quite unknown. It appears to be an inherent weakness of the muscular system, an inability of the muscles to carry on the fight for existence. They fail to survive as various parts of the nervous system may fail.

The lesions of muscular dystrophy are essentially the same, no matter what the type. The individual muscle fibers waste. They become round instead of polygonal and eventually they disappear, leaving the sarcolemma sheath, with greatly increased nuclei. Certain of the fibers may actually hypertrophy to several times the size of the normal fiber, but this is only a temporary process. Eventually the hypertrophic fibers share in the general atrophy. Replacement of the muscle fibers by connective tissue occurs as the atrophy goes on, and coincident with the muscular atrophy a deposition of fat takes place in the muscle. This may largely compensate in amount for the atrophy of true muscular substance so that the diminution in size of the whole muscle may be very gradual. This deposition of fat may even be excessive and thus the pseudo-hypertrophy is caused. Upon the relative amounts of the muscular tissue, connective tissue and fat, depends the appearance of the muscles as a whole. They are lighter in color than normal, perhaps even yellow and soft. Eventually, the fat largely disappears and only firm, fibrous and contracted strands of connective tissue are left.

Pseudo-Hypertrophic Paralysis.—This is the most frequent and best-known variety of the muscular dystrophies. The symptoms as a rule come on early in childhood, nearly always before the tenth year, and generally between the second and seventh. The earlier symptoms relate to a general weakness of the lower extremities, which is accompanied by a marked increase in the size of certain muscular groups,

usually those of the calves, but sometimes more of the thighs or the gluteal regions. The enlargement may affect almost any muscular group of the lower extremity. Children walk unsteadily, and fall very easily. They have special difficulty in rising from the floor and in mounting stairs. The method of rising in well-advanced cases is quite characteristic; the patient lifts his body until he touches the floor only with the hands and feet; then he proceeds to "climb up himself" by putting first one hand upon the knee, and then the other, gradually moving his hands higher and higher up the thighs until the erect position is attained. This is seen in many of the cases, but not in all.

FIG. 127.—MUSCULAR PSEUDO-HYPERTROPHY. Showing to a moderate degree the large calves and gluteal regions with a marked lordosis. (From a photograph by Dr. M. A. Starr.)

Most of these patients exhibit, while standing, a marked degree of lumbar lordosis, due to the weakness of the extensors of the hip and later of the muscles of the back. They stand with their shoulders far back. This is well shown in Fig. 127. The patient may be so weak upon his legs that the slightest touch will cause him to fall, even with his apparently immense muscular development. The small muscles are generally weaker than those which are enlarged.

With the progress of the disease, the muscles of the arms and shoulders become involved. Some of these atrophy at once, others may exhibit pseudohypertrophy for a time. The infraspinatus is the most frequently enlarged, next the supraspinatus and the deltoid. The pectorals and latissimus dorsi are never enlarged but are generally markedly wasted. The weakness of the shoulder muscles makes the characteristic difficulty in picking the child up by grasping him under the arms. They slip through the hands. The rhomboids and the levator angulae scapulae, the biceps and the triceps gradually are involved, and later in the disease there is such marked atrophy with corresponding weakness of all the affected groups that the patient may be unable to walk or even stand.

and is absolutely helpless with the exception of the use of his hands. The knee-jerk is at first normal, but gradually diminishes until it is finally lost. The electrical reactions are normal until marked wasting occurs, when there is a lessened response to faradism and galvanism, but never the reaction of degeneration. There are no fibrillary contractions, and no sensory disturbances. The progress of the disease is generally slow, and sometimes irregular. It is often more rapid in early childhood, and slower after puberty. Many of these children, though apparently bright, are distinctly below the average for their ages.

The prognosis is grave, most patients dying in from five to ten years. Death seldom results from the disease itself, but rather from some intercurrent disease, especially of the lungs. Nothing can be done to stay the course of muscular dystrophy. The diagnosis is generally easy from the apparent hypertrophy and actual weakness of the muscular groups. The disease is incurable.

The Juvenile Form of Muscular Atrophy (Erb's Type).—This is much less frequent than the form just described and usually begins somewhat later in life, between the tenth and sixteenth years. It is characterized by progressive wasting of certain muscular groups, especially those about the shoulders and pelvis, and hypertrophy of other groups. Of the shoulder and upper extremity, the muscles affected are the pectorals, the trapezius, the latissimus dorsi, the serrati, the rhomboidei, the muscles of the upper arm and the subscapularis. The deltoid, infraspinatus and supraspinatus for a long time escape, and may be hypertrophied. The hand and forearm are not involved. In the lower extremity, the muscles of the pelvis, thighs, and gluteal regions are affected, while those of the leg and foot escape until late in the disease. Weakness and atrophy of the muscles of the back cause lordosis of great severity. In this disease there are no fibrillary contractions, no reaction of degeneration, and no sensory disturbances. The course and result of this form are essentially the same as in the preceding variety. It is now regarded as the same disease pathologically, the only difference being that of localization. In the terminal stages differentiation may be impossible and mixed cases that demonstrate from the beginning the predominant characteristics of both types are encountered.

Landouzy-Dejerine Type.—In this, wasting begins in the muscles of the face; the lips are thickened and weakened. They cannot be firmly closed, but all the rest of the facial muscles are markedly atrophied, giving a peculiar expression to the mouth known as "the tapir mouth." Speech may be greatly interfered with but the muscles of mastication and deglutition are not affected. This serves to differentiate the disease from bulbar paralysis. Later, the atrophy extends to

the shoulders and arms, but does not involve the supraspinatus or infraspinatus, or the flexors of the hand and forearm. This type is sometimes described as beginning in the shoulders, or even in the legs. The description therefore corresponds to the juvenile form of Erb, with the addition of the facial symptoms, and it is undoubtedly a variety of the same disease.

CONGENITAL MYATONIA

(Congenital Amyotonia—Oppenheim's Disease)

This disease was first described by Oppenheim in 1900. Its cause is unknown. The symptoms are usually noticed in the early months, sometimes very soon after birth. In some cases it has been observed even during pregnancy that fetal movements were less vigorous than usual. There is a general flaccid paralysis. That of the lower extremities is usually complete; but in the upper extremities feeble movements of hands or arms may be present. The intercostal muscles and those of the neck are usually but not always involved. The diaphragm and all the muscles supplied by the cranial nerves escape. There is no ocular or facial paralysis.

In the well-marked cases the child lies completely helpless and motionless; the knee jerks are absent; but sensation is not affected and the mentality is normal. The electrical reactions are feeble or even may be absent. Owing to involvement of the intercostals the respiration is usually labored, panting and diaphragmatic in character. Secretions accumulate in the pharynx and air passages and choking attacks often occur. These may result in fatal asphyxia, or in aspiration pneumonia. The pulse is normal and regular. There are apparently no subjective symptoms. The infants are usually well nourished and may even be very fat. In those who live for several months or years the intelligence is apparently normal and control over the sphincters complete. Deformities of the chest are often produced as a consequence of the paralysis of the respiratory muscles.

Besides the marked form of the disease, to which the above description refers, it is now recognized that myatonia may occur in all degrees of severity. In the mild form there may be only very great weakness and atony of the muscles. The ability to hold up the head or to walk may then be greatly delayed though the intelligence may be quite normal. These cases are often confounded with rickets; but the weakness in myatonia is permanent. Owing to the greater involvement of some muscular groups, contractions of opposing groups may occur. This may lead to confusion with poliomyelitis. The milder forms of myatonia

may be readily overlooked and may cause but little interference with function. Between these and the severe forms of the disease there are seen all degrees of muscular weakness.

The lesions are chiefly in the muscles, which show great atrophy and degeneration. They may waste to fibrous cords or may largely be replaced by connective tissue and fat. In several of the cases the cells of the anterior horns of the cord have been found reduced in number, sometimes almost absent, and the anterior nerve roots atrophic. The brain is normal. The nervous lesions are to be regarded as a failure of development rather than a degeneration. It is believed by some that they are the primary condition, the lack of muscular development being the result of deficient innervation.

Many of these infants suffering from this disease die in the first year, most frequently from bronchopneumonia to which they are especially predisposed by reason of the condition of the respiratory muscles. The duration of the mild forms of the disease is indefinite. We have seen a few older children and young adults with this form of the disease. In some cases a slight improvement has taken place; but no cures have been reported. The condition is not influenced by treatment. The disease usually either remains stationary or very slowly progresses, the child dying of some intercurrent disease.

CHAPTER V

DISEASES OF THE PERIPHERAL NERVES

MULTIPLE NEURITIS

Under the term multiple neuritis are included those cases in which several nerves are involved in an inflammatory process, which may at times be general. In its distribution multiple neuritis is usually symmetrical, but it is not necessarily so.

Etiology.—The chief cause of multiple neuritis in children is diphtheria, although it is occasionally seen after other infectious diseases, especially malaria, typhoid or scarlet fever, measles, and mumps. In diphtheria the inflammation is due to the direct action of the toxins upon the nerve structures, since it can be induced in animals by injecting toxins into the circulation. There is little doubt that in all infectious diseases the inflammation is excited in a similar way. The metallic poisons, lead and arsenic, are rarely the cause of multiple neuritis in early life, and the same is true of alcohol, although a marked case from

this cause has come under our observation in a child only three years old.[1] Lastly, there are cases in which the cause assigned is simply exposure to cold—those classed as rheumatic.

Lesions.—Almost any nerves in the body may be affected, although the distribution varies somewhat with the cause of the disease. The musculo-spiral and the anterior tibial nerves are most frequently involved, but the inflammation may affect any of the spinal nerves, including the phrenic, and occasionally the cranial nerves, especially the pneumogastric, hypoglossal, oculomotor, and abducens. Several nerves in different parts of the body are usually affected, the lesion being in most cases symmetrical.

The affected nerve is sometimes red and swollen, owing to acute congestion and edema or to a sero-fibrinous exudation. In other cases the changes are almost entirely degenerative. The microscope shows the changes sometimes to be chiefly interstitial and sometimes chiefly parenchymatous. There is an exudation of cells into the sheath, between the sheath and the nerve fibers, and even between the nerve fibers themselves. The myelin breaks up into granules, and in places may completely disappear. The late changes are those of subacute or chronic degeneration of the nerve fibers.

With these changes in the nerves there are associated, in some cases, inflammatory and degenerative changes in the ganglion cells of the spinal cord, although they are much less severe than are the lesions in

[1] This case was in many respects a remarkable one. The boy completely emptied a decanter containing twelve ounces of whisky, but almost immediately vomited the greater part of it. He soon after showed the symptoms of alcoholic intoxication, and in a few hours became comatose, in which condition he continued for twelve hours. After this he gradually lost power in his legs, and at the end of a week was unable to walk at all. He had convulsions, and after this there developed the usual symptoms of meningitis at the convexity, with which he was admitted to the Babies' Hospital, three weeks after drinking the whisky. The child was then unconscious and there was present incomplete paralysis, affecting all four extremities, with anesthesia of the arms. The active inflammatory symptoms continued for six weeks longer, during which time there were repeated convulsions, continuous stupor, fever, gradually increasing deformities, marked atrophy, loss of reflexes, and great diminution in the faradic contractility of all the paralyzed muscles; in the thighs, left leg, and abdominal muscles there were no responses to a strong current, but there was nowhere the reaction of degeneration. The child was at death's door for three or four weeks. Three months after the attack the first signs of improvement were observed in the cerebral symptoms. Shortly afterward he began to use his hands, and at the end of six weeks he was walking alone and talking freely. The improvement was very rapid, and eight weeks from the date of the first change for the better, and five months from the time of taking the whisky, he was as well as ever. The diagnosis was multiple alcoholic neuritis, with a convexity meningitis. (Fig. 128 is from a photograph taken while the symptoms were at their height.)

the nerves. However, they were once regarded as the explanation of some of these cases, particularly of diphtheritic paralysis.

Symptoms.—The onset of multiple neuritis is in most cases a gradual one, it being usually from two to four weeks before the paralysis reaches its height. Very exceptionally the onset may be abrupt, with fever, and marked paralysis in a few days. It is characteristic of this disease that both motor and sensory symptoms are present, and that they are the same in their distribution. The symptoms are usually symmetrical. There is first noticed a general weakness in the affected muscles, which slowly increases to complete paralysis. As the extensor groups of the hands and feet are apt to be affected, there are wrist-drop and foot-drop (Fig. 128). The paralysis may begin in the feet and hands,

FIG. 128.—ALCOHOLIC NEURITIS, SHOWING CHARACTERISTIC DROPPING OF THE FEET. This position of the lower extremities was maintained for over a month. Boy three years old.

and gradually extend until it involves not only the four extremities, but even the muscles of the trunk and the neck, although this is rare. The child may then be absolutely helpless, unable to sit up, or even to support his head. In such cases the head seems loosely attached to the body, and rolls about on the shoulders like a ball. Weakness of the spinal muscles leads to deformities (Fig. 129) which may be mistaken for Pott's disease, even by experienced observers. In most of the muscular groups the paralysis is incomplete. The symptoms which relate to the phrenic and the cranial nerves will be described with Diphtheritic Paralysis, for they are rarely seen in any other form. It is characteristic of multiple neuritis that the bladder and rectum escape.

The sensory symptoms are marked only in the early stage of the disease, while the paralysis is increasing; they improve so much more rapidly than the motor symptoms, that they may be altogether wanting at the time that the paralysis is at its height. In some cases they are so slight as to be overlooked. There is usually pain along the course

of the affected nerves, which is sharp and neuralgic in character, and generally associated with acute tenderness of the nerve trunks and of the muscles. Often there is a general hyperesthesia in the early part of the attack, followed by partial anesthesia. The sensations of touch, pain, temperature, and the muscular sense are all about equally affected.

Ataxia is not uncommon, and may be a more striking symptom than the loss of power. All the reflexes are diminished or lost, especially the knee-jerk, as the legs are usually most affected. Sometimes, particularly after diphtheria, there is loss of the knee-jerk, when there is no other symptom of neuritis. In the severe cases muscular tremor is frequently present.

Fig. 129. — Multiple Neuritis after Diphtheria in a Child Four Years Old. The position of the head and spine is due to partial paralysis of the trunk and neck. The legs were also affected.

Atrophy is a prominent symptom of neuritis, and it is evident early in the disease, often being quite as rapid as in poliomyelitis. The electrical reactions are altered—every grade of reduction in the responses being seen, from a slight diminution in the reaction to faradism to the complete reaction of degeneration. Vasomotor symptoms, such as edema of the affected parts, glossiness of the skin, etc., are often present. Deformities from muscular contraction occur early; they may be severe, and in some cases, permanent.

Course and Prognosis.—The usual course of the disease is for the symptoms gradually to increase for three or four weeks and then improve, sometimes rapidly, but more often slowly, the case usually going on to complete recovery in the course of a few months. Exceptionally the paralysis may be permanent. The sensory symptoms always disappear before the motor ones. Multiple neuritis may prove fatal, from paralysis of the heart or the muscles of respiration, or death may be due to asphyxia from the entrance of food or foreign bodies into the air passages, owing to anesthesia of the epiglottis and paralysis of the muscles of deglutition. Death sometimes follows from complications, especially pneumonia. The electrical reactions are of much prognostic value

in regard to the persistence of the paralysis. If the reaction of degeneration is present the paralysis is certain to last many months, and some muscles are sure to be permanently affected. Where there is simply a diminution in the faradic responses, even though accompanied by marked atrophy, complete recovery may be expected, although it is often slow.

Diagnosis.—The diagnostic features of multiple neuritis are the combination of motor and sensory symptoms with the same distribution, the occurrence of atrophy, and the diminution in the electrical responses, even the reaction of degeneration. The gradual onset and the widespread distribution of the paralysis are also characteristic. If all four extremities are paralyzed, it is altogether the most probable disease; and if to this is added paralysis of the neck and spinal muscles, the diagnosis is almost certain. The facts that the paralysis is often incomplete, and that it involves parts distant from each other, are also important. Neuritis may be mistaken for poliomyelitis, for Landry's paralysis, or for Pott's paraplegia; an important diagnostic point from the last mentioned is the condition of the reflexes—being greatly exaggerated in Pott's paraplegia, while they are diminished or lost in multiple neuritis.

Treatment.—As this disease tends in the great majority of cases to spontaneous recovery, it is difficult to estimate the value of any method of treatment. Causes, such as lead, arsenic, alcohol, and malaria, are to be sought and removed as the first step. During the acute stage the pain may be so severe as to require relief, which is best accomplished by the application of heat. In using counter-irritation much care is necessary, for troublesome ulceration may follow. After the acute stage has passed, or at the end of three or four weeks, electricity should be begun, faradism being used if the muscles respond to a moderate current, otherwise galvanism. This should be continued daily until recovery. Strychnin is much used in these cases, but it is doubtful whether it has any specific influence, although as a tonic it is valuable. Other tonics, such as iron, quinin, and cod-liver oil, should also be given. Massage is also beneficial. The special treatment of cardiac and respiratory paralysis will be discussed in the following article.

DIPHTHERITIC PARALYSIS

This is not only the most frequent variety of multiple neuritis, but it has some peculiarities which make a separate consideration of it desirable.

Frequency.—According to the statistics of various observers, paraly-

sis, including all varieties, occurs after diphtheria in from 5 to 15 per cent of the cases. Sanné gives 11 per cent in 2,448 cases; Lennox Browne, 14 per cent in 1,000 cases (in neither of these groups did the patients receive antitoxin); the Report of the Collective Investigation by the American Pediatric Society, 9.7 per cent of 3,384 cases which were treated by antitoxin. The most recent figures are those of J. D. Rolleston. He encountered some form of paralysis in 20.7 per cent of 2,300 cases, all personally observed by him.

There can be little doubt that since the introduction of treatment with antitoxin more cases of post-diphtheritic paralysis are observed than in the pre-antitoxin days. The undoubted explanation of the frequency with which paralysis is seen after antitoxin treatment is that patients now live long enough to develop paralysis, when without antitoxin the same patients would have died during the early stage of the disease.

Neuritis is more likely to follow severe than mild cases. Its occurrence after some very mild attacks shows how great is the susceptibility of the nervous tissues to the action of the poison. But the great determining factor is the duration of the action of unneutralized toxin upon the nerves. The frequency of neuritis is in direct relation to the length of time elapsing before the administration of antitoxin. Rolleston's figures upon this point are illuminating. When antitoxin was given on the first day of the disease, 3.6 per cent of the cases subsequently developed paralysis; on the second day, 14.09 per cent; on the third day, 21.4 per cent; on the fourth day, 26.9 per cent; on the fifth day, 26.3 per cent; on the sixth day, 27.1 per cent. No better proof of the protection of the nervous system by antitoxin can be adduced.

Time of Occurrence.—During the second week, and sometimes even during the latter part of the first week, the early paralysis occurs, usually affecting the palate. The most frequent and most characteristic paralysis—that affecting the throat, eyes, extremities, and respiration—begins at a later period, usually not before four or five weeks after the throat has cleared off, and sometimes even later than this.

Extent and Distribution of the Paralysis.—Ross gives the following statistics of 171 collected cases of diphtheritic paralysis: palate affected in 128; eyes in 77, in 54 of which the muscles of accommodation were involved; lower extremities in 113; upper extremities in 60; trunk or neck in 58; muscles of respiration in 33. In the 477 cases reported by Rolleston the paralysis was distributed as follows: palate, 331 (74 per cent); ciliary muscles, 236 (53 per cent); extra-ocular muscles, 80 (18 per cent); pharynx, 36 (11 per cent); diaphragm, 16 (3.6 per cent).

Symptoms.—In the great majority of cases the throat is affected, and usually the paralysis is first noticed there. It may involve the palate alone, or the muscles of the pharynx or larynx in addition. The muscles of the extremities or of the eye are often next attacked. In severe cases there may also be involved the muscles of the trunk and neck, and sometimes the diaphragm. Paralysis of the throat and diaphragm distinguishes diphtheritic paralysis from other forms of multiple neuritis. Whatever the extent or situation of the paralysis, the knee-jerk is nearly always lost. The symptoms in the extremities and the trunk do not differ from those of multiple neuritis from other causes. The throat paralysis shows itself by a nasal voice and by regurgitation of fluids through the nose, sometimes by difficulty in swallowing or by the entrance of food into the larynx, owing to anesthesia of the epiglottis and paralysis of the muscles of deglutition. There may be difficulty in protruding the tongue or in articulation. Facial paralysis is rare. On the part of the eye there is most frequently seen inability to read, owing to paralysis of the muscles of accommodation; there may be dilatation of the pupils, rarely strabismus or ptosis.

Respiratory paralysis may be due to involvement of the phrenic or the intercostal nerves, more frequently the former. Extensive paralysis of other parts—the throat, extremities, or trunk—usually precedes. The first warning is generally in the form of occasional attacks of dyspnea, sometimes accompanied by cough. Gradually these attacks increase in frequency and severity. The voice is reduced to a whisper. As the diaphragm is usually affected, the breathing is entirely thoracic. The respiratory movements are rapid, but irregular, shallow, and ineffectual. There is cyanosis, also great subjective as well as objective dyspnea. The anxiety, distress, and apprehension of the patient are sometimes terrible. There is a constant dread of impending suffocation, and the respiratory movements are continued only by the patient's constant efforts, otherwise they would cease altogether. After a few hours these severe symptoms may subside, to return after a short respite. There may be several such attacks during two or three days, in each of which death seems imminent. Unfortunately, this is the most frequent termination. Of thirty-three such cases collected by Ross, only eight recovered. Associated with these respiratory symptoms others may be present. There may be attacks of abdominal pain, vomiting, and disturbance of the heart's action—usually an irregular or intermittent pulse, which may be either unnaturally slow or very rapid. In many cases the heart continues to beat normally, even though the respiration is so much disturbed.

The premonitory symptoms of cardiac paralysis are an irregular or intermittent pulse, often slow, but becoming very rapid from even the

slightest exertion. It is always weak and compressible. The first sound of the heart is feeble and may be reduplicated. Heart block, the dissociation of auricular and ventricular contraction, has been reported. As the symptoms increase there is marked pallor, coldness of the extremities, great restlessness, anxiety, precordial distress, and perhaps orthopnea. Within twenty-four hours from the beginning of such symptoms death usually occurs. In other cases it may come suddenly without any warning, or with a warning so slight as to be overlooked. At such times it often follows some muscular exertion, such as getting out of bed, walking across the room, or so slight an effort as sitting up suddenly in bed. Fits of temper or other excitement have at times produced it.

It is by no means certain that cardiac paralysis is due to a lesion of the cardiac nerves. Toxic myocarditis appears to be a more important factor in producing the fatal result.

Death in diphtheritic paralysis is usually due either to cardiac or respiratory paralysis. Of 171 cases of all varieties collected by Ross, 45 were fatal, while of Rolleston's 477 cases, 85 were fatal. Death can be ascribed to the paralysis in only a small proportion of cases. It results usually from cardiac failure which is due to myocarditis and not to true neuritis. Cardiac failure was the cause of death in 80 of Rolleston's 85 fatal cases. The prognosis of diphtheritic paralysis is grave because it indicates that a serious form of diphtheria has been present and usually that antitoxin has been given late. The pharyngeal and diaphragmatic paralyses may of themselves be fatal, the former by causing aspiration pneumonia.

Treatment.—Cases of paralysis of the trunk or extremities are to be managed like others of multiple neuritis. In severe forms of throat paralysis feeding by a stomach tube should be employed, on account of the danger of the entrance of food into the air passages. It must in most cases be continued for several days. The tube may be passed either through the mouth or the nose.

The great mortality attending the myocarditis occurring with diphtheritic paralysis shows how unsuccessful is treatment in most of the cases; still, no doubt there are instances where life may be saved by judicious treatment. In cases of threatened cardiac failure the drug most to be depended upon is morphin, hypodermically; this should be used every two or three hours in sufficient doses to keep the patient under its influence while threatening symptoms are present. The patient should be kept absolutely quiet, not even being allowed to turn in bed. In respiratory paralysis the general reliance is upon atropin or strychnin used hypodermically in full doses, and faradization of the respiratory muscles, particularly the diaphragm.

FACIAL PARALYSIS

Peripheral paralysis of the face occurring as a result of injury inflicted during delivery has already been described. There remain to be considered here cases which arise from causes that operate at a later period. The facial nerve may be affected in any one of three situations—after its exit from the cranium, in the bony canal, and within the cranium.

In the first situation, the principal cause of neuritis is exposure to cold, the "rheumatic" cases; but it occasionally occurs as a complication of mumps and disease of the lymph glands of this region. The nerve is affected just after it has escaped from the stylomastoid foramen, and all the branches given off beyond its exit are involved. There is paralysis of the muscles of the forehead, those about the eye, cheek, nose, and mouth. The affected side of the face is smooth, there is inability to wrinkle the forehead, contract the eyebrows, close the eye completely, raise the nostril, whistle, or blow. The mouth is drawn to the healthy side (Fig. 130). If the paralysis is complete, there may be difficulty in drinking or in articulation. In partial paralysis the symptoms may not be noticeable while the face is at rest. There are no sensory symptoms. The electrical reactions resemble those of other forms of neuritis; there is diminution in the response to the faradic current, which is more or less marked according to the severity of the lesion, and there may be the reaction of degeneration.

FIG. 130.—FACIAL PARALYSIS OF RIGHT SIDE FROM MIDDLE-EAR DISEASE IN A CHILD TWO AND A HALF YEARS OLD.

In the bony canal, the facial nerve is usually involved as a result of disease of the ear. In children this is much more frequent than from the other causes just mentioned. While it occasionally occurs with acute otitis, it generally accompanies the chronic form with caries of the petrous bone, which in our experience is very often tuberculous. In addition to the paralysis there is present or there is a history of a discharge from the ear, and generally there is some deafness upon the side affected. The facial symptoms are usually the same as in the cases first described. However, when the nerve is affected between the stape-

dius and the geniculate ganglion, there is a disturbance of the sense of taste, and of the secretion of saliva. Facial paralysis may also occur as a result of injury to the nerve during the mastoid operation.

At the base of the brain the trunk of the nerve may be involved in cerebral tumor, basilar meningitis, and in fracture of the skull. In any of these conditions the auditory nerve also is likely to be affected. A not infrequent cause of central paralysis is poliomyelitis. Facial paralysis occurs in the cerebral form with hemiplegia, or more commonly it is associated with paralysis from a spinal lesion. Occasionally the facial nerve alone may be involved. The whole nerve may be affected or only one of its branches.

Prognosis.—The result is greatly modified by the causes in the different cases. In those which are due to cold, spontaneous recovery usually occurs in the course of a few weeks or months. In those depending upon disease of the ear, the outlook is not so favorable, and though there may be improvement, it is not rare for some paralysis to be permanent. In the third group of cases, facial paralysis is only one of the symptoms, and the result depends entirely upon the nature of the cause. In poliomyelitis the prognosis is good though in some cases a certain degree of paralysis may remain.

Diagnosis.—Facial paralysis is easily recognized. It is important to separate the peripheral paralysis from that due to a lesion above the pons, as in cases of ordinary hemiplegia. In the latter group only the lower half of the face is affected, the muscles of the forehead and those about the eye escaping, and the electrical reactions are unchanged.

Treatment.—This is essentially the same as in other cases of neuritis. In cases due to ear disease the primary lesion should receive appropriate treatment.

SECTION VIII

DISEASES OF BLOOD, LYMPH NODES, DUCTLESS GLANDS, BONES, AND JOINTS

CHAPTER I

DISEASES OF THE BLOOD

THERE are several particulars in which the blood of infancy and early childhood differs from that of older persons.

Specific Gravity.—This has no constant relation to the number of white or red corpuscles, but varies with the amount of hemoglobin. The highest specific gravity is seen in the blood of the newly born. During the first two weeks of life it sinks rapidly to its lowest point, where it remains until about the end of the second year; after this time it rises gradually until about puberty. The average specific gravity during childhood is 1.050 to 1.055.

Hemoglobin.—The percentage of hemoglobin is highest in the blood of the newly born, and falls rapidly during the first few days after birth. Throughout childhood it is considerably lower than in adult life. The hemoglobin is lowest between the third month and the second year; after the second year it gradually increases up to puberty. The usual range in young children, as measured by the adult standard, is between sixty-five and eighty-five per cent, seventy-five per cent being a low limit in healthy children.

Red Corpuscles.—The number of red corpuscles is highest in the newly born. At this time it is from 4,350,000 to 6,500,000 in each cubic millimeter. In infancy it is from 4,000,000 to 5,500,000; in later childhood, from 4,000,000 to 4,500,000 (Hayem). In size a much greater variation is seen in the red cells of the newly born than in those of older children and adults. In the blood of the fetus there are present nucleated red corpuscles or normoblasts (Plate XII, A). These diminish in number toward the end of pregnancy. They are always found in the blood of premature infants, but in infants born at term they are seen only in small numbers and disappear after a few days. In later infancy their presence is always pathological.

Normal White Cells.—The following varieties are found in health:

1. *Lymphocytes.*—These are small cells about the size of a red blood cell. The protoplasm is small in amount, forming merely a narrow rim about the nucleus; it stains with basic dyes rather more deeply than does the nucleus. The nucleus is relatively large, is centrally situated, and shows at times one or two nucleoli. The protoplasm may have a reticular structure. These cells form in adults from twenty-two to twenty-five per cent of the white corpuscles, but in young children they are often as high as fifty or sixty per cent (Plate XII, B, 10).

2. *Large Mononuclear Leucocytes and Transitional Forms.*—These cells are two or three times the size of ordinary red cells (Plate XII, D, 10). The oval nucleus is not so centrally situated as in the lymphocytes, and stains feebly but rather more deeply than the protoplasm, which is poorly stained by basic dyes. The protoplasm is homogeneous and relatively large in amount.

The transitional forms occasionally contain a few feebly staining neutrophilic granules; their nuclei are bent or curved and stain more deeply.

3. *Polymorphonuclear Neutrophiles.*—These are smaller than the large lymphocytes (Plate XII, B and C, 8). The nucleus consists of three to four parts, usually connected by narrower portions, and stains darkly. The protoplasm stains with acid dyes and shows a great number of granules which stain only with neutral dyes. In adults these cells form about seventy per cent of the white cells; but in children they are less numerous, the increase in the lymphocytes being at the expense of the neutrophiles.

4. *Eosinophiles.*—These are about the same size as the neutrophiles (Plate XII, C, 9); they have deeply staining nuclei, usually divided into two parts. The protoplasm has many large granules that stain deeply with acid dyes, and often a narrow outer layer stains more deeply than the rest. They form from one to two per cent of the total number of white cells.

5. *Mast Cells.*—They are only occasionally found, their proportion being about 0.5 per cent of the white cells; they are polymorphonuclear cells whose granules stain only with basic dyes, not at all with tri-acid; often they are metachromatic (Plate XII, C, 12).

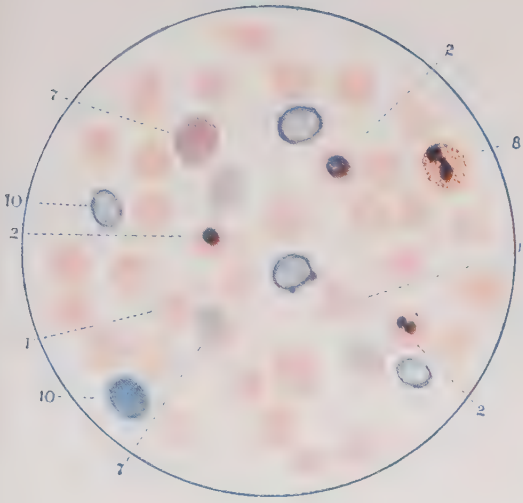
Pathological White Cells.—Of these there are three principal forms:

1. *Myelocytes, neutrophilic.*—They have neutrophilic granules and a single rounded nucleus (Plate XII, C, 11). Ehrlich's myelocytes differ from those of Cornil in that the cells as a whole are smaller, the nuclei are more centrally situated and stain more deeply.

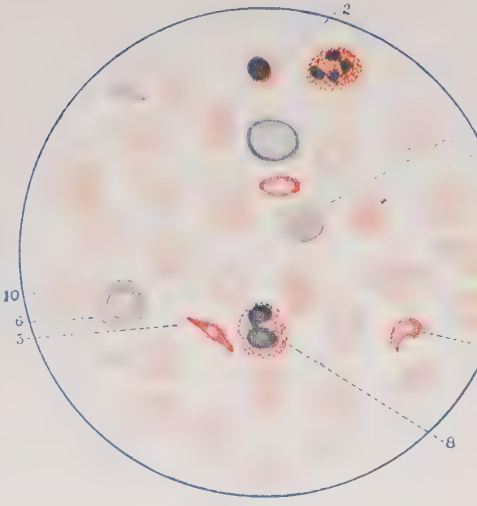
2. *Myelocytes, eosinophilic.*—These resemble the polynuclear eosinophiles, except for the round, undivided nucleus.

3. *Myelocytes, basophilic.*—These are similar to the mast cells, differing only in the form of the nucleus.

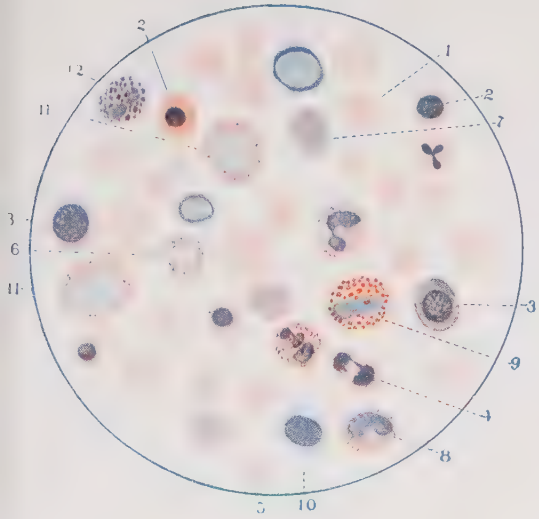
PLATE XII



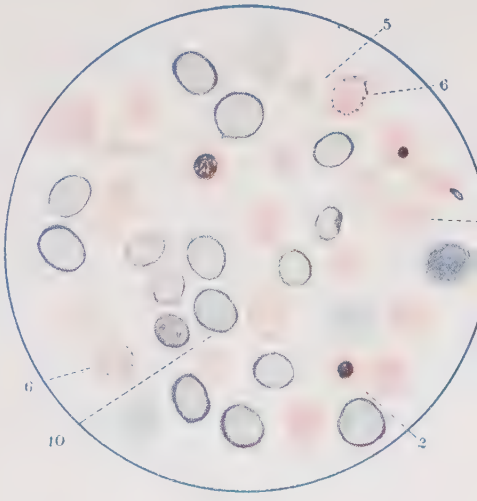
A.



B.



C.



D.

Drawn by Dr. F. C. Wood

A. Blood of an Eight-Months' Fetus.

B. Simple Anemia.

C. von Jaksch's Anemia.

D. Acute Lymphatic Leukemia.

1. Red cells, normal.
2. Red cells, normoblasts.
3. Red cells, megaloblasts.
4. Red cells, showing mitosis.
5. Red cells, poikilocytes.
6. Red cells, granular degeneration.
7. Red cells, polychromatophilia.
8. White cells, polynuclear neutrophiles.
9. White cells, eosinophiles.
10. White cells, lymphocytes.
11. White cells, myelocytes.
12. White cells, mast cells.

These myelocytes all represent immature forms, originating in the bone marrow. Pathologically, these may be immature forms of the leucocytes, or these may undergo acute or chronic degeneration, with swelling and fragmentation, nuclear changes, hydropic degeneration, etc.

The number of leucocytes in the blood of the newly born, according to Rieder, is at birth from 14,200 to 27,400 per cubic millimeter; from the second to the fourth day, from 8,700 to 12,400; after the fourth day, from 12,400 to 14,800. The normal variations in infancy are from 9,000 to 18,000, and in later childhood from 8,000 to 12,000.

SECONDARY ANEMIA

This consists in an impoverishment of the blood, especially the red cells, and a corresponding diminution in the specific gravity, and in a greater proportional decrease in the amount of hemoglobin. It occurs apart from disease of the blood-making organs. Infancy and childhood are themselves strong predisposing causes of anemia on account of the great demands made upon the blood in the rapid growth of the body.

Etiology.—The causes of anemia embrace a wide range of pathological conditions. A child born of a delicate mother or of one suffering from tuberculosis or syphilis may show a marked anemia at birth. It sometimes occurs in the first two or three months of life in a severe form without any discoverable cause. It may follow any severe hemorrhage or occur in any of the blood dyscrasiae—purpura, scurvy, etc. It accompanies any prolonged infection with or without suppuration, also nephritis, many forms of gastro-intestinal disease and malignant growths. It is especially marked in general sarcomatosis. Certain of the specific infections, notably diphtheria, malaria, tuberculosis and rheumatism, produce a marked degree of anemia as one of their effects. It is found with great severity with some of the intestinal parasites, particularly varieties of the tape-worm and hook-worm. Anemia is at times due to mineral poisons—lead, mercury or potassium chlorate.

Much more frequent in young children than any of the above are the anemias due to improper feeding, rickets, and unhygienic surroundings. How important these causes are and how severe a grade of anemia may be produced by them, is not usually appreciated. The physician is often led to suspect some serious organic or constitutional disease when none exists, and to overlook such common conditions and obvious causes as those mentioned. Anemia is seen when lactation is unduly prolonged. It is a frequent result of an exclusive diet of milk or

infant foods into the second or third year. Older children who drink tea and coffee and eat largely of indigestible food, pastry, cake, etc., are frequently anemic. Lack of fresh air, confinement to overheated rooms and the crowding of young children in hospitals and institutions, are common and important causes of anemia.

Symptoms.—Anemic children usually exhibit many symptoms of malnutrition. Their tissues are flabby; they are generally below average weight and suffer from digestive disturbances and chronic constipation. The associated nervous symptoms are many; headaches, indefinite pains, insomnia or disturbed sleep, general irritability and a high degree of nervousness. There is easy fatigue, shortness of breath on exertion, and sometimes fainting attacks. The peripheral circulation is poor; the hands and feet are often cold. The pulse may be slightly irregular. Murmurs may be heard over the base of the heart or the large vessels, and so loud even in infancy as to be mistaken for organic disease. A venous hum may be heard in the neck. Epistaxis is not uncommon. There may be enuresis. Edema is rare in older children, but in severe anemias of infancy it is sometimes marked. In a certain number of cases, even of moderate severity, the spleen is much enlarged. Pallor of the skin and mucous membranes is present in most cases, but is not an accurate guide as to the degree of anemia. This can only be determined by an examination of the blood.

The Blood.—There is a reduction of the number of red cells and to a still greater degree in the hemoglobin. In a case of moderate severity the red cells are from 3,500,000 to 4,000,000, and the hemoglobin from fifty to sixty per cent. In severe cases the red cells may fall to 2,000,000 or 2,500,000 or even lower, and the hemoglobin to twenty or thirty per cent. These figures are not uncommon. Occasionally there is seen a reduction of the hemoglobin to as low as fifteen per cent and of the red cells to 1,500,000. The red cells are pale. There is usually poikilocytosis and anisocytosis; and, especially in infancy, a few normoblasts and megalocytes may be found (Plate XII, B).

There is generally a slight leucocytosis. The differential count of the white cells shows an increase in the lymphocytes, chiefly the small variety; the polymorphonuclear cells are relatively reduced in number.

Prognosis.—The course and termination of anemia depend upon its cause. If this is one that can be removed, as in cases depending upon improper feeding and surroundings, very rapid improvement often takes place and prompt recovery. In the most severe cases death may occur, rarely from the anemia, usually from some complicating disease.

In making a prognosis in a given case the general symptoms and the cause of the anemia are much more important than the examination of the blood. If the digestive organs are in good condition and good

surroundings can be secured, even though the hemoglobin and red cells are very greatly reduced, the prognosis is good. But in unfavorable surroundings and with a greatly disordered digestion, the outlook is more serious.

Typical blood examinations of a moderate and of a severe case of secondary anemia in a young child are as follows:

Severe Anemia		Moderate Anemia	
Hemoglobin	20 per cent.	Hemoglobin	50 per cent.
Red blood cells	2,500,000	Red blood cells	4,000,000
White cells	12,000	White cells	10,000
Polymorphonuclear	30 per cent.	Polymorphonuclear	40 per cent.
Small mononuclear	45 per cent.	Small mononuclear	25 per cent.
Large mononuclear	25 per cent.	Large mononuclear	20 per cent.
Other forms	5 per cent.	Other forms	5 per cent.

The treatment of all the forms of anemia will be considered together at the close of the chapter.

CHLOROSIS

Chlorosis usually occurs in young girls about the time of puberty. It is characterized by a peculiar greenish-yellow tint of the skin, and is not accompanied by emaciation. The changes in the blood consist in a very great reduction in the hemoglobin without a corresponding diminution in the red corpuscles.

Etiology.—The exact cause of chlorosis is not yet understood. The disease rarely occurs in males; it is usually seen in girls between the fourteenth and seventeenth years, and more often in blondes than in brunettes. Heredity appears to be a factor in some cases. Other causes are occupations deleterious to health, such as employment in factories or confinement in ill-ventilated rooms; insufficient food or clothing; psychical disturbances, like grief, care, or fright; excessive mental or physical strain, and disorders of menstruation—although the latter are perhaps more frequently a result than a cause of the disease.

Lesions.—Chlorosis is not a fatal disease. In the few cases with chlorosis that have died of other diseases the lesions noted have been dilatation of the right heart with hypertrophy of the left ventricle, a small aorta, small uterus and ovaries, and occasionally round ulcer of the stomach. Under the microscope there may be found a very marked degree of fatty degeneration of the heart muscle, and sometimes of the inner coat of the blood-vessels.

Symptoms.—The general symptoms of chlorosis are very much like those of simple anemia. There are observed shortness of breath upon exercise, palpitation, syncope, attacks of vertigo, disturbances of diges-

tion, amenorrhea, and almost invariably constipation. The appetite is capricious, it being a peculiarity of these patients to crave all sorts of indigestible articles. Instead of the usual pallor of anemia, the skin has a yellowish-green tint, from which the term "green-sickness" has arisen. Occasionally patches of pigmentation are seen. Anemic cardiac murmurs may be heard in various situations, most frequently a systolic murmur at the base of the heart, and usually loudest over the pulmonic area. There may be a venous hum in the neck. In some marked cases there is evidence of slight cardiac dilatation, especially of the right heart, and there may be hypertrophy of the left ventricle. The pulse is weak and soft, edema of the feet is frequent, and sometimes there is slight albuminuria. In some cases there is fever. Nervous disturbances, such as vague, indefinite pains, attacks of migraine, supra-orbital neuralgia, various hysterical manifestations, and chorea, are common. Ulcer of the stomach is sometimes seen as a complication.

The Blood.—The specific gravity is reduced in proportion to the loss of hemoglobin. The characteristic feature of chlorosis is a loss of hemoglobin which is out of proportion to the reduction in the red cells. The hemoglobin in an ordinary case is frequently as low as thirty-five or forty per cent, while the red cells may be 3,500,000 to 4,000,000, or even higher.

Morphologically the cells are pale with a wide central clear area. Poikilocytosis may be present, but is not marked; rarely normoblasts may be found. The presence of megalocytes is disputed. The leucocytes are usually unchanged in number and proportion, but the lymphocytes may be relatively increased.

Prognosis.—The course of the disease is essentially a chronic one, often lasting for a year. Relapses are quite frequent. These cases regularly recover when proper treatment can be carried out.

Diagnosis.—A probable diagnosis is in most cases easily made from the etiology, the functional derangement of the heart, the color of the skin, and a positive diagnosis always by an examination of the blood.

PSEUDO-LEUKEMIC ANEMIA OF INFANCY
(*Von Jaksch Disease*)

This form of anemia was first described by von Jaksch in 1889, and is by him believed to be peculiar to infants and young children. It is characterized by marked leucocytosis, marked reduction in the number of red cells and in the hemoglobin, a great enlargement of the spleen, and sometimes a moderate enlargement of the liver and the lymphatic glands. This disease is not to be confounded with pseudo-leukemia or Hodgkin's disease.

The existence of pseudo-leukemic anemia as a distinct disease is denied by most authorities on diseases of the blood, who regard it as a symptom-complex. They hold that the reported cases can be classed either as severe secondary anemia, pernicious anemia, or leukemia.

Etiology.—Of the cases thus far recorded the majority have been between the ages of seven and twelve months. Of twenty cases collected by Monti and Berggrün, sixteen showed evidences of rickets and one was syphilitic. The exact cause of the disease is still unknown, and its essential nature is a matter of some doubt.

Lesions.—The most characteristic change is found in the spleen, which is very much enlarged, often forming an abdominal tumor of considerable size. It is firm, hard, and there may be evidences of perisplenitis. The microscope shows a simple hyperplasia. Enlargement of the liver is less constant, it being normal in more than half the cases. There is no relation between the size of the spleen and that of the liver. The hepatic cells are unchanged. Enlargement of the lymph glands has been noted in about half the reported cases, the swelling affecting the cervical, axillary, or inguinal glands; but it is rarely great. It is due to simple hyperplasia. Inconstant changes in the bone-marrow have been described.

Symptoms.—*The Blood.*—The main features noted are the following (Plate XII, C):

The specific gravity is lowered, the usual range being between 1.035 and 1.044. The reduction of the hemoglobin is very great; in many of the cases it has been as low as twenty-five per cent, and in a few below twenty per cent.

The red cells are always diminished; they are frequently below 2,000,000. There is also great inequality in their size and shape. Nucleated red cells are found in considerable numbers; as a rule, these are chiefly normoblasts, but when the anemia becomes more severe, it is usually the megaloblasts that predominate. The leucocytes vary from 20,000 to 50,000. They may show an increase in the mononuclear or in the polymorphonuclear forms. The eosinophiles are usually increased, but not to the extent to suggest leukemia. All varieties of cell degeneration are found.

The general symptoms of the disease develop slowly and with the usual signs of anemia. In some cases the infants continue to be plump and well nourished. Pallor is usually very marked. Enlargement of the spleen is so great that it can hardly be overlooked if the abdomen is examined. The glandular enlargements are not marked, and in many cases are wanting altogether.

The course of the disease is essentially chronic. Cases have been seen in which pseudo-leukemia developed from an ordinary severe simple

anemia in the course of a few weeks. The symptoms and blood changes generally come on slowly in the course of weeks or months, and sometimes remain nearly stationary for as long a period as several months, and then slowly improve. In other cases they grow gradually worse. In the cases going on to recovery there is noticed improvement in the general symptoms coincident with a diminution in the size of the spleen, a reduction in the number of leucocytes, an increase in the red cells, the hemoglobin, and the specific gravity, and a gradual disappearance of the nucleated red cells.

Prognosis.—In Monti's list of twenty cases, four proved fatal; one recovered, in which the proportion of leucocytes to the red cells had been one to twelve. The prognosis should be guarded, for, although improvement may take place, many patients die from intercurrent disease.

PERNICIOUS ANEMIA

This is the most severe form of anemia known. Its cause and essential nature are as yet very imperfectly understood. It is characterized by quite uniform blood changes and by the general symptoms of a very marked anemia, and it tends to go on from bad to worse, terminating fatally in the great proportion of cases.

Etiology.—Pernicious anemia is a rare disease in childhood, and especially rare in infancy. Its essential cause is quite unknown. In a few instances intestinal parasites, particularly tapeworms, have produced in children an anemia indistinguishable from pernicious anemia.

Lesions.—There is found a very high grade of anemia in all the internal organs, fatty degeneration of the heart and blood-vessels, and sometimes also of the liver and kidneys, with numerous capillary hemorrhages in the various organs. A striking post-mortem change consists in the deposit of iron in the hepatic cells. This is found, however, with other severe forms of anemia. Its distribution is peculiar and unlike that seen in most other diseases. The bone marrow is also markedly altered in that the red cells may be of the megaloblastic instead of the normoblastic type. In aplastic anemia there may be a yellow bone marrow instead of the normal red bone marrow of childhood.

Symptoms.—*The Blood.*—The specific gravity of the blood in pernicious anemia is constantly and considerably reduced, and its coagulability is feeble. The hemoglobin is always reduced; usually it is as low as from twenty to thirty per cent. The red cells are always much diminished in number and to a degree greater than the reduction in the hemoglobin. Their number is seldom greater than 2,000,000, and

frequently less than 1,000,000. Megalocytes are present, often in great numbers, and a preponderance of them is regarded essential to the diagnosis. Microcytes are rare. It is characteristic of pernicious anemia that owing to the relatively high hemoglobin content the red cells have a high color index and stain well, usually deeper than in normal blood. A striking feature of these cases is the presence of extreme poikilocytosis. Nucleated red cells are also present, megaloblasts in greater numbers than normoblasts. The red cells do not readily collect to form rouleaux. The blood platelets are greatly reduced and frequently almost absent.

The total number of leucocytes is markedly diminished, but the lymphocytes may be relatively increased. An occasional myelocyte may be found.

The general symptoms are those of a most intense anemia. There is marked pallor of the skin and mucous membranes, with great weakness and prostration. Various accidental heart murmurs are heard. There may be dyspnea. There may or may not be emaciation. The late symptoms are hemorrhages from the nose and other mucous membranes, subcutaneous ecchymoses with dropsy of the feet and ankles, and sometimes of the large serous cavities of the body, but without albuminuria. In many cases fever is present. This may be so high as to lead to the suspicion of some acute infectious process.

The course of the disease is, as a rule, more rapid than in adults, the duration being in most cases but a few months; it is marked by periods of exacerbation and remission. During the exacerbations all the symptoms are intensified, and as a rule some fever is present. During the remissions marked improvement may take place in all the symptoms and an increase in the hemoglobin and red cells occurs. In general, the progress of the disease is downward and sometimes the rate is very rapid. The only exceptions are the cases in which the disease depends upon some intestinal parasite, when improvement and even recovery may occur.

Treatment of the Different Forms of Anemia.—In *secondary anemia* the thing of the first importance is to discover and treat the primary condition upon which the anemia depends. In infancy, special attention should be given to diet and hygiene. A mixed diet composed of fruits, beef juice, eggs and green vegetables should be substituted for one consisting mainly or exclusively of milk. Also important is an abundant supply of fresh air. The whole manner of life of these patients must be carefully studied and managed according to the directions laid down in the chapter upon Malnutrition, with which condition, especially in infancy, a very large number of these cases are associated. The general treatment referred to is often more important than the

administration of the preparations of iron, which, however, should never be omitted.

The preparations of iron especially adapted to infants are the albuminate, bitter wine, sweet wine, saccharated carbonate, malate, and citrate. The dose should be regulated according to the age of the child. Older children may take the same preparations as adults, especially reduced iron and Blaud's pills. Much benefit is seen from combining arsenic with iron, or from alternating the two. In addition to these remedies, cod-liver oil should be given if the condition of the digestive organs will permit.

In *chlorosis* more decided results are seen from the use of iron than in any other form of anemia. Blaud's pills are here the favorite form of administration, and are advantageously combined with small doses of nux vomica and aloin to overcome the tendency to constipation. Arsenic is useful in these cases also. Great benefit in chlorosis results from change of air and change of scene, thus removing the patient from all sources of nervous excitement or disturbance. The general condition, diet, and habits of life should also receive careful attention, particularly the condition of the bowels.

It is important that the administration of iron should be continued for some time after the disappearance of all symptoms, on account of the tendency to relapse.

In *the pseudo-leukemic anemia* of infants, arsenic is decidedly the most valuable drug, but should be given in combination with iron. Fowler's solution is the best preparation for infants; the dose should rarely be more than one drop, which should be repeated four or five times daily after feeding, and continued for a long time. The general treatment of these patients is the same as in cases of simple anemia. When rickets is present cod-liver oil and phosphorus should be added.

In *pernicious anemia*, arsenic offers a much better prospect of improvement than does iron. Beginning with small doses, the amount should be gradually increased up to the point of tolerance, very much as in cases of chorea.

In every case of anemia the most careful attention should be given to the general condition, particularly guarding against exposure to cold and dampness. The feeble circulation of these patients renders them peculiarly susceptible. Caution should also be given against much muscular exercise.

In cases of secondary anemia transfusion is a remedy of the greatest value. In acute anemia following loss of blood its effects are little short of marvelous. In the primary anemias and in pernicious anemia its effects are much less evident and in the great majority of cases only temporary improvement is seen.

LEUKEMIA

This is a disease in which the essential feature is a great increase in the number of leucocytes, with a moderate reduction in the number of red corpuscles, and the presence in the blood of cell forms not found in health.

Etiology.—Leukemia is a rare disease in childhood, but it is seen even in early infancy. Its greater frequency in males holds good even in childhood. In a small number of cases heredity has been noted. Leukemia may follow syphilis, rickets, malaria, or even simple anemia, or it may occur apparently as a primary disease in children previously healthy. The cause is unknown.

Lesions.—The essential lesions of leukemia are found in the spleen, the lymphatic glands, and the bone-marrow. In some cases the most important changes are in the lymphatic glands, giving rise to the lymphatic form of leukemia. Any of the external glands of the body may be affected—the cervical, axillary, and the inguinal, or the mesenteric, tracheobronchial, the tonsils, and even the lymph nodules of the tongue, pharynx, and intestines. The changes in the glands are generally those of a simple hyperplasia. The liver is enlarged in most of the cases, chiefly from an infiltration with lymphoid tissue, which may be diffuse or may occur in patches. Less frequently similar lymphoid masses are seen in other organs. Lesions may be present in almost any of the viscera due to secondary infections. In lymphatic leukemia the changes in the spleen and marrow may be slight. Changes of a severe form in the spleen and marrow are, however, usually seen together, giving rise to what is known as the *splenomyelogenous* form of the disease. The spleen is usually enormously enlarged, sometimes filling half the abdominal cavity. In the early stage it is soft, vascular, and of a dark-red color; in the late stages it is firm and hard. There may be perisplenitis. On section, light-gray patches of lymphoid tissue may be seen scattered throughout the organ, and in some instances there may be wedge-shaped infarctions. The microscope shows thickening of the trabeculae and deposits of lymphoid tissue, especially about the arteries.

Symptoms.—In *acute lymphatic leukemia,* which in our experience, is the most common form of leukemia in early life, the symptoms are so severe and the progress so rapid as to suggest an acute infection. It is often preceded by some other infection such as pneumonia, multiple abscesses or inflammation of the tonsils. The onset may be abrupt with severe symptoms—fever, general and articular pains and great prostration, but not much that is definite; or it may be more gradual with only

local symptoms for several weeks. The swelling of the external lymphatic glands may be the first thing noticed; this is most marked usually in the cervical region, but the axillary, inguinal, femoral and epitrochlears may also be involved. The individual glands may be no larger than an almond, but often reach the size of a walnut. There is no redness and seldom tenderness. The glandular swelling is usually progressive; the spleen and liver soon become large and hemorrhages often occur. These may be subcutaneous in the form of small petechiae or larger purpuric areas, or there may be bleeding from the nose, the bowels, the bladder, or blood may be vomited. The mouth often is the seat of disease resembling scurvy. In fact, these symptoms may dominate the clinical picture. The gums are much swollen and bleed easily; there may be sloughing in the gums, tonsils or buccal mucous membrane. The submaxillary glands are swollen and there is much local pain and discomfort. The general symptoms at this stage are usually severe. The temperature is nearly always somewhat elevated and it may be as high as 103° or 104° F.; there is marked dyspnea and great muscular weakness; the pulse is rapid and feeble and the loss of weight usually marked.

The blood picture varies greatly in the different cases and in the same case at different stages of the disease. The constant feature is the great relative increase in the lymphocytes, which usually form from 90 to 98 per cent of the white cells, and a corresponding reduction in the polymorphonuclear cells. The lymphocytes are chiefly of the large variety and many of them are degenerated so that they stain with difficulty. The total leucocytes in the early stage may not be increased and there may even be a leucopenia—3,000 or 4,000. Sometimes the total leucocytes fall greatly toward the end of the disease; but generally they are increased, numbering from 50,000 to 150,000; the red cells are uniformly reduced in number to from 1,000,000 to 3,000,000 and the hemoglobin to twenty or thirty per cent or even lower. The coagulability of the blood is diminished. The course of this form of the disease is usually rapid. It may last only three or four weeks, and rarely more than two or three months. Death is due to hemorrhages, to exhaustion, or to some acute intercurrent infection.

Other cases run a less acute course and may be marked by irregular and prolonged attacks of fever which in some cases may be high and last for months, but with few other symptoms except enlargement of the lymphatic glands. The blood picture varies much from time to time, the constant feature being the high percentage of lymphocytes and a moderate degree of anemia. The total leukocyte count may be low for a long period but a marked relative increase in the lymphocytes is a constant feature. The chronic form of lymphatic leukemia does not

differ greatly from that in the adult but in our experience is very uncommon in children.

In the *splenomyelogenous* form of the disease the progress is usually less acute and resembles that seen in the adult, but its course is always more rapid in early life. In the case reported by Knox, death occurred two weeks after the first symptoms. In most of the cases the early symptoms are latent. A sudden and alarming hemorrhage is sometimes the first thing to call attention to the serious condition. In other cases there are only the symptoms of general weakness and anemia. Sometimes the splenic tumor is the first thing noticed. In the early part of the disease the usual symptoms of anemia are present—digestive disturbances, shortness of breath, weak and rapid pulse. Hemorrhages may occur as an early or late symptom; they are most frequently from the nose, but severe hemorrhages may occur from the stomach, the mouth, the intestines, or there may be ecchymoses upon the skin. The enlargement of the spleen may be sufficient to form an abdominal tumor, so as to attract the attention even of the parents. The swelling of the liver is not so great. The lymphatic glands are enlarged only to a moderate degree, and in many cases this symptom is absent altogether. They are painless, movable, and usually several groups are affected.

The late symptoms are dropsy of the feet or general anasarca, hemorrhages, diarrhea, headaches, general weakness, and attacks of syncope. Fever is quite constant in the late stages of the disease, and the temperature may be from 101° to 103° F. The urine may contain albumin and casts. Vision is sometimes disturbed by the formation of leukemic plaques in the retina. It is rare that there are any symptoms referable to the bones, although expansion and tenderness of the flat bones have been observed.

In the splenomyelogenous form the white cells may be from 100,000 to 500,000, but, especially under the influence of arsenic, a marked temporary diminution may occur, so that their number may be scarcely above the normal; both Ehrlich's and Cornil's myelocytes are present, and the presence of a large number of these is pathognomonic. The number of polymorphonuclear neutrophiles is greatly increased, although their proportion is diminished. The eosinophiles are very much increased in number, mononuclear forms being present. The number of lymphocytes is increased, but they vary according to the type and stage of the disease; basophilic (mast) cells, both mononuclear and polymorphonuclear, are present in considerable number, this being the most reliable diagnostic sign.

Prognosis.—The prognosis of leukemia of all varieties in children is very bad, nearly all cases terminating fatally within a few weeks or

months from the first definite symptoms. The usual causes of death are exhaustion, hemorrhages, and bronchopneumonia.

Diagnosis.—The general symptoms are likely to be misleading, especially fever, dyspnea and prostration. The buccal symptoms frequently suggest scurvy. A rapid general enlargement of the external lymphatic glands always is suspicious, but without a blood examination, a diagnosis is impossible. The chief reliance is to be placed in cases of lymphatic leukemia upon the great relative increase in the lymphocytes and reduction in the polymorphonuclears more than upon the total number of leucocytes; in other cases the diagnosis rests upon the enormous increase in the leucocytes, and especially upon the presence of numerous mast cells and neutrophile and eosinophile myelocytes.

Treatment.—Leukemia is little influenced by treatment. The reported cures must be taken with some allowance, for most of these were published before leukemia was sharply differentiated from simple anemia with leucocytosis. Temporary improvement in some cases has followed the use of arsenic in full doses. Hemorrhages may be relieved at times by calcium lactate. Benzol is often of distinct value in the treatment of chronic splenomyelogenous leukemia. Its effect is to diminish markedly the number of white cells, especially those developed from the bone marrow. The total number of leucocytes may be reduced from a hundred thousand or more to less than ten thousand. In giving benzol, care should be observed that the reduction does not take place too rapidly. As the effect is continuous for a time after the drug is omitted, no more should be given after the total number of cells is 15,000 or 20,000. Benzol may be given in capsules beginning with seven or eight grains (gram 0.5) once a day. The dose may be increased gradually, depending upon its effect, but should not exceed thirty to thirty-five grains (gram 2.5).

Coincident with the fall in the number of white cells there is usually a marked increase in the red cells and a great amelioration of the patient's condition. This is unfortunately not permanent though it may last for many months. Subsequent courses of treatment with benzol bring less improvement, or may be without influence. Striking improvement has followed transfusion, but this is usually only temporary. In the great majority of cases the disease goes on to a fatal termination in spite of the measures employed.

HEMOPHILIA

Hemophilia is an hereditary disease, in which there is a tendency to profuse or even uncontrollable bleeding from slight wounds. The

hemorrhage may even be spontaneous. Persons so affected are known as "bleeders."

Etiology.—The hereditary tendency of the disease is very strongly marked, and it has often been traced through seven or eight generations. Males are much more frequently affected than females, the proportion being about twelve to one. In the matter of inheritance, the disease is most often transmitted through the mother, who, however, usually escapes herself. Patients suffering from hemophilia may have nothing else about them that is abnormal. It has no connection with either purpura or scurvy. Howell, from his extensive studies upon hemophilia, has come to the conclusion that it is due to a relative preponderance of antithrombin. The antithrombin may be normal in amount or absolutely increased but on account of the absolute diminution in the prothrombin there is always a relative increase in the factors that delay the coagulation of blood.

Symptoms.—The first manifestations of hemophilia are not often seen before the second year. The hemorrhages of the newly born have no relation to this condition. The discovery of the disease is generally quite accidental. The first hemorrhage may be traumatic or spontaneous. In traumatic hemorrhages there may be very severe bleeding after so slight a wound as the drawing of a tooth; sometimes a large hematoma forms between the muscles as the result of a moderate contusion.

The following is the relative frequency of spontaneous hemorrhages in 334 cases collected by Grandidier: Bleeding from the nose in 169, mouth in 43, intestines in 36, stomach in 15, urethra in 16, lungs in 17. There may be hemorrhage from the skin or from any mucous membrane of the body. The attacks of spontaneous hemorrhage are often periodical, and may be accompanied by arthritic symptoms resembling rheumatism. There are hemorrhages into the joints in some instances with severe resulting deformity.

The severity of the hemorrhages varies much in the different cases. From a slight wound a patient may bleed until he is exsanguinated, and even until death occurs. Such a result from the first hemorrhage, however, is rare. In some cases the disposition to bleed is outgrown in later life. Grandidier states that, of 152 boys, over one-half died before reaching the seventh year. It is striking that when the disease affects females there is no tendency to excessive bleeding at menstruation or parturition.

Treatment.—The indications at the time of bleeding are, to arrest the hemorrhage by the use of the ordinary surgical means—especially compression. Calcium lactate and gelatine may be used as described in the hemorrhages of the newly born; but little benefit is to be ex-

pected from drugs. In all marked cases transfusion should be practiced. Its effects are sometimes very striking. In convalescence after attacks of hemorrhage, iron and general tonics should be given. In all patients who are bleeders everything which might by any means excite hemorrhage should be avoided.

PURPURA

The term purpura is used to designate a condition in which there is a tendency to spontaneous hemorrhages beneath the skin, from the various mucous membranes, and in some cases into the internal organs. The term *purpura simplex* is applied to those cases in which the hemorrhages are limited to the skin; *purpura hemorrhagica* to those in which there is in addition bleeding from the mucous membranes or visceral hemorrhages. It is impossible to draw a line sharply between these two classes of cases, as the chief difference between them seems to be one of degree. Purpura is sometimes known as *morbus maculosis* or as *Werlhof's disease.*

Symptomatic Purpura.—This occurs in quite a variety of conditions, the hemorrhages generally being limited to the skin, but not always so. These cases may be grouped in the following classes:

1. *Infectious.*—This form of purpura is very constantly seen in malignant endocarditis, in the hemorrhagic forms of the various eruptive fevers—measles, scarlet fever, variola, vaccinia, and typhus—also in epidemic meningitis and occasionally in diphtheria, pyemia, and septicemia. The occurrence of hemorrhages in these cases appears to depend upon an altered condition of the blood-vessels, which is a direct result of the infection, and it is a bad prognostic sign.

2. *Cachectic.*—Purpura occurs late in the course of many protracted and exhausting diseases, especially in infancy. It is most frequently met with in bronchopneumonia, empyema, tuberculosis, ileocolitis, in both the tuberculous and the simple forms of meningitis, and in malignant disease. It also occurs from apparently similar causes in several of the diseases of the blood, particularly in leukemia and pernicious anemia. In most cases of cachectic purpura the hemorrhagic spots are small, not very abundant, and occur either upon the abdomen or the lower extremities. This form is quite common in hospital practice, and is almost invariably indicative of a fatal result. In cachectic purpura the hemorrhages are usually limited to the skin.

3. *Toxic.*—Certain drugs, such as phosphorus, quinin, potassium chlorate, and sometimes others, may in rare cases produce hemorrhages when long continued or in large doses. The hemorrhage of jaundice may also be considered in this group.

4. *Mechanical* hemorrhages, such as those occurring in pertussis or epilepsy, are sometimes classed with purpura. In convalescence from protracted illness there are sometimes seen, when patients first stand or walk, purpuric spots on the lower extremities. They may occur after the confinement of a limb in bandages or splints. In both these cases the cause is partly mechanical and partly due to the weakened condition of the blood-vessels.

5. *Neurotic.*—These cases are occasionally seen in diseases of the spinal cord and sometimes in hysteria in young adults, but very rarely in children.

Primary Purpura.—This occurs in children of all ages, being not uncommon in infancy. Hemorrhages of the newly born have not generally been included in this class. The age at which primary purpura is most frequently seen is from two to ten years. The sexes are about equally affected; of Steffen's 56 cases, 27 were males and 29 females. The disease may occur in children who are cachetic, rachitic, or anemic, and in those whose surroundings are poor, but it has not, like scurvy, any close relation to diet. It may follow any acute disease, being associated most frequently with derangements of the stomach and bowels. Quite often the disease develops abruptly, without any assignable cause, in children previously healthy.

Lesions.—The external hemorrhages may occur upon any part of the body. There are smaller or larger ecchymoses or an infiltration of the tissues with blood, which undergoes gradual absorption with the usual changes. With the hemorrhages, various forms of inflammation of the skin may be associated, especially erythema and urticaria, with sometimes more or less edema. Hemorrhages from the mucous membranes are more frequent, because of the feebler resistance of the tissues. There are seen ecchymoses upon the visible mucous membranes which resemble those upon the skin. At autopsy they are occasionally seen in the trachea or bronchi, but more often in the digestive tract. In the colon, and occasionally in the small intestine, ulcers may be found; but they are rarely, if ever, seen in the stomach. They may be superficial or deep, and have even been known to cause perforation.

Intracranial hemorrhages are rare, and are usually meningeal. These may be sufficient to cause severe symptoms. We have seen an instance in an infant six months old of extensive meningeal hemorrhage covering a large part of the brain. In Steffen's article several such cases are mentioned. Pulmonary hemorrhages are not frequent. Ecchymoses may be found beneath the pericardium; but endocarditis and pericarditis are extremely rare, probably occurring only in the rheumatic cases. The spleen is occasionally enlarged, but by no means uniformly so, and it may be the seat of hemorrhages.

While hematuria is one of the most frequent of the visceral hemorrhages, severe nephritis is rare. Acute degeneration of the renal epithelium of the tubes is quite common. There may be punctiform hemorrhages, and occasionally larger ones beneath the capsule or in the mucous membrane of the pelvis of the kidney. The suprarenal capsules may be the seat of extensive and even fatal hemorrhage. There may be effusions of a sero-sanguineous fluid into any of the large serous cavities, most frequently into the peritoneum. The articular lesions of purpura may be of a rheumatic character, with which purpura occurs as a complication; or there may be hemorrhages into the tissues about the joint, or even into the joint itself—usually the knee or elbow.

The blood shows the changes of secondary anemia—a moderate reduction in the hemoglobin and the red corpuscles with occasional irregularities in size and the appearance of nucleated red cells. In the most severe cases there is a moderate degree of leucocytosis. Duke has demonstrated a constant and marked diminution in the blood platelets.

Pathogenesis.—Why it is that under certain circumstances the blood-vessels will not hold their contents, it is difficult to understand. There have been described by Cassel, Riehl, Wilson, and others, changes in the small blood-vessels, usually a form of endarteritis, but the lesions are not constant. Howell has found no changes in the factors of the blood that influence coagulation. They are present in normal quantity and proportion. Henoch has suggested the vaso-motor origin of purpura, in which there is first a paralytic distention of the small vessels, followed by stasis, hemorrhage, or edema. In certain forms, as in malignant endocarditis, it is well established that the cause is an infectious thrombosis. Although the bacteriological examinations made thus far in purpura have not been conclusive, there is reason to believe that infection is the essential factor in some forms of the disease, particularly in the cases characterized by sudden onset, high temperature, and cerebral symptoms, and which run a rapidly fatal course. There are, no doubt, now included under this term purpura several diseases quite distinct from one another.

The Clinical Types.—1. The Ordinary Form.—In the mild cases the hemorrhage is confined to the skin (purpura simplex), or it is accompanied by slight bleeding from the mucous membranes. There is usually some general indisposition of an indefinite character for a day or two before the purpuric spots are noticed; most frequently a disturbance of digestion with vomiting, diarrhea, and sometimes slight fever. The hemorrhages appear as small petechiae, varying in size from a pin's head to a pea, usually first upon the lower extremities. There may be only a few widely scattered spots or the body may be covered. The color is first a bright red, then purple, gradually fading in the course

of a few days. New spots come as the old ones disappear, so that the amount of eruption may not diminish. They do not disappear upon pressure.

The course of these cases is generally favorable, recovery taking place in from one to four weeks. Relapses are, however, very frequent, and such attacks may come at intervals of a few weeks or months for a considerable period. One must be guarded in giving an absolutely favorable prognosis in any case of purpura, for it occasionally happens that in a patient who for several days has had symptoms of mild purpura, there suddenly develop those of the most severe type with a rapidly fatal termination.

2. The Severe Form.—Such cases are characterized by hemorrhages from the mucous membranes (purpura hemorrhagica) from the outset. These may even appear before the spots upon the skin. In severe attacks the petechial spots are more likely to appear suddenly, and large ecchymoses, varying in size from a pea to the palm of the hand, are more frequent. There may be bleeding from the nose, gums, mouth, or pharynx, and ecchymoses may be seen upon these mucous membranes, also upon the conjunctivae. Vomiting of blood and bloody discharges from the bowels are quite frequent symptoms. The urine may contain enough blood to give it a bright-red color. Less frequently there are seen hemorrhages of the retina or choroid and from the female genitals. In one of our cases there was almost continuous bleeding from one ear. Cutaneous ecchymoses are increased by slight injuries, such as the pressure from a bandage or from scratching. Epistaxis may be copious enough to necessitate plugging of the nares. The amount of blood vomited is not often large; its source may be the stomach, the mouth, or the pharynx. The blood in the stools is usually dark colored, but there may be some bright-red blood even when there are no ulcers present. In one of our cases so much blood was lost by the bowels as to produce the symptoms of a very marked cerebral anemia. In certain cases the gastrointestinal symptoms are very prominent, and there may be slight icterus. The discharge of blood from the stomach or intestine may be accompanied by very severe attacks of colic and tenesmus. In some of these cases there are pains and slight swelling of the joints. Renal symptoms are generally present. The attacks of abdominal pain with purpura and the discharge of blood may come on paroxysmally every few days for a period of several weeks. They have been ascribed to thrombosis of the intestinal vessels. This is sometimes known as "Henoch's purpura."

Constitutional symptoms are present in most of the severe cases. There is usually fever, from 101° to 103° F., and sufficient prostration to keep the patient in bed. If the amount of blood lost is large, there are the usual symptoms of severe anemia. The loss of blood may be

sufficient to cause death, particularly in infants. Cerebral symptoms may depend upon anemia or upon meningeal hemorrhage. They are not frequent in this form of the disease. Edema, especially of the face and feet, may exist without albuminuria, and albuminuria may be present in cases in which there is no renal hemorrhage.

In some of the cases beginning with severe general symptoms, and occasionally when the onset is mild, the patients after a few days pass into a typhoid condition with low delirium, great prostration, weak and irregular pulse, dry, cracked tongue, and high temperature. Such cases are almost always fatal. They are not to be confounded with ordinary typhoid fever complicated by purpura.

The course varies much in the different cases. It lasts from one to six weeks, the symptoms slowly subsiding, but often showing a strong tendency to recurrence. The prognosis depends upon the age of the patient, the extent of the hemorrhage, and the presence or absence of septic symptoms.

3. The Hyperacute Form (purpura fulminans).—This is a rare form, especially in young children. Its development is usually sudden, with a chill, vomiting, marked prostration, and high temperature. The purpuric spots come out with great rapidity, and in the course of a few hours or a day they may be very extensive. In addition to the ordinary subcutaneous hemorrhages, bloody vesicles may form upon the skin. In many cases the hemorrhages are limited to the skin, the mucous membranes and the viscera escaping altogether. There is no tendency to gangrene. Cerebral symptoms are invariably present and usually prominent; there may be delirium, dulness, stupor, and finally coma. The spleen is apt to be enlarged. The urine is nearly always albuminous. This form of purpura has all the characteristics of a general infectious disease, and it is almost invariably fatal.

4. The Gangrenous Form.—Sloughing is not common in purpura, but it is most often seen in the mucous membranes. Osler refers to two cases affecting the uvula. We once saw a slough which caused perforation of the soft palate. Wickham Legg reports a case with gangrene of the prepuce. Gangrene of the skin is even less frequent, although cases have been reported even in young children. Charron's patient was only three years old, and several others in children are collected in Gimard's monograph upon this subject. The gangrene may involve the skin only, or the subcutaneous tissues, and even the muscles. It has been seen upon the upper and lower extremities, and even upon the face, and may extend over quite a large surface. In some of the milder forms of purpura, gangrene results from some slight injury, such as a blow, the pressure from a bandage, or, in the nose, from the pressure of a tampon. These cases are almost invariably fatal. Those in which the sloughing

is confined to small areas of the mucous membrane of the mouth often recover.

5. The Rheumatic Form.—The term "rheumatic purpura" (peliosis rheumatica) is applied to cases, not so common in children as in older patients, in which subcutaneous hemorrhages, and sometimes bleeding from the mucous membranes, are associated with painful joint swellings. These are to be regarded as cases of rheumatism complicated by purpura. The joints most frequently affected are the knee and the ankle. The arthritic symptoms are usually less severe than in attacks of acute rheumatism. There may be present erythema exudativum or erythema nodosum or urticaria. Usually there are throat symptoms and fever, and frequently edema of the face and eyelids with albuminuria. The spleen may be enlarged. The usual duration is from one to three weeks, and although relapses may occur, the cases usually recover.

Joint symptoms, particularly articular pains, are not infrequent in the course of milder attacks of purpura without the febrile symptoms mentioned. In severe cases extravasations of blood have been reported as occurring in the tissues about the joints, and even in the joints themselves, these being cases of true arthritic purpura. It is probable that in the past some cases of scurvy have been included in this group.

Diagnosis.—The rapid acute cases may be confounded with the hemorrhagic forms of the various eruptive fevers. The ordinary subacute or passive forms are chiefly to be differentiated from scurvy. The diagnosis is not difficult, and the mistake need not be made if the essential features of scurvy are borne in mind—its dietetic cause, bleeding gums, hyperesthesia, and deep rather than subcutaneous hemorrhages which are usually near the joints.

Prognosis.—This depends very much upon the form of the disease. Of 128 cases of all varieties occurring in children in Steffen's collection, there were 40 deaths. In 12 cases of severe primary purpura reported by Gimard, there were 3 deaths and 9 recoveries. Purpura simplex is rarely fatal; cases of purpura hemorrhagica usually recover unless marked febrile symptoms are present. The forms classed as typhoid, gangrenous, and purpura fulminans are almost invariably fatal. The tendency to relapse exists in all varieties.

Treatment.—The treatment of symptomatic purpura should have reference to the cause of the disease. The mild cases of primary purpura usually recover promptly under a tonic plan of treatment. The more severe cases require confinement in bed, absolute quiet, and care to avoid exposure and even the slightest injury or extra pressure upon any part. Drugs do not seem greatly to influence the course of the disease. Those most frequently employed are epinephrin, hydrastis, hamamelis,

aromatic sulphuric acid, the vegetable acids, ergot, and gallic acid. Whether or not it is true, as claimed by some, that all hemorrhagic diseases are related to scurvy, the striking improvement seen in this disease from the use of fresh fruit and vegetables suggests their employment in purpura. In some cases very decided benefit seems to follow their use in the acute stage, but more particularly in convalescence. For hyperacute and gangrenous cases, little can be done except to treat the symptoms. Surgical means of arresting the hemorrhage are rarely successful. In all severe cases transfusion should be tried.

CHAPTER II

DISEASES OF THE LYMPH NODES (LYMPHATIC GLANDS)

It is characteristic of infancy and childhood that the lymphoid tissues—tonsils, adenoids, external and internal lymph glands, and many smaller lymph nodules throughout the body—are prone to swelling and hyperplasia. In robust children infectious processes of the nose, pharynx, or bronchi cause acute swelling of the lymph nodes in the neighborhood, which rapidly subside when the cause is removed. In others, in whom this vulnerability of the lymphoid tissues exists, the hyperplasia in the lymph nodes is out of proportion to the exciting cause and continues after the cause has ceased to operate. Certain children have at birth an excessive development of lymphoid tissue, particularly in the region of the throat in the form of enlarged tonsils, adenoid vegetations of the pharynx, etc.

The influence of heredity in causing this condition is too often seen to be passed over as a coincidence. Frequently the parents, during childhood, suffered from the same condition, and often every member of a large family of children is affected. They may be in other respects healthy, reared amid good surroundings, and show no evidence of any other constitutional disease. Any disease in the parents in consequence of which children are born with tissues having less than normal resistance, may be regarded in the light of a remote cause.

The condition is seen in perfection in children reared in institutions and in crowded tenements. It is more common in cities than in the country. Anything which produces malnutrition or lowers the general vitality of the tissues may be ranked as a cause. Rickets is often associated.

During infancy, the lymphoid structures most frequently affected are those connected with the gastro-enteric and the bronchial mucous mem-

branes; in later childhood it is those which are connected with the pharynx and tonsils.

The degree of enlargement of the lymph nodes which is sometimes found in the different situations has often led to misinterpretation. They have often been connected with pathological conditions or clinical symptoms with which they have really nothing to do.

As age advances we usually see retrograde changes in the different groups of glands unless they become the seat of tuberculous infection. Those connected with the digestive tract generally begin to diminish after the second year, and by the fifth or sixth year the enlargement has almost disappeared; while the tonsils, adenoid growths of the pharynx, and enlarged cervical glands are usually stationary after the seventh or eighth year, and frequently undergo quite a marked atrophy about the

	Name of the Group.	Number and Situation.	Organs or Areas from which they Receive Lymphatics.
1	Suboccipital.	One or two; at nape of neck.	Scalp, posterior portion.
2	Mastoid.	Four or five small ones; in mastoid region.	Receive efferent vessels from group 1, and through them from part of scalp.
3	Parotid.	Five to ten; on the surface and in the substance of the parotid gland.	Scalp, frontal and parietal portions; orbit, posterior part of nasal fossa, upper jaw, posterior and upper part of pharynx.
4	Submaxillary.	Twelve to fifteen; along base of jaw, beneath cervical fascia.	Mouth, lower lip, gums.
5	Suprahyoid.	One or two; median line between chin and hyoid bone.	Chin and middle portion of lower lip.
6	Superficial cervical.	Five or more; along external jugular vein, beneath platysma, but superficial to the sternomastoid.	Auricle, part of scalp, skin of face and neck, and some efferent vessels from groups 1 and 2.
7	Deep cervical, upper set.	Ten to sixteen; about bifurcation of common carotid and along internal jugular vein. They are just above upper border of the thyroid cartilage and on a level with the hyoid bone.	Lower part of pharynx, larynx, palate, tonsils and part of tongue, part of nasal fossa, deep muscles of head and neck, and from inside the cranium. Receive also efferent vessels from groups 3 and 4.
8	Deep cervical, lower set.	A chain in the supraclavicular fossa.	Connect with axillary group by a chain along axillary artery; also with glands of mediastinum and with groups 7 and 9.
9	Sub-hyoid.	A few small glands below hyoid bone and near median line.	Communicate with group 8, and may connect below with chain of bronchial glands.
10	Retropharyngeal	Two small glands in front of spine and upon prevertebral muscles.	Pharynx and part of nasal fossa.

time of puberty. The presence of these enlarged lymph nodes and the catarrhal condition of the mucous membranes with which they are associated, are important in relation to all acute infectious diseases which affect these mucous membranes. They bring about an increased susceptibility to scarlet fever, measles, diphtheria, and most of all to tuberculosis.

In the table on the preceding page are given the situation and drainage areas of the various groups of lymph nodes of the head and neck which play so important a rôle in infancy and childhood.

SIMPLE ACUTE ADENITIS

This is an acute inflammation of the lymph nodes which in infancy frequently terminates in suppuration. A certain amount of inflammation of the lymph nodes occurs in children in all acute processes affecting the mucous membranes, especially when they are severe or prolonged. Those in connection with the various internal organs are considered with the diseases of those organs. Acute inflammation of the external nodes is of sufficient frequency to require separate consideration. While this is probably always secondary to some pathological process in the skin or mucous membranes, the primary condition may be so slight as to be overlooked, and the adenitis may be the more important condition or may even assume the appearance of a primary disease. It is particularly in infants that this is seen, and it depends upon the unusually active absorption and upon the susceptibility of the lymphoid tissues at this age. The cervical glands are frequently affected, occasionally those of the axillary and inguinal regions.

Etiology.—Acute adenitis occurs in children of all ages in connection with diphtheria, scarlet fever, measles, and epidemic catarrh. In such cases it is often severe, and after scarlet fever not infrequently terminates in suppuration. With the simple acute catarrhal processes of the pharynx and rhinopharynx adenitis also occurs, but it is usually mild and rarely ends in suppuration. In infancy, on the other hand, acute adenitis from simple catarrh is not only very common but often severe, and frequently terminates in suppuration. Ulcerative stomatitis, carious teeth, eczema of the scalp or traumatism, may excite adenitis in children of all ages. Axillary adenitis may result from vaccination; inguinal adenitis, from balanitis or vulvovaginitis.

Of 109 cases of acute adenitis from our records, not including any associated with diphtheria, measles, or scarlet fever, more than three-fourths occurred in the first two years, and half of them in the first year of life. This susceptibility of infants is very striking. The disease

occurs frequently in those who were previously healthy, and often when the evidences of disease of the mucous membrane are slight. This is true not only of the cases of cervical adenitis, but also of others in which the inguinal glands are involved. The inflammation is usually associated with the streptococcus or staphylococcus, occasionally with the pneumococcus or influenza bacillus.

Lesions.—The changes taking place in the glands are acute congestion, with swelling, edema, and active hyperplasia of the lymphoid elements. The process may terminate in resolution or in suppuration according to the intensity of the infection and the susceptibility of the tissues. When severe enough to cause suppuration, the adenitis is accompanied by considerable inflammation of the surrounding cellular tissue.

In the series of 109 acute cases to which reference has been made, not including the specific infectious diseases, 96 were cervical, 9 were inguinal, and 4 axillary; sixty-two per cent terminated in suppuration, the latter being nearly all in infancy. Suppurative otitis was present in sixteen per cent of the cases. Suppurative retropharyngeal adenitis (retropharyngeal abscess) was associated in several cases.

Fig. 131.—Acute Suppurative Adenitis in an Infant One Year Old. Showing the most frequent situation of the tumor in the cervical region.

In infancy the disease is usually unilateral, or, if bilateral, the glands of one side are more severely affected than those of the other. Suppuration is nearly always of one side, and usually the abscess starts in a single gland.

Symptoms.—The symptoms and course of the adenitis of the specific infectious diseases belong to their clinical history. Suppuration is infrequent, except after scarlet fever.

The typical cases of acute adenitis are those which occur in infancy. There are present the symptoms of the original disease—usually acute catarrh of the nose or rhinopharynx, mouth, or ear, which may not be severe, and sometimes is overlooked. The glands most frequently affected are the deep cervical group. The tumor appears just below the angle of the jaw at the anterior border of the sternomastoid muscle (Fig. 131). The swelling during the acute catarrh is not rapid or great, but continues after the original process has subsided until it reaches the

size of a walnut or a hen's egg. In the most acute cases there is marked inflammation of the periglandular cellular tissue, with pain, tenderness, and extra heat. If suppuration occurs, it is generally evident in the latter part of the second week, but sometimes it may be as late as the third or even the fourth week. In the axillary or inguinal region (Fig. 132) the symptoms of adenitis are essentially the same as in the neck. In the inguinal cases the degree of catarrh of the mucous membrane is often very slight.

Most cases run their course with slight fever and few general symptoms; but in young infants the constitutional symptoms are often severe and the physician may be in doubt whether the local process is sufficient to explain them. The temperature may be from 102° to 104° F. for several days, with considerable prostration, which is much increased if there is complicating otitis. After suppuration, if freely opened at the proper time, the abscess heals rapidly and permanently, a sinus being rare. Occasionally the infection extends from one gland to another, and a succession of these glandular abscesses occurs.

FIG. 132.—ACUTE SUPPURATIVE ADENITIS (INGUINAL) IN AN INFANT THREE MONTHS OLD.

In the non-suppurative cases the swelling may be even greater than in those which suppurate; but it is less diffuse and apparently limited to the gland. It subsides slowly in the course of from four to eight weeks, often leaving a small tumor which may be apparent for several months. In susceptible children recurrent attacks of acute inflammation may lead to chronic enlargement which may last indefinitely. These glands do not become cheesy, except from subsequent tuberculous infection.

The acute cases in infancy in which suppuration occurs, appear to recover about as promptly and quite as completely as those terminating in resolution, although in the former the constitutional symptoms are more severe.

Diagnosis.—This is usually easy if it is remembered that, with the exception of the specific infectious diseases, and occasionally local causes like eczema of the scalp, carious teeth, etc., acute suppurative adenitis is essentially a disease of infancy. It is often mistaken for mumps when the swelling is severe, but on close examination there is but little

resemblance between the conditions. The disease is usually acute, and has little in common with the slow suppuration seen in later childhood from the breaking down of tuberculous glands. In the occasional cases seen in which the disease runs a slower course a diagnosis from the tuberculous form may be aided by a tuberculin test.

Treatment.—Prophylaxis requires that in all acute catarrhs the mucous membrane should be kept as clean as possible by the use of nasal or pharyngeal sprays, or by careful syringing with simple solutions like Dobell's or Seiler's, or a simple saline.

In the stage of acute inflammation very hot applications or an ice-bag may be used for the relief of pain. It is very doubtful whether either of these means has much influence in preventing suppuration. If abscess forms, incision should be deferred until pointing has taken place. If this plan is followed, refilling is rare. A simple incision with proper aseptic treatment is all that is required. Curetting may be done if there is much broken-down tissue present, but it is not usually necessary. In most of the cases the abscess promptly heals and a perfect cure takes place. Benefit is seldom seen from painting with iodin or from inunctions of iodin ointment or the oleate of mercury. If adenitis is secondary to carious teeth, eczema, or ulcerative stomatitis, these conditions should receive appropriate treatment. Such cases do not usually suppurate, but subside rapidly when the primary cause is removed.

SIMPLE CHRONIC ADENITIS

This consists in a simple hyperplasia of the lymph nodes which is non-syphilitic and non-tuberculous. There are considered here only the external glands, but those of the cavities of the body are affected in a similar way, in diseases of the mucous membranes with which they are connected.

Simple chronic adenitis is not so frequent as the acute form in infants, and it is less common after the third year. It may follow one or more attacks of acute adenitis, or it may result from subacute or chronic inflammations of the skin or of the various mucous membranes, infection from which causes the acute form. Chronic enlargement of the glands of the neck is very common with adenoids, diseased tonsils and with pediculosis of the scalp.

Symptoms.—The glands upon both sides of the neck are usually involved, and more often a group than a single gland. The degree of swelling is not generally great, being much less than in acute adenitis, and usually less than in the tuberculous form. There are no constitutional symptoms. Hypertrophy of the tonsils and adenoid growths of

the pharynx are frequently associated. There is no tendency to suppuration or caseation. The swelling usually increases slowly for one or two months, then remains stationary for about the same length of time, after which it slowly subsides. A subacute course is more frequent than a very chronic one.

Diagnosis.—These cases are especially to be distinguished from the much more frequent cases of tuberculous adenitis. The most important points for differentiation are, that they occur most frequently in children under two years, a period when tuberculous adenitis is not very common; some definite exciting cause is usually present; caseation and suppuration do not occur; the glands do not become adherent to the skin or to the deeper tissues; they usually enlarge more rapidly than do the non-caseating tuberculous glands; and they are influenced to a greater degree by constitutional treatment. The children do not usually respond to the tuberculin test.

Treatment.—Operative measures are not called for in simple adenitis. Local causes usually found in the pharynx, nasopharynx, or mouth should be removed if possible. Pediculosis should be treated. Often more can be accomplished by removal to a climate in which the child's catarrhal symptoms are relieved than by all else. Little benefit is seen from local applications. The most useful internal remedies are, the syrup of the iodid of iron (twenty drops three times a day to a child of four years), and arsenic (two or three drops of Fowler's solution three times a day). Cod-liver oil should be given continuously except during warm weather.

SYPHILITIC ADENITIS

It is quite rare that a marked degree of glandular enlargement is seen as a symptom of hereditary syphilis; indeed, it is so rare that it is often forgotten that chronic multiple glandular enlargements are ever due to this disease. In the few examples that have come under our observation, this has been a late symptom of hereditary syphilis. The glandular enlargements were cervical and multiple, and the degree of swelling was often marked. They may be associated with disease of the bones or of the mucous membrane of the throat or of the nose, or without signs of such disease. The diagnosis of syphilis rests upon the association of other late manifestations of the disease—keratitis, periostitis, deformities of the teeth, the Wassermann reaction, and the prompt improvement under antisyphilitic treatment. In their local appearance they resemble tuberculous glands.

TUBERCULOUS ADENITIS

(*Scrofula*)

Tuberculous disease of the lymph glands of the cavities of the body is discussed elsewhere; only that of the external glands is here considered. This condition presents some striking peculiarities: it is not common in infancy, although one of the most frequent forms of tuberculosis in older children; it often exists as the only apparent tuberculous lesion in the body. In the great majority of cases it is the cervical glands which are affected.

Etiology.—The age at which tuberculosis of the cervical lymph glands is most often seen is from three to ten years. In tuberculosis in infancy, the external glands are not usually involved, while the bronchial glands are almost invariably the seat of infection.

The cervical glands become involved as the result of a descending infection from the rhinopharynx or of an ascending infection from the bronchial glands. The descending infection is altogether the most common one. The tonsils and less commonly the adenoid tissue of the rhinopharynx become tuberculous from the sputum coughed up from the lungs or from organisms received into the mouth from outside. From the foci in the pharynx the path is direct to the cervical glands. Local pathological conditions that affect the tonsils and adenoid tissue and so favor the development of tuberculosis are chronic pharyngitis, disease of the tonsils and carious teeth. Attacks of grippe, measles and scarlet fever, frequently play the rôle of exciting causes. The question often arises whether the process is at first a simple one and later becomes tuberculous, or whether it is a tuberculous one from the beginning. Our own belief is that in practically all cases the process is a tuberculous one from the outset.

Of 97 cases of tuberculous adenitis in children studied by Park and Krumwiede, 51 showed the human type of bacillus and 46 the bovine type. The proportion of cases of bovine infection was much higher in children under five years of age than in those who were older (61 and 38 per cent respectively). These findings showing the frequency of bovine infection are in striking contrast to those obtained by them in other forms of tuberculosis in children and point unmistakably to food or mouth infection, most probably tuberculous milk, as a cause.

Lesions.—It has already been stated that in the great majority of cases the cervical lymph nodes are involved, and generally they are the only ones affected. In 155 cases of tuberculous glands in the series reported by Treves, those of the neck were the seat of disease in 145 and

the only seat in 131; those of the axilla were involved in 17, but alone only in 4; the groin in 8, and alone in 6. The nodes first affected are most frequently the upper set of the deep cervical group; sometimes, however, it is the superficial nodes of the submaxillary, or the parotid group, and occasionally the submental or the pre-auricular. The chain of deep cervical nodes which is involved, follows the carotid artery, and often extends some distance below the clavicle. These deep nodes are sometimes connected with the bronchial group, but it is much more frequent to trace them upward to the tonsils which in a very large proportion of the cases are tuberculous.

The process in all tuberculous glands is essentially a chronic one, but pathologically the cases may be divided into two groups, corresponding somewhat to the forms of disease seen in the lungs. In one group the process is more rapid, and tends to early caseation and softening; the products of inflammation are mainly cellular, and the amount of fibrous tissue is small. In another group the course is slower, and fibrous tissue predominates, caseation and softening being late or absent.

In the first group the glands in the early stage are swollen, of a pale pink color, and homogeneous; later they become more firm, and show, as the first gross evidence of tuberculous deposits, small grayish-white spots, which are generally numerous and scattered through the affected gland; these spots enlarge, and may coalesce to form one large gray mass, involving nearly the whole gland. Subsequently there is caseation and then softening, usually beginning in the center of the caseous area. Inflammation within the gland is followed by that of the surrounding tissues, which may result in adhesions or in the formation of a peri-glandular abscess. The first change in the gland is the production of epithelioid and giant cells, about which there is a zone of small round cells; cheesy degeneration then begins in the center. The caseous masses may become encapsulated by the production about them of fibrous tissue; or softening may occur at one or more foci, and an abscess form. Such an abscess contains curdy material, but very little true pus, the contents being chiefly detritus from the broken-down node. Tubercle bacilli are usually more numerous in the early stages of the process, but are often difficult of detection in broken-down tissues, and the curdy pus is sometimes sterile. As the glands soften, the process gradually extends from the center to the surface, and they become adherent to the surrounding structures—blood-vessels, nerves, or the fascia—they fuse together and form large knotty masses, and when they ultimately break down they lead to the formation of an abscess in the cellular tissue, finally involving the skin. In the form of suppuration which occurs in and about tuberculous nodes, an important part is often played by other bacteria, usually the staphylococcus or the streptococcus.

In the second group of cases, where the process goes forward more slowly, the changes are not quite the same, the essential difference being that the amount of fibrous tissue is much greater. These nodes are not so vascular; they are tough and hard, appearing like small fibrous tumors. The capsules are greatly thickened, and under the microscope is seen fibrous tissue arranged in concentric layers, often inclosing small caseous masses. These nodes less frequently form adhesions to the surrounding tissues, and consequently are freely movable, while suppuration is quite exceptional. Although the separate tumors are much smaller than in the first group, the glandular mass is often a large one, because of the number of glands involved.

It is seldom in either group of cases that the process is limited to a single node or even to two or three nodes. Very often an entire chain is involved.

Tuberculous infection of the lymph nodes may terminate in resolution, encapsulation, calcification, or suppuration. The inflammation may subside before caseation has taken place and the inflammatory products undergo absorption. After caseation has occurred the masses may become encapsulated and contract to small fibrous nodules. Calcification of the glands in this location is rare. In other cases caseation is followed by breaking down, liquefaction, and an external abscess. The course which the local disease takes will depend upon the intensity of the infection and the general vigor and resistance of the child. There is seen in most cases a tendency of the inflammation to subside spontaneously about the time of puberty. Cure has sometimes followed an attack of intercurrent disease, such as erysipelas of the face, and even scarlet fever.

Symptoms.—In the early part of the disease there are no symptoms but the glandular swelling, and this usually begins gradually. In many cases both sides are involved, but as the disease progresses the advanced changes are usually confined to one side. In other cases the first swelling noticed is an acute one, but, unlike other acute enlargements, it does not subside, but persists. The symptoms in most cases are characterized by remissions and exacerbations; the glands increase for a time and then remain stationary or even diminish, to take a new start from the stimulus of some fresh infection of the mucous membrane with which the glands are associated, such as an attack of measles or influenza, or simply from a deterioration in the patient's general health. During exacerbation the glands may be painful and tender and show the usual signs of local inflammation.

The whole course of the disease varies from several months to as many years. As a rule the younger the patient the more rapid its progress. Treves gives three and a half years as the average duration when

suppuration occurs, but in infancy the glands sometimes break down in two or three months. The glands first affected are usually those situated near the bifurcation of the common carotid artery. Such tumors usually make their appearance just in front of the sternomastoid muscle —sometimes behind it—and at the level of the upper border of the larynx or the hyoid bone. In the more rapid cases the tumors usually attain a considerable size in three or four months, sometimes in half that time. The usual size reached is from that of an almond to an English walnut. At first the tumors are movable and preserve their distinct outline; later they become adherent, first to the deeper tissues and to each other, finally to the skin, and there is formed an irregular nodular mass in which it is sometimes difficult to make out the individual glands. As the process approaches the surface there are small spots of softening; then there is distinct fluctuation; the skin becomes discolored and finally gives way, and there is a discharge of thick, curdy pus, which may continue for an indefinite time, until the whole of the broken-down gland has been thrown off. This course is repeated with each successive gland which breaks down. In cases progressing more slowly the glands become adherent chiefly to one another, and suppuration is less frequent.

In what proportion of tuberculous lymph nodes suppuration occurs, it is difficult to say. Like other tuberculous lesions in the body, this one is much more frequent than was once supposed; formerly, if glands did not break down in a few years, they were usually regarded as non-tuberculous. We now know that a large number of tuberculous glands do not break down for many years and some never do. Two forms of suppuration occur in connection with tuberculous glands—one an abscess of the gland proper, the other outside of and usually over it. In a typical case of the first variety, the gland is distinctly outlined and often superficial, there is very little inflammation, the spot of softening and fluctuation is small, and the pus discharged is always curdy. In the second variety the abscess is preceded by a more diffuse swelling, and the outline of the gland may not be made out; the signs of inflammation are more marked, the area of fluctuation is larger, and the pus is more like that of any ordinary abscess. Often the two varieties are combined; as when a gland beneath the deep fascia breaks down and there is formed directly over it an abscess in the cellular tissue, which communicates through a narrow opening with the gland beneath. In such cases the sinus continues open for a very long time, until the whole of the gland has been discharged. If healing occurs before this, the cicatrix soon breaks down.

When abscesses are allowed to open spontaneously, large, irregular, and usually very intractable ulcers form. The skin is undermined for a considerable distance, and it has an unhealthy appearance. Such ulcers

sometimes continue for many months in spite of all treatment, particularly if the patient's general health is poor. The scars left after them are large and unsightly, and sometimes positively deforming (Fig. 133). Their appearance is quite characteristic. They often have many tabs of skin attached to them; they may form prominent ridges which undergo contraction like those after burns; they are of a purplish-red color, and adherent to the deeper tissues. They are often sensitive and painful. As time passes they atrophy and become less conspicuous, though they remain throughout life.

Fig. 133.—Cicatrices Following a Neglected Case of Tuberculous Adenitis, in a Girl Seven Years Old. There is also a tuberculous patch upon the skin of the cheek in a not infrequent location.

The general health of children with tuberculous glands of the neck is usually but little affected. Although the local process is often extensive the absence of general symptoms is striking, and the secondary development of generalized tuberculosis is infrequent. Both these facts indicate that bovine infection in the human subject is relatively mild. At any time in the course of the disease an examination of the throat often shows enlarged tonsils, but even when they are not grossly altered, serial section proves them to be tuberculous in a large proportion of the cases.

Prognosis.—Tuberculosis of the external lymph nodes is seldom if ever the direct cause of death; although the course is often very protracted, ultimate recovery can usually be predicted. Treves states that the percentage of those who die from general tuberculosis is so small that this danger is not to be considered an argument for operation. Poore reports that of 58 cases treated by operation, only 2 were known to have died from tuberculosis. Dowd has collected reports of 309 cases, chiefly hospital patients, treated by removal more or less complete, whose course was followed for several years after operation. Of these, 202, or

65.4 per cent, were apparently cured; 57, or 18.4 per cent, were living, though suffering from either local or general tuberculosis; 50, or 16.2 per cent, died of tuberculosis. These statistics hardly support the hopeful views of the writers first quoted, but they are, we believe, more in accord with general experience in the class which makes up hospital patients. In private practice the results are much better.

Diagnosis.—The diagnostic features of tuberculous glands are the age of the patient—usually from two to ten years—the site of the primary swelling, the indolent course, the trifling original cause, and the disposition to slow caseation, softening, and abscess. The cutaneous tuberculin reaction is of great assistance in diagnosis; in a young child a positive reaction is significant, while at any age a negative reaction is usually conclusive. The cases of simple inflammation are usually in children under three years, their progress is much more rapid. If they do not break down they generally disappear in the course of four or five months. They usually suppurate, if at all, during the first month. Chronic glandular enlargements which persist are usually tuberculous, no matter how good the surroundings or the general health. Syphilitic disease of the cervical glands is relatively rare in children. It is recognized by the Wassermann test, by the evidence of syphilis elsewhere, and by the effect of treatment. In Hodgkin's disease, glandular groups in other parts of the body are involved simultaneously or in rapid succession. There are no signs of inflammation or caseation; and the swellings are usually accompanied by very marked and definite general symptoms and blood changes. Malignant growths are very rare; they increase rapidly, often attaining a great size in a few months.

Treatment.—As the tonsils are so frequently the seat of infection it is important to examine these most carefully in every case. Unless it is entirely clear that they are free from disease they should be removed. Removal of tuberculous tonsils is sufficient in many cases to bring about cessation of the process in the cervical glands. Many begin to diminish in size shortly after tonsillectomy. If it is done early in the disease suppuration of the glands is much less likely to occur. Adenoid growths of the rhinopharynx and carious teeth should also receive attention.

A child from the city should be sent into the country whenever this is possible. The seaside has a great reputation in such cases and no doubt the majority do very well there, but some are benefited even more by a dry mountain climate. Climatic treatment is to be recommended particularly for those children who have pulmonary lesions and therefore infection with the human type of organism. Those with only tonsillar and glandular tuberculosis do well with the removal of the focus. This should not be neglected in any case.

Drugs are of little benefit. Cod-liver oil, arsenic and iron are useful

only as general tonics. Local applications are of little value. The parts should not be rubbed or handled.

Brilliant results have been reported by Rollier of Switzerland of treatment by heliotherapy, or the exposure of the diseased parts directly to the sun's rays. This is especially to be recommended for old cases with extensive lesions, when complete removal is impossible or when operation wounds do not heal.

Operative Measures.—These are indicated, if after the removal of the probable foci and a trial for a few months of climatic and general measures, the glands do not diminish but rather increase in size and number, or if there are signs of softening. The advantages of operation are that it leaves a clean scar which when the incision is properly made is almost imperceptible; that it shortens the disease; that if thoroughly done and the deep as well as the superficial glands are removed, it is a radical measure. The best results follow when operation is done reasonably early before the skin is involved or the glands have softened or have formed extensive adhesions to the great vessels and neighboring structures; also when a chain of glands is involved and when the inflammatory process is slow or indolent. A thorough operation by a good surgeon in the great majority of cases will result in a permanent cure. However, the operation is not contra-indicated in cases which have gone on to a later stage, although the results may not be quite so satisfactory.

If more radical measures are for any reason impossible, glandular abscesses should be opened as soon as pus forms, to prevent the extensive undermining of the skin, which is likely to occur. The opening should be a small one, and all squeezing of the gland or surrounding tissues avoided.

As an alternative to operative measures, or when these are refused, exposure to the X-ray may be tried and in a certain proportion of cases it is curative. The best results are seen in the early cases. The first exposures should be short, and they should be repeated not oftener than once a week.

Tuberculin Treatment.—This has been employed extensively with a number of different preparations obtained from cultures of tubercle bacilli.[1] It is the general consensus of opinion that this method of treatment is of benefit, and that it diminishes the tendency to softening and promotes resolution. Our own belief is that it should not and can not take the place of operative measures.

---

[1] The preparations of tuberculin most widely used are B.F. (bouillon filtré) of Denys; O.T. (original tuberculin); T.R. (tuberculin residue), and B.E. (bacillary emulsion).

The doses are calculated in milligrams, it being considered that one cubic centimeter of the fluid weighs one gram, which is nearly if not quite the case.

The purpose is to give enough tuberculin to affect the local process, but never enough to produce a general systemic reaction—fever, malaise, swelling of the glands, etc. It is necessary to begin with a very small dose and to increase this gradually. If there is any elevation of temperature following an injection, the amount should be diminished to a quarter or less of the dose given and a return made to the amount causing the reaction only after several weeks. The best indication that one has reached the point where an increase in dosage is to be made with especial care, is the reaction produced at the site of injection. When this is made subcutaneously there may be around the point of injection a slight swelling, induration and tenderness for some days. Injections should be repeated every four or five days. An initial dose of .00002 mgm. is proper for an average child of two or three years. The dose may be doubled at each injection until .05 mgm. is injected. After this it is safer to repeat the same dose two or three times before increasing further and to give this dose at weekly intervals. It is not advisable to increase beyond .1 gm. as the maximum dose. The duration of treatment will depend upon the effect upon the glands. It is usually several months. Even when the results have been favorable it is considered advisable by many to repeat the course of treatment after an interval of some months.

HODGKIN'S DISEASE

(*Pseudo-Leukemia*)

Hodgkin's disease at the present time is to be considered a distinct clinical and pathological entity. For many years there was no general agreement regarding its determining characteristics and in the older literature many cases were included which were undoubtedly not Hodgkin's disease. The condition is relatively rare. In infancy it is almost unknown, but after the age of three years it is found with increasing frequency throughout childhood. It is much more common in males. The essential cause of Hodgkin's disease is unknown. Numerous organisms have been described in connection with it, especially modified forms of the tubercle bacillus and more recently diphtheroid bacilli. It is doubtful if the disease results from infection with any of them.

Pathology.—The chief lesion is in the lymph nodes, which become greatly enlarged and in addition new ones develop during the course of the disease. Those first affected are usually in the neck, but any of the external or internal groups of lymph nodes may be affected and in severe cases the disease may involve almost every chain of glands in the body. Of the internal glands those of the mediastinum and retroperitoneal region are usually most affected. Large masses are formed

by the growth and multiplication of the lymph nodes, but even in the largest masses the individual nodes are discrete and are held together only by loose connective tissue. The spleen is usually, the liver less frequently, involved and somewhat enlarged by the formation of lymphomatous masses, which may also infiltrate almost any tissue of the body. Microscopically, the early changes in the glands consist in an increase in the lymphoid tissue. Later there is proliferation of the endothelial cells, the formation of giant cells and an overgrowth of connective tissue. The eosinophile cells are frequently present in the tissues in great numbers. The lymphomatous masses in the spleen and other organs have the same structure microscopically as the diseased nodes.

Symptoms.—The first evidence of disease is usually the swelling of one or more cervical glands. Thereafter there is a progressive involvement of other glands, though the rapidity with which this occurs may vary greatly. At the beginning the general health remains unaffected and this usually continues until the glandular enlargement is widespread. Then a more or less persistent fever may develop or anemia supervene or pressure symptoms make themselves evident.

The fever may be irregular, with wide excursions and periods of remission, or, what is more common, it may be only of a degree or two but persistent. The blood shows the characteristics of a secondary anemia, which increases in severity. The leucocytes may be slightly diminished or increased, but in the late stages there is usually a polymorphonuclear leucocytosis (20,000-30,000 or more). There are two constant features, an increase in the blood platelets and an increase in the transitional leucocytes. Eosinophiles, while usually somewhat diminished, may be present in great numbers.

The glandular masses can be felt to be made up of discrete glands. These are elastic, sometimes distinctly soft, at others, firm. They are more or less movable and not adherent to the deeper structures nor to the skin over them. At any time symptoms may appear as the result of the mechanical pressure of the glands. This may be on the vessels of the neck or extremities, producing edema; upon the esophagus, producing dysphagia; or upon the trachea or bronchi, producing dyspnea. Intra-abdominal pressure may cause jaundice or chylous ascites. In most cases enlargement of the spleen can be made out. In some instances it is extreme.

The duration of the disease is usually less than three years, sometimes only a few weeks. There may be periods in which the progress seems arrested, but they are usually short. Death results from asthenia, or from pressure usually upon the respiratory tract, producing slow suffocation with most distressing symptoms. The prognosis is bad. We know of no children with Hodgkin's disease that have recovered.

Diagnosis.—The diagnosis of Hodgkin's disease may be difficult at the beginning, when only a few cervical glands are enlarged. It may be confounded with glandular tuberculosis, with lymphosarcoma and with leukemia. From tuberculosis it is to be differentiated by the wide distribution of the progressively enlarging glands; by their failure to coalesce, to exhibit inflammatory reaction or to suppurate; by the frequent absence of the von Pirquet reaction and by the more malignant course and pressure symptoms. Lymphosarcoma is more rapid in its course, does not usually cause fever, the glands do not remain so discrete as in Hodgkin's disease and the spleen is seldom involved. Leukemia is distinguished by less lymphatic enlargement, by greater rapidity of progress, especially in the lymphatic form, and especially by the character of the blood findings. In doubtful cases the excision and examination of a gland will almost always give reliable information as to the presence or absence of Hodgkin's disease.

Treatment.—This is very unsatisfactory, but some remedies apparently are of temporary benefit. Arsenic in full doses appears to benefit some patients. The use of the X-ray has produced striking but not permanent improvement in the external glands. Recently vaccines prepared from the diphtheroid bacilli cultivated from the glands have been employed. It is too early to judge of the influence of this method of treatment. Tracheotomy occasionally is employed to relieve dyspnea, but is seldom indicated because the obstruction to respiration is usually situated very low in the neck or in the thorax.

CHAPTER III

DISEASES OF THE DUCTLESS GLANDS

THE SPLEEN

Weight.—From 110 observations made at the New York Infant Asylum the following were the weights recorded at the different ages:

Age.	Ounces.	Grams.
Birth	¼	7.7
Three months	½	15.5
Twelve months	¾	23.2
Two years	1¼	38.5
Three years	1½	46.4

Position and Methods of Examination.—The normal position of the spleen is close against the diaphragm, its external surface being opposite the ninth, tenth, and eleventh ribs. Its anterior border comes as far forward as the middle axillary line, its posterior border being usually near the vertebral column. In infancy it is practically impossible to outline the spleen by percussion unless it is enlarged. During full inspiration the spleen is often depressed enough to be felt at the free border of the ribs, but at other times it can not be felt unless it is enlarged or pushed downward by some pathological condition in the chest. Normally, the long axis of the spleen is nearly parallel with the ribs, but when the organ is much enlarged, its axis corresponds nearly with a line drawn from the axillary line at the border of the ribs to the middle of Poupart's ligament.

The thin abdominal walls of young children render palpation of the spleen much easier than in adults; and this is a much more satisfactory method of examination than is percussion. For satisfactory palpation it is necessary that the abdominal walls should not be tense. The child should lie upon his back with the thighs flexed and the skin, of course, bared. The physician, always having taken the trouble to warm his hands, should stand upon the left side of the patient and make pressure with the tips of the fingers, which are semi-flexed. The pressure should be at first light, and gradually increased, the fingers being then held stationary during two or three respiratory movements. Under ordinary conditions the spleen can easily be felt when it is sufficiently enlarged to be of any diagnostic importance.

When moderately enlarged, the lower border of the spleen is an inch or so below the free border of the ribs; when greatly enlarged, it forms a tumor which may nearly fill the left half of the abdomen. A tumor in the left hypochondriac region is recognized to be the spleen, by the fact that it is freely movable laterally and at its lower border or extremity, while it is attached above; also its inner border can usually be felt to be thin and sharp, and marked about its middle by quite a deep notch.

ENLARGEMENT OF THE SPLEEN

In Acute Disease.—The spleen is most frequently and most constantly enlarged in malarial and typhoid fevers, but it is occasionally so in all the acute infectious diseases.

In most of these cases the enlargement is chiefly from congestion, but there may be acute hyperplasia and an increase in size of the Malpighian bodies. It may contain small hemorrhages, and in extremely rare cases the spleen may rupture. It is generally dark-colored, soft, and some-

what friable. In the cases which recover, the splenic swelling subsides with the original disease.

In Chronic Disease.—Like the lymph nodes, the spleen is much more often enlarged in children, particularly young children, than in adults. Enlargement is seen at times in almost all the chronic diseases of early life; but it occurs most frequently in rickets, syphilis, malaria, tuberculosis, the blood diseases, and in amyloid degeneration. Besides, it may be the seat of a primary growth, either benign or malignant.

Rickets.—The splenic enlargement which accompanies rickets is generally seen during the first year; at this period it is very frequent. The swelling is usually moderate, but occasionally it is so great that the lower border is three or four inches below the ribs.

Syphilis.—Enlargement of the spleen is one of the most constant lesions of hereditary syphilis. It is present with great uniformity in children born with syphilitic lesions, and very frequently during the active period of the disease in early infancy. It is seen at a later period during infancy or childhood, associated with other late symptoms.

Malaria.—The swelling in cases of chronic malaria may be very great. The liver is not so often enlarged as in syphilis.

Tuberculosis.—It is rare to find anything more than a moderate swelling of the spleen in pulmonary tuberculosis. In general miliary tuberculosis, enlargement of the spleen is an almost constant finding. The enlargement is usually progressive, due to an increase in the number and size of the tuberculous deposits which are regularly present.

Diseases of the Blood.—Marked enlargement of the spleen is found in many cases of secondary anemia. The spleen is constantly swollen, and usually greatly so, in the pseudoleukemic anemia of infants, in leukemia, and in Hodgkin's disease. In the last two diseases the liver is also enlarged, but to a much less degree than the spleen; in the others it is but slightly changed.

Amyloid Degeneration.—The spleen is constantly involved in amyloid disease, and the enlargement of this organ, as well as that of the liver, may be very great.

Cardiac Disease.—In all forms of cardiac disease, and in other conditions in which there is obstruction to the systemic venous circulation, the spleen is enlarged. It is seen in congenital as well as in acquired cases. The liver is usually enlarged, and there may also be edema of the feet or general anasarca.

New-growths, Tumors, etc.—It is seldom in early life that the spleen is the seat of new-growths; these are usually varieties of sarcoma, but carcinoma has also been reported.

Banti's Disease—Splenic Anemia.—These are rather unsatisfactory terms which are used to designate a clinical condition which is, at

times, capable of sharp differentiation, but which pathologically has no especially distinguishing features. In the late stages, the lesions are essentially those of periportal cirrhosis of the liver. The spleen is greatly enlarged and shows a marked increase in the fibrous tissue both of the capsule and reticulum. In the early stages the Malpighian bodies may be enlarged. In the late stages they are small and infrequent.

The onset is late in childhood, usually not before the tenth year, and the progress is slow. Attention is generally first attracted to the anemia and the symptoms that accompany it, such as dyspnea on exertion and cardiac palpitation. The anemia has the characteristics of a secondary anemia. There is usually a moderate, relative increase of the lymphocytes. There may be from time to time slight rises of temperature and occasionally epistaxis. Physical examination shows in such instances a moderately enlarged and firm spleen. The splenic enlargement is very slow but progressive. It is never extreme. After a time a slight increase in size of the liver occurs. The progress of the disease is very gradual. A fair degree of health may be maintained for ten or twelve years. Then there are superadded the evidences of hepatic cirrhosis. The liver diminishes in size until it can no longer be felt. There may be icterus and urobilinuria and eventually ascites with dilatation of the abdominal veins, hematemesis and submucous hemorrhages. Death usually occurs from some intercurrent disease before the development of the evidences of hepatic insufficiency and obstruction.

The justification for considering Banti's disease a clinical entity, distinct from cirrhosis of the liver, with which the pathological findings are nearly identical, rests upon the duration of the symptoms, the disproportionately large spleen and the frequent absence of ascites and icterus. The course of true cirrhosis of the liver in the young is often rapid; the duration is usually a year or less. The enlargement of the spleen is generally slight, while ascites often develops early and is very obstinate. Syphilis of the liver and spleen may be difficult to differentiate from Banti's disease by physical examination alone, and several cases diagnosed as Banti's disease have been shown at autopsy to be syphilitic in origin. The evidence afforded by the Wassermann reaction and by careful examination for syphilis of other parts of the body should be sought. Hemolytic jaundice may be excluded if there is no increased fragility of the red cells. In Gaucher's disease the progress is also slow and a reasonable degree of health may be maintained for many years. There is often, however, a history of several cases in the same family; there may be a brownish discoloration of the skin; after some years the liver is also enlarged and the spleen eventually reaches proportions found in no other disease.

It has been maintained by Banti that the spleen is the primary factor in the disease and that the liver is secondarily affected. There is little to substantiate this view, except that in the early stages of the disease striking benefit results from splenectomy. Sufficient time has not yet elapsed, nor have sufficient cases been recorded, to prove how permanent the benefit will be. It is clear, however, that splenectomy is indicated in the stages of the disease before serious involvement of the liver. When ascites has developed palliative treatment alone should be employed.

Hemolytic Jaundice—Chronic Family Jaundice.—This disease is usually hereditary, but it occasionally exists in several brothers and sisters, the parents being unaffected. Similar cases may be seen without a family association. There are records of many families in which jaundice has existed through three or four generations. It is transmitted alike through the male and female descendants, and not all of the children in a family are affected. The descendants of unaffected members escape. The jaundice may be noticed shortly after birth, or it may develop at any time during childhood, sometimes not until later. This is the most striking feature of the disease. The discoloration may be very slight and noticeable only in the sclerotics, or the skin may be icteric. The color is never very intense. It varies somewhat in degree and is increased after intercurrent gastro-intestinal attacks, which are rather frequent. When once developed, the icterus never entirely disappears.

This jaundice is not obstructive; the stools are usually darker than normal and the urine contains urobilin in excess, but no bile. There is an increased production of biliary pigment. The liver is normal or slightly enlarged. The spleen is regularly, and often excessively, enlarged, and even in youth there may be attacks of biliary colic and of perisplenitis. Anemia of a moderate grade is the rule. Both the red cells and hemoglobin are reduced, and a few nucleated red cells may be found. Reticulated red cells may be demonstrated by means of vital staining. As many as 20 per cent of the total red cells may be reticulated as opposed to the normal of 1 per cent or less. Very characteristic of the disease is the increased fragility of the red cells to hemolytic agents, especially to hypotonic salt solutions. Normal red cells are not hemolyzed by solutions of sodium chlorid of a concentration of 0.5 per cent or more. With salt solutions of 0.45 per cent hemolysis begins and is complete with those of 0.35 per cent. With hemolytic jaundice hemolysis usually begins with solutions of a concentration between 0.7 and 0.6 per cent and is complete with those between 0.55 and 0.45 per cent.

The growth and development of children go on uninfluenced by the condition, and many affected persons have lived to an advanced age. There are no characteristic post-mortem findings. Various drugs, among

them iron and arsenic, have been employed in treatment. The only effective method is surgical. Splenectomy has been employed with marked improvement in several instances. In some cases, symptomatic cure has been reported. Splenectomy should be done if there is much interference with the patient's general health.

Gaucher's Disease.—This is a rare disease, which frequently attacks two or more members of a family, but is not hereditary. It usually begins before the age of ten years and cases have been reported in the first year of life. The most striking feature is an enlargement of the spleen, which is slowly progressive and may eventually nearly fill the abdomen. It is firm, smooth and not tender. While never reaching the proportions of the spleen, the liver may be considerably increased in size. It is also smooth. A secondary anemia with leucopenia is constantly present but is not severe. Associated with this is a peculiar brown discoloration of the skin, particularly of the face. In some instances, there is a yellowish wedge-shaped thickening of the conjunctiva on either side of the cornea. The superficial lymph glands may be palpable, but are not materially increased in size. The general health may be fair for many years. The splenic and hepatic enlargements may cause abdominal discomfort and even pain, but it is rare for jaundice or ascites to develop. Eventually hemorrhages may occur from slight traumatism or spontaneously from the mucous membranes.

The disease may last many years. Death usually results from some intercurrent disease. While the origin of the disease is obscure, the pathological findings are entirely distinctive. Microscopically it is seen that the enlargement of the liver and spleen is due to the accumulation of characteristic cells which widely invade these organs. The cells are very large, with small excentrically situated nuclei and with slightly granular cytoplasm. These cells are found not only in the spleen but also in the bone marrow and lymph glands. The accumulation in the lymph glands is not sufficient to cause marked enlargement, but is important as showing that the disease is a systemic one, and not primarily one confined to the spleen. The presence of the distinctive cells in the glands may be of assistance in diagnosis, as in a case reported by Knox, in which the suspected diagnosis was confirmed by the microscopical examination of an excised lymph node.

Medical treatment does not influence the course of the disease. On *a priori* grounds it does not seem likely that splenectomy will produce permanent cure in a disease whose lesions are so widely distributed in other organs. A number of cases, however, have been operated upon and some have shown a distinct improvement. The time that has elapsed in the majority is, however, too short to enable a definite conclusion as to the final result to be reached.

DISEASES OF THE THYROID

SPORADIC CRETINISM

(Athyreosis; Myxedematous Idiocy)

Since the early descriptions of this disease by Fagge, in 1871 and 1874, numerous cases have been published in England, on the continent of Europe, in America, and in fact, all over the world, showing that sporadic cretinism is not confined to any country. The condition is a relatively rare one, but in a large dispensary and hospital service one or more examples of it are seen every year.

Etiology.—It is now well established that this condition depends upon the absence of the internal secretion of the thyroid gland. In almost all the autopsies in cases of sporadic cretinism that have been reported there has been an entire absence of the thyroid gland. Not even a trace of it has been found. In one or two instances cysts have been met with in the region of the lateral rudiments of the thyroid gland, or at the root of the tongue in the region of the median rudiment. These cysts may contain a few cells resembling thyroid tissue, but nothing that is apparently capable of functionating. There are no recorded observations upon cases of sporadic cretinism that would indicate that an already developed thyroid gland had been affected by injury or disease. The absence is due to a congenital lack of development such as produces anencephaly or the absence of other parenchymatous organs. As a rule only one case occurs in a family, the other members of which present nothing abnormal in mental or physical development.

There are associated no constant changes in the other ductless glands. In the few cases in which the parathyroids have been searched for at autopsy they have been found. Alterations in the pituitary gland have been quite frequently reported. It has been found hypertrophic and occasionally cystic, but this is not constant.

Symptoms.—The symptoms of cretinism in most cases make their appearance during the second half of the first year, but are sometimes so slight as not to be noticed until children are two or three years old. Very rarely the condition is recognized as early as the third or fourth month. The delay in the development of the symptoms is to be ascribed to the protection afforded the infant by the thyroid secretion of the mother during intrauterine life. This view is substantiated by the rare but undoubted instances where women with either goiter or hyperthyroidism have borne infants with cretinism which was clinically recognizable at birth. Failure to grow and to develop mentally are usually the first

things to attract attention. The peculiarity of the facial expression is soon noticed. The general appearance of the cretin is striking, and so characteristic that when once seen the disease can hardly fail to be recognized (Fig. 134). The body is greatly dwarfed, and children of fifteen years are often only two and a half or three feet in height. All the extremities, the fingers and the toes, are short and thick. With cretins of ten years of age, or even more, the relative infantile proportions

FIG. 134.—A TYPICAL CRETIN; TWO AND A HALF YEARS OLD. A patient in the Babies' Hospital.

FIG. 135.—SAME PATIENT AT SIX AND ONE-THIRD YEARS.

of the body are maintained. There is almost complete lack of growth at the epiphyseal junctions and there is great delay in the development of the centers of ossification. X-ray studies show that the nuclei of the tarsal and carpal bones may be absent until the tenth year and that the epiphyses of the long bones may not be ossified until the twentieth or thirtieth year. The subcutaneous tissue seems very thick and boggy, but does not pit upon pressure like ordinary edema. The facies is extremely characteristic. The head seems large for the body; the fontanel is often open until the eighth or tenth year, and it may not be closed even in

adults, but the cranial bones are often very thick; the forehead is low and the base of the nose is broad, so that the eyes are wide apart; the lips are thick, the mouth half open, the tongue usually protrudes slightly; the cheeks are baggy, the eyelids thick, the hair coarse, straight, and generally light-colored. The teeth appear very late and are apt to decay early. The second dentition may not begin until adult life.

Fatty tumors are quite constant in older children, although they are often wanting in infantile cases. They are seen in the supraclavicular region, just behind the sternomastoid muscle, sometimes in the axilla, or between the scapulae, and sometimes in other parts of the body. In distribution they are apt to be symmetrical, and are usually about half the size of a hen's egg. The neck is short and thick. No thyroid gland can be made out by palpation, but a small cyst may sometimes be felt at the root of the tongue. The chest is not deformed. The abdomen is large and pendulous. An umbilical hernia is almost always present The skin is dry, perspiration scanty, and eczema is common. The voice is hoarse and rough. Frequently patients may not walk until they are five or six years old, and then they waddle in a clumsy way. All the movements of the body are slow and lethargic, and everything indicates mental and physical torpor. The rectal temperature is usually subnormal. We had once an opportunity to observe an attack of acute bronchopneumonia in one of these cretins two years old. The symptoms and physical signs were typical, but during the greater part of the disease the rectal temperature fluctuated between 95° and 98.5° F. Only once was a temperature above 99° F. recorded. On account of their low temperature and torpid condition these patients are very sensitive to cold. They live upon a low plane of metabolism and the energy exchange is small. The mental condition is always greatly impaired. Some are even imbecile. Cretins are dull, placid, and good-natured, rarely troublesome or excitable; and when fifteen or eighteen years old they appear like children of three or four years. Speech may be impossible. The ability to say a few words is acquired late, and in some cases not at all. Almost invariably cretins suffer from constipation. At the age of puberty there is an absence of development of the sexual organs.

Diagnosis.—The diagnosis of the fully developed condition is very easy. The facial expression, the protruding tongue, the pendulous abdomen with umbilical hernia, the fatty tumors, torpor and low temperature are sufficient to characterize cretinism. The mistake is sometimes made of confusing Mongolian idiocy with cretinism. The former may be recognized by the peculiar formation of the eyes, the normal bone formation and growth and by the presence of the symptoms at birth. The therapeutic test with thyroid extract is conclusive.

Prognosis and Treatment.—There is no tendency to spontaneous

improvement. If untreated, cretins may live to an advanced age, but remain dwarfs, seldom attaining a height of more than three or three and a half feet. Their mental condition remains unimproved. Treatment with preparations of the thyroid gland brings about an extraordinary change. Transplantation of the gland has been employed as well as subcutaneous injection of extracts and the ingestion of fresh glands and various substances obtained from the gland. All these methods are

FIG. 136.—DR. J. P. WEST'S CASE OF CRETINISM, SEVENTEEN MONTHS OLD, BEFORE TREATMENT.

FIG. 137.—AFTER SIX MONTHS' TREATMENT WITH THYROID EXTRACT.

effective, but the preparation most employed is the dried, powdered gland, usually called thyroid extract, given by mouth. It is nearly a specific remedy for this disease. The improvement after its use is truly remarkable (Figs. 136 and 137). After a few weeks' treatment the entire appearance of the child is changed. The idiotic expression of the face is lost; the thickening of the skin and subcutaneous tissues disappears; there is a marked increase in height and in the circumference of the head; muscular power is rapidly developed, so that many soon become able to walk; and progress is seen in dentition, and in some older

girls in the establishment of menstruation. Intellectual progress is much slower than physical changes; however, nearly all the children become much brighter and more intelligent and learn to speak.

If treatment is begun early, physical development may be apparently normal, but normal mental development we have not seen, even in cases in which treatment was begun during the first year. We have under observation several cretins who have been treated from ten to fifteen years. Many of these children seem quite intelligent and are able to attend school, but without exception they are much below other children of their ages in mental and usually in physical development. As the thyroid gland is absent in these patients it is necessary for them to continue taking the thyroid extract as long as they live. If it is omitted relapses occur in a few weeks, even in cases well advanced toward recovery.

Most of the thyroid extracts on the market are prepared from the glands of the sheep. A reliable extract should be given if results are to be expected. The thyroid extract of Burroughs and Wellcome we have found to be more satisfactory than many of those on the market. Of this half a grain may be given once or twice a day at first; after the child becomes somewhat accustomed to it the daily dose may be gradually increased to five or six grains. Some disturbances are often seen at the beginning of the treatment—perspiration, marked irritability, and sometimes a rise in temperature—but these soon pass off. For old cases at least five grains daily should be given for an indefinite period.

HYPOTHYROIDISM

(*Infantile Myxedema*)

Cases of undoubted thyroid deficiency are met with that differ from sporadic cretinism in the time of their development and in the severity of the symptoms. Among them should be classed those cases closely resembling cretinism but not showing symptoms until the second or third year or even later and then only slightly marked symptoms. The deficiency of the thyroid under such circumstances occurs in extrauterine life or is incomplete. There are no pathological studies to show the condition of the gland and the etiological factors causing its degeneration are unknown. In a certain number of instances the condition has followed some acute infectious disease. The symptoms are those that have been mentioned under sporadic cretinism, differing only in degree. It is usually the failure of mental or physical development that first attracts attention; the child is unable to learn, pays

no attention to commands, is not cleanly in his habits, or he is much smaller than his fellows. More rarely he is noticed to have lost the ability to do things which he had formerly acquired. The height of these children is much below the average but the degree of dwarfism depends upon the time of onset of the thyroid deficiency. Some are greatly stunted, others less so; but normal growth does not occur and increase in height is very slight or absent. X-ray pictures show, as a rule, the presence of some carpal and tarsal centers of ossification which indicate that for a time at least the thyroid has been active. The facial expression varies from the characteristic facies of cretinism to one that is only slightly expressionless, stupid or stolid. The lips are apt to be somewhat thickened, the tongue also, but by no means always protruded. The hair is often coarse and generally thick. The children are usually well nourished, often stout. The skin is dry and thickened and the subcutaneous tissue firm. Fat pads are exceptionally present. The abdomen is usually large and in the more pronounced cases there is a hernia in the umbilical region. In the less marked cases this is often lacking. The children readily complain of cold. Constipation is frequent but by no means the rule. Dentition is late and irregular and the second dentition delayed. The voice is usually deep and hoarse.

FIG. 138.—INFANTILE MYXEDEMA.

These children are quiet and placid. Their intelligence varies according to the severity of the disease. Some are imbecile, some have quite a high degree of intelligence, so that, though several years behind their fellows, they are able to attend school. In the marked cases it is hardly possible to err in diagnosis. The mild cases can only be determined positively by the effect of thyroid extract upon the symptoms and especially upon growth. Thus, in one of our cases aged three and a half (Fig. 138) the height which had been stationary for some months increased nearly four inches in six months as the result of thyroid medication.

Treatment with thyroid brings about prompt improvement which will vary in extent according to the severity of the condition. Striking mental and physical improvement occurs. It is doubtful if complete intellectual development takes place. It is not to be expected that recovery of function in the diseased thyroid can occur. For this reason,

thyroid extract should be given continuously in the doses advised in the previous chapter. Mental and physical deterioration occur if its administration is interrupted.

GRAVES' DISEASE

(*Exophthalmic Goiter, Basedow's Disease*)

Typical Graves' disease in young children is rare. The determining cause of the perversion of the thyroid activity is unknown. Hereditary influences, especially goiter, Graves' disease and alcoholism are believed to play a part. Much more important is the effect of sex and age. Girls are affected three times as often as boys. As the age of puberty is approached the cases become much more frequent. Under five years of age Graves' disease is almost unknown. The youngest case that has come under our observation was in a girl of five and a half years. Between five and ten years a number of cases have been reported, but after ten years it is not very infrequent.

The disease as it occurs in childhood differs chiefly in two respects from the type seen in adult life. The symptoms develop and disappear with much greater rapidity, perhaps even in the course of a few days, and it is generally believed the outlook with the child is much more favorable.

Symptoms.—Attention is usually first called to the disease by restlessness and excitability or by the rapidity of the heart's action. Enlargement of the thyroid may not be evident at first but is regularly present at some time during the disease. The gland is generally uniformly enlarged, sometimes to a marked degree; it is firm, often hard, and can be felt to pulsate. With improvement in the symptoms there is a marked diminution in size, but a slight degree of permanent enlargement usually remains.

Exophthalmus is present in about four-fifths of the cases. It may be extreme. The ocular signs of von Stelwag and von Graefe are both present in the majority of cases. The fine tremor so commonly present with adult patients is usually lacking. Involuntary movements, if present, are generally coarse incoördinate movements. The skin is often fine and moist. Perspiration is readily excited, and flushing is frequent. Pigmentation is unusual. The heart's action is usually rapid and its violence is often complained of. A slight amount of cardiac dilatation may frequently be determined by physical examination. Nervousness is pronounced and is in most cases an early symptom. The children are constantly in motion and can be kept quiet with difficulty. The first improvement is often noticed in a diminution of the restless-

ness. The appetite is usually fair and the digestion good, but, as with adults, the increased metabolism which accompanies excessive thyroid activity causes loss of weight. Marked emaciation occasionally results.

The diarrhea, so troublesome a symptom with the adult form of the disease, is seldom marked. In general it may be said that the disease is milder than with adults and that its course is shorter. It may last only a few weeks but at times remains for several years.

The prognosis is relatively good. The mortality from recorded cases has not been more than 10 per cent, while recovery is the rule. There may remain indefinitely a slight degree of exophthalmus and enlargement of the thyroid and a tendency to cardiac palpitation with tachycardia.

The treatment should be directed toward securing, for a time at least, complete mental and physical rest. Everything tending to excite or irritate should be avoided. It is best to remove the child from contact with other children. Prolonged warm packs may assist in producing rest and in inducing sleep which should be encouraged in every way. As the nervousness diminishes mild exercise may be indulged in and according to the improvement of symptoms the normal régime gradually may be resumed. Studies, school attendance and contact with other children should only be allowed after many weeks or months and when a nearly normal condition has again been reached. The use of drugs, except occasionally, and for the relief of special symptoms, has no place in the treatment. Surgical measures are only to be considered when prolonged medical treatment has failed and when the progress of the disease is such as to threaten the life of the child. The indications for the various forms of operation are the same as with adults.

HYPERTHYROIDISM

Much more common than fully developed Graves' disease is the condition which is to be referred to a moderate increase of or perverted function of the thyroid gland. To this the term hyperthyroidism is applicable. The condition is found mostly in girls and usually between the eighth and fifteenth years. Several children in the same family may suffer from the condition and it usually occurs in distinctly neuropathic children. The chief symptoms are restlessness, irritability and nervousness. The children are constantly active. They are apt to be irritable and cry and laugh readily. They sleep badly and complain frequently of headache and of cardiac palpitation, especially upon exertion. Their appetite and digestion are usually good but there may be for some weeks or months moderate loss of weight and strength. A mild

degree of anemia is often present. Physical examination reveals in the majority of instances a slight enlargement of the thyroid gland which does not pulsate. Exophthalmus, beyond a slight staring expression of the eyes, is not found, and von Stelwag's and von Graefe's signs are absent. The heart's action is slightly exaggerated and rapid. Cardiac palpitation may be a cause of complaint. The hands of these children are apt to be constantly moist. The symptoms may last for some weeks or months. They usually disappear entirely, especially if proper measures are instituted, and in girls when menstruation becomes established. A marked increase in the severity of the symptoms is unusual, and the development of severe hyperthyroidism or Graves' disease from a mild form is rare. The treatment is the same as for Graves' disease—rest, quiet and removal from an exciting or irritating environment should be provided for. Tea, coffee and alcohol are to be entirely interdicted. The treatment is hygienic and not medicinal.

DISEASES OF OTHER DUCTLESS GLANDS

A large number of conditions which cannot be classified among any of the generally recognized diseases have been ascribed to disturbances of function of the various endocrine or ductless glands. It is necessary in most of these instances to assume that the disturbance is only functional since pathological changes are either entirely wanting or are recorded in an insufficient number of cases to establish a connection between the symptoms and the condition to which the symptoms are attributed.

Lesions of the pituitary gland seldom if ever produce acromegaly in children. Tumors of this gland or in its neighborhood may give rise to a group of symptoms known as "Fröhlich's syndrome," i. e., adiposity, delayed sexual development, increased sugar tolerance, and sometimes associated mental dulness.

Tumors of the pineal gland are in rare instances associated with precocious sexual development; tumors of the adrenals, more frequently. The exact association of the interference with the function of the glands and the precocious development is difficult to determine since the overwhelming majority of pineal tumors cause no such symptoms and because experimental removal of part or all of these glands in animals does not produce comparable effects.

Polyglandular disturbances affecting two or more of the ductless glands are held accountable for many conditions, particularly the various types of infantilism. This is an attempt, in the absence of any other explanation, to ascribe a train of symptoms to a number of organs whose

individual functions are largely unknown. At the present time our knowledge regarding the normal function of these glands and the results of their disturbed function is so very indefinite that it seems unsafe to ascribe to them, individually or collectively, an exact clinical importance. As yet this has not been established.

The use in practice of the various glandular extracts, though prevalent and increasing, has been in our experience with most unsatisfactory results. It can, however, be definitely stated that their administration by mouth is free from danger.

DISEASE OF THE THYMUS

STATUS LYMPHATICUS

The term status lymphaticus is applied to a very definite pathological condition which is associated with clinical manifestations, less constant and not characteristic. The relation between the lesions and the symptoms is little understood, and almost nothing is known of the etiology or pathogenesis. The most striking part of the lesion is the great enlargement of the thymus gland, with which is found a hyperplasia of the lymphoid tissues throughout the body, more marked than is seen in any other condition in childhood. The two most frequent symptoms are convulsions and attacks of asphyxia.

The status lymphaticus is most often seen between the sixth and twelfth months, but may be met with in children of any age. Enlargement of the thymus to a degree sufficient to be regarded as pathological, is not an infrequent condition. An association with rickets is often observed, but it is doubtful whether this is anything more than a coincidence.

Since the large thymus is so important a lesion, it is desirable to know what may be regarded as normal. The most extensive observations upon this point have been made by Bovaird and Nicoll, who weighed the thymus in 495 consecutive autopsies in children under five years. They found that the weight was greatest at birth, the average being 7.7 grams. After this time the change in weight was very slight for the period of five years, the average for the entire 495 observations being 5.9 grams, which was about the same as the average for each of the years taken separately. Excluding cases in which the organ was so large as to be considered abnormal (10 grams or over), the average weight at birth was 6.5 grams; during infancy and early childhood, 4 grams. The results of these observations do not differ essentially from those of Friedleben, which have been so extensively misquoted. It may therefore be

assumed that the average weight of the normal thymus at birth is from 6 to 7 grams; from birth to five years, from 3 to 4 grams. Anything over 10 grams may be considered abnormal.

In the status lymphaticus the thymus is often from five to ten times larger than normal. In the marked cases its weight is from 30 to 40 grams; in the less marked cases from 15 to 20 grams. The appearance of the enlarged thymus is well shown in the accompanying illustra-

Fig. 139.—Enlarged Thymus. The lungs, heart, and thymus are shown in the picture. The lungs have been turned back, showing the two lateral lobes of the thymus overlapping the heart; the central lobe, above, covers the trachea. *History.*—Breast fed, male child, nine months old, well developed; ill less than twenty-four hours; dyspnea, slight cyanosis, with death from asphyxia. T. 103° F. *Autopsy.*—Besides the large thymus there were present the general lesions of the status lymphaticus to a marked degree; lungs deeply congested.

tion (Fig. 139). A thymus of the size shown weighs about 45 grams, or 1½ ounces. In this instance it was nearly as large as one of the lobes of the lung. In general appearance, the enlarged thymus is rather more vascular than normal, but other than hyperplasia, shows no constant or essential changes, either by gross or microscopical examination.

The lymph nodes of the tracheobronchial region are greatly enlarged, often to the size of small cherries, and are found in great clusters. Those

of the mesenteric region may be still larger. Peyer's patches are very prominent, and the solitary follicles of the small intestine appear like mustard seeds upon the folds of the mucous membrane. Those of the colon are also very prominent. The lymphoid tissues about the pharynx and all the lymph nodes of the body are greatly hypertrophied. The spleen is usually enlarged, with prominent follicles. There are no other constant changes. Those present are usually accidental, depending upon the cause of death.

Symptoms.—In very early infancy this is one of the explanations of sudden death occurring after slight causes, and in some cases without any apparent cause. Death is often attributed to overlying, to asphyxia from aspiration of food, or to some other condition affecting respiration, or infants are simply found dead in their cribs without evidence of anything abnormal in history or symptoms.

Even in children who live until they are several months, sometimes several years, old, there may be nothing in their condition to indicate the presence of the status lymphaticus until something acute occurs. This may be in the nature of a slight accident, a surgical operation of a trivial character, the administration of an anesthetic, or some acute disease, frequently one affecting the respiratory tract. The symptoms associated with this condition are frequently of a nervous character, usually attacks of convulsions, or they affect the respiration, causing paroxysms of dyspnea, cyanosis, and even asphyxia. A frequent history is somewhat as follows: A child previously regarded as healthy, often well nourished and perhaps entirely breast fed, is taken with convulsions followed by high fever, preceding which there may have been some pulmonary symptoms suggesting a commencing bronchopneumonia. The convulsions recur at short intervals; the temperature remains steadily high; the signs in the lung are few and not proportionate to the other symptoms; and death occurs in from twelve to thirty-six hours often in convulsions.

In other cases convulsions are absent and the prominent symptom is asphyxia, which comes in paroxysms and may be so complete as to lead to the suspicion of laryngeal obstruction. If intubation or tracheotomy is performed, no relief follows. The child may die in the first severe attack, which may be preceded for a few hours by moderate dyspnea, or may come on almost without warning. It is more frequent, however, for the first attack to be less severe, the child perhaps being resuscitated with some effort, after which he may breathe almost as well as usual. In a few hours the attack of asphyxia is repeated; after several of these, each one growing more severe, death occurs. In these cases the elevation of temperature is usually slight and may be wanting.

Symptoms similar to the above but of less severity and resulting in

recovery would suggest status lymphaticus, although the diagnosis can not be established.

The cause of the symptoms is not definitely known. The asphyxia has been ascribed to pressure of the large thymus upon the lungs, the trachea, the pneumogastric nerves, or the auricles of the heart. Pressure would seem at times to be a factor in the production of the dyspnea, but apparently not the chief one. Constant dyspnea, even with a very large thymus, has never in our experience been present. It does not seem that the large thymus produces its symptoms mechanically.

There is another group of cases, perhaps the largest of all, in which there are no symptoms distinctly referable to the status lymphaticus, and yet this condition appears to be the factor which determines the fatal outcome of what was apparently an infection or an inflammation of only moderate severity. What is seen here is simply a greatly diminished resistance to disease. In these cases it is only the autopsy which reveals the explanation.

Diagnosis.—The diagnosis of enlarged thymus is possible only by physical examination, the symptoms being too indefinite to be relied upon. In percussing the thymus the child should be placed upon the back and the neck completely extended. In some cases of marked enlargement a definite area of dulness can be made out over the base of the sternum. The X-ray is also of distinct value, the shadow being sometimes so marked especially to the right as to be conclusive. Unfortunately in many, perhaps most of the cases, both these means of diagnosis give probable results only, so that while we may suspect the condition we can not do more. Marked enlargement of the tonsils and the adenoids exists so frequently without thymus enlargement, that this can hardly be regarded as suggesting the condition. The hyperplasia of the tracheobronchial or mesenteric lymph nodes or of the follicles of the intestine produces no especial symptoms.

Prognosis.—While this condition apparently may exist for an indefinite time without producing any symptoms, it undoubtedly often determines a fatal outcome of what might otherwise have been a mild illness or a trivial accident. It is especially important in connection with acute bronchitis and bronchopneumonia, with attacks of convulsions, with the shock of slight operations, and with the administration of anesthetics, particularly chloroform. It is one of the most frequent explanations of unexpected death from such slight causes as an exploratory puncture or even a hypodermic injection.

At present no known treatment has any influence upon the condition. There is experimental evidence that the X-ray produces involution of the thymus gland; but that it cures the condition of status lymphaticus in the human subject has not yet been established.

CHAPTER IV

DISEASES OF THE BONES AND JOINTS

OSTEOGENESIS IMPERFECTA

(*Osteopsathyrosis–Fragilitas Ossium*)

Of the etiology of this rare affection, little is known. No especial disease can be held responsible for it and the condition is not usually hereditary. It is at times, however, found in certain families associated with a peculiar blue coloring of the sclerotics, and in such circumstances is distinctly hereditary. In affected families those children with a tendency to fractures have blue sclerotics, but not all the children have this weakness of the bones. The explanation of the association is not clear.

Despite the etiological uncertainty the pathological changes are characteristic. They are found only in the bones but are present in all the bones, those formed in membrane as well as those formed from cartilage. The cartilage itself is in no way affected so that the growth of the bones in length is normal. The formation of bone, however, both from the periosteum and in the shaft, is greatly interfered with on account of deficient numbers and activity of the osteoblasts. The result is that the bony trabeculae are infrequent and small. Thus the bones are thin and very fragile. No changes have been demonstrated in any of the ductless glands.

The most striking feature of the disease is the fragility of the bones—the ease with which they undergo fracture. This takes place even in intrauterine life, so that infants are at times born with forty or fifty fractures and with greatly distorted extremities (Fig. 140). The majority of children with osteogenesis imperfecta are born dead or die shortly after birth. The bones of the skull are frequently so slightly formed that the whole cranium is soft and of a parchment-like consistency with widely separated sutures. As the result of the numerous intrauterine fractures, distinct shortening of the extremities may have taken place. Thus there may be at birth a certain similarity to the configuration of chondrodystrophy. This shortening can also be made out by the X-ray; but confusion of the two is impossible for the density of the bones is always greatly diminished and multiple fractures are almost always in evidence. Any of the bones, including the ribs, may be fractured.

Those infants who survive show a greater or less marked fragility of the bones. Fracture sometimes occurs from ordinary handling which

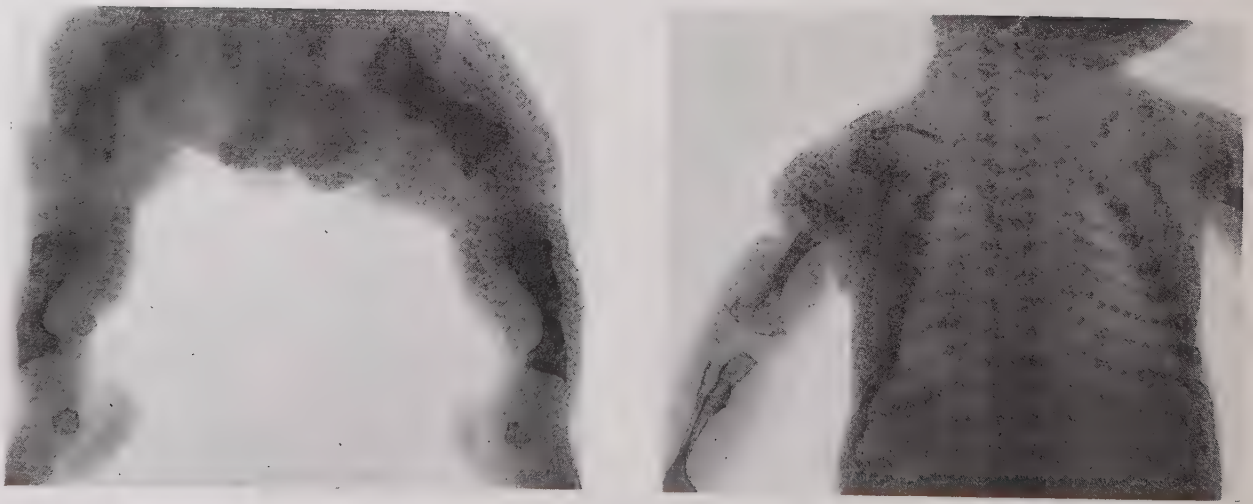


FIG. 140.—OSTEOGENESIS IMPERFECTA. Numerous fractures of all the long bones. (Infant still-born at full term.)

it is quite impossible to prevent, or in other instances only when a moderate degree of force is applied. Callous formation is slight and the process of repair of longer duration than with the normal child. In exceptional instances the fragility of the bones is only manifested after several years so that there may be no suspicion of any trouble until a number of fractures occur as the result of very little traumatism. Following the numerous fractures and the difficulty of healing, there is usually greater or less shortening and deformity of the bones. It may be extreme.

The progress of the disease varies much in the different cases; in some children there is no tendency to improvement; in others, usually in those in which the fragility is considerably less, there seems to be improvement in the condition of the bones so that about the time of puberty, or shortly after, fractures do not occur except when there is the application of unusual force. There is no known treatment that influences either the severity or the course of the disease.

CHONDRODYSTROPHY

(*Achondroplasia*)

This rather rare condition, often improperly called congenital or fetal rickets, is the cause of some of the most marked examples of dwarfism known. It was recognized as an abnormality by the early Egyptians and has figured in art in various ways since that date. Paintings show that many of the old court jesters were of this type. Because of their striking appearance, these dwarfs have always excited much curiosity and interest.

The causes of chondrodystrophy are unknown; only in rare cases has any hereditary connection been traced. The pathological process begins in fetal life and consists in a disturbance of the normal ossification of primary cartilage. It affects endochondral ossification only, never intramembranous ossification. The flat bones, therefore, escape entirely. The vertebrae are only slightly affected while the long bones of the extremities suffer most but not equally, though the disturbance is symmetrical. The humeri and femora are almost always the seat of the greatest interference with growth. One of the most striking changes in the skull is the synostosis or early ossification of the tribasilar bone; this is formed of two parts of the sphenoid and the sphenoidal process of the occipital bone. Normally this ossification does not take place until adult life; in children with chondrodystrophy it often begins *in utero*. This prevents a normal expansion at the base of the skull, and

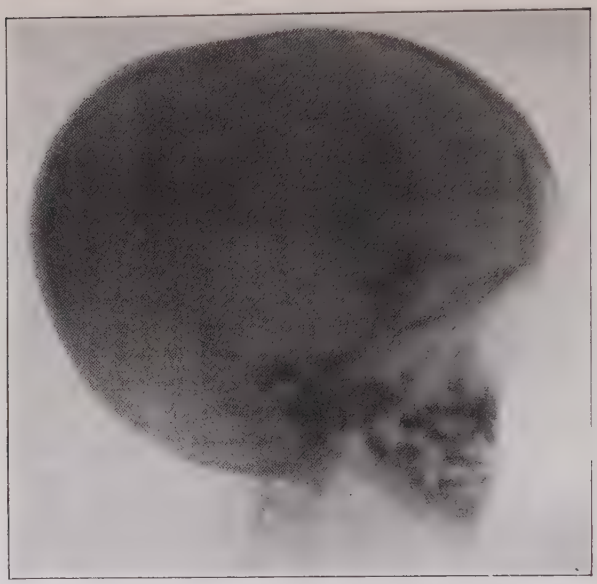


FIG. 141.—SKULL IN CHONDRODYSTROPHY, SHOWING FRONTAL PROMINENCE AND PROGNATHISM. Girl six years old.

the brain, as it grows, is thus crowded upward and forward, causing the great prominence of the forehead (Fig. 141). The upper jaw appears very prominent on account of the depression at the root of the nose.

FIG. 142. — NORMALLY DEVELOPED LONG BONES OF A FETUS COMPARED WITH THOSE OF CHONDRODYSTROPHY. (Spillmann.)

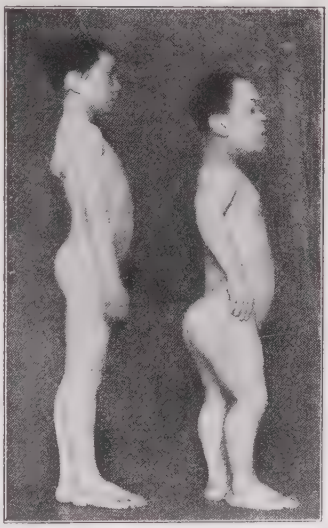
In the long bones there is a marked interference with the normal proliferation of cartilage cells. This interference may be seen in all degrees. In some cases a periosteal lamella pushes its way between the epiphysis and diaphysis, still further restricting the growth of the long bones. As bone formation beneath the periosteum goes on normally, the bones in chondrodystrophy are thick as well as short.

Symptoms.—The majority of children suffering from this condition are either born dead or die shortly after birth. Those who survive are delicate during infancy, but afterward may become strong and healthy. The most striking thing about their appearance is the very short legs and arms as compared with the length of the body. At birth the arms in many cases do not reach to the waist line,

and the length of the body may be less than the circumference of the head. The epiphyses appear somewhat enlarged, the abdomen is prominent, the skin of the extremities is in deep folds, the soft parts seeming to be much too abundant for the shortened bones (Fig. 143). In infancy these children are often quite fat. The facial expression is characteristic. There is usually a deep depression and flattening at the base of the nose, with a very marked prominence of the forehead. The head may not only seem large, but by measurement may be one or even two inches above the normal average. An erroneous diagnosis of hydrocephalus is often made in the early stage. Dentition is slightly later than normal, but not more so than is seen in moderate rickets. Marked relaxation of the ligaments and rather feeble muscular power often delay walking until the third or fourth year. If the head is large, the fontanel may not close till the fourth or fifth year. The so-called "trident hand" is characteristic. The fingers are very short and of nearly equal length, and an angular separation is seen at the second joint (Fig. 144).

FIG. 143.—CHONDRODYSTROPHY. INFANTILE FIGURE. (Marie.)

FIG. 144.—CHARACTERISTIC HAND OF CHONDRODYSTROPHY. (Marie.)



A B

FIG. 145.—*A*, NORMALLY DEVELOPED BOY, AGE EIGHT YEARS. *B*, TYPICAL CHONDRODYSTROPHY. AGE EIGHTEEN YEARS (Marie.)

These dwarfs are usually somewhat subnormal in their mental development but cannot be classed as defectives. They are good-natured,

often amusing, easily controlled, and frequently live to a great age. With advancing years the figure assumes a very peculiar and characteristic appearance. The prominent hips, with the marked lordosis, shortened extremities, and late bowing of the legs, present a striking picture (Fig. 145). The maximum height attained is often not more than three and a half or four feet. Although while young of feeble muscular power, later in life they often become very muscular. When adult life is reached the sexual powers are normal; if the women become pregnant, Cesarian section is almost always required on account of deformity of the pelvis.

In infancy, chondrodystrophy is often confounded with rickets, hydrocephalus, cretinism and osteogenesis imperfecta; but its features are so characteristic that the mistake can hardly be made if the child is carefully examined. In severe osteogenesis imperfecta the femora may be very short but the association with multiple fractures determines the diagnosis. No known treatment has any influence upon the condition. The use of the thyroid extract is entirely without effect.

ACUTE ARTHRITIS OF INFANTS

The terms *acute purulent synovitis, acute epiphysitis, pyemia of bone,* and *acute osteomyelitis,* have all been applied to this condition. The disease is really a form of pyemia. The causes and lesions may differ considerably in the different cases, but clinically they all have certain features in common, viz., an acute joint inflammation with suppuration.

The acute arthritis of infants is essentially a disease of the first year, and is much more frequently seen in the first six months. The inflammation may begin in the joint, at the epiphyseal junction, or in the medullary canal; but, however it may start, the joint is soon invaded. The nature of the arthritis varies somewhat with the exciting cause. When it is due to the gonococcus, it is usually confined to the joint; there is in most such cases a superficial inflammation involving the synovial membrane, but rarely leading to destructive changes in the cartilage, ligaments, or bone. When it is due to the streptococcus or staphylococcus, it may begin elsewhere than in the joint, which, however, is usually soon involved, and complete disorganization may follow. It may also result in a diffuse osteomyelitis, in a subperiosteal abscess, or a separation of the epiphysis. As a late result there may be a pathological dislocation or a "flail joint"; less frequently there is ankylosis.

Etiology.—The cause of acute arthritis in infants is the entrance

of pyogenic organisms into the circulation. In cases occurring in the newly born the most frequent organism is the streptococcus, at other times the gonococcus. Less frequently are found the staphylococcus or the pneumococcus and very rarely the influenza bacillus. In most cases occurring during the first two months of life, the portal of entry is the umbilical cord, though infection may take place through the skin, conjunctiva, genital tract, or the mouth. In the cases developing later it is often difficult to determine the point of entry, especially when the cause is the gonococcus. Of 26 cases of acute gonococcus arthritis observed in the Babies' Hospital, only 2 occurring during the first month could be classed as infections of the newly born. The cases were observed during a hospital epidemic of gonococcus vaginitis, and yet 19 were in male children, in no one of whom was there any genital lesion, and in only one was there conjunctivitis. Of the 7 cases occurring in girls, only 2 had vaginitis. The portal of entry in these cases could not be definitely determined. We have also observed isolated cases of gonococcus arthritis in the course of a gonococcus pyemia when it was impossible to determine the mode of entrance of the organism into the circulation.

Symptoms.—General symptoms often precede the local ones. In the most acute cases the temperature is high and widely fluctuating, accompanied by other symptoms of a severe infection. The earliest local symptoms are pain and tenderness, soon followed by swelling, which may develop quite rapidly in a single joint, or in several joints simultaneously. In those superficially situated there is redness of the skin, and fluctuation may be evident in three or four days. In cases coming on more gradually the temperature may be only from 100° to 102° F., and suppuration may not occur for two or three weeks. In the most severe cases the progress is rapid, one joint after another being involved, with general symptoms of pyemia, and death may occur in a week or ten days, usually from some visceral inflammation, pneumonia, pericarditis, or meningitis. In such cases blood cultures usually show the presence of the organism to which the infection is due. In the less severe type, which is more often seen, the symptoms may last for five or six weeks. When pus is not evacuated extensive burrowing often takes place.

In Townsend's collection of 73 cases, the joints were involved in the following order: hip, in 38; knee, in 27; shoulder, in 12; wrist, in 5; ankle, in 4; elbow, in 4; small joints, in 4. In three-fourths of these cases only a single joint was affected. In the 26 gonococcus cases referred to the localization was as follows: finger or metacarpus, in 20; ankle, in 18; knee, in 17; wrist, in 12; toe or metatarsus, in 10; shoulder, in 9; elbow, in 5; temporo-maxillary, in 1; hip, in 1. The average number of joints involved was 4 or 5, the largest number being

8. The tendency of the gonococcus infections to involve the small joints is striking.

Diagnosis.—When several joints are involved, the disease is often mistaken for acute articular rheumatism, which, however, at this age is so rare that it may be ignored. Blood cultures are of diagnostic value. Syphilitic epiphysitis resembles it in the localized tenderness and disability; but the rapid swelling and the severe constitutional symptoms are lacking.

Treatment.—Cold applications or wet dressings may be useful in relieving the symptoms. In some cases, most frequently when the cause is the gonococcus, the inflammation subsides without suppuration. In infections due to other organisms, suppuration almost invariably occurs and early free incision should be made, followed by fixation of the joint. The results depend in no small degree upon the promptness with which the pus is evacuated. In the gonococcus cases there may be complete recovery. In most of the others the functions are impaired.

The use of vaccines is to be advised in all these cases. The best results are seen in infections due to the staphylococcus and next, those due to the gonococcus. In such cases, autogenous appear to have little if any advantage over stock vaccines. Injections should be repeated every five or six days in increasing doses.

CHRONIC ARTHRITIS

(Atrophic Arthritis, Still's Disease)

Under the heading of chronic arthritis are probably included a number of chronic joint affections which as yet we are unable to separate. They all have as a common characteristic a crippling of the joints, not on account of primary changes of the bones or cartilages but as the result of lesions of the synovial membrane, capsule, ligaments and periarticular structures which may later cause secondary changes in the bone and cartilage. As there is no sharp line of demarcation between these conditions it is convenient to discuss them all under one heading.

Etiology.—The frequency with which these forms of arthritis begin in the young is very striking. They are often seen in children under three years of age, and the histories of those seen later often date back to this period of life. Boys are rather oftener affected than girls. While no history of infection may be obtained, in quite a number of instances the disease immediately follows or occurs shortly after some infectious disease or suppurative process. Scarlet fever and measles, particularly the former, are the exanthemata after which chronic arth-

ritis is most often seen. Demme has described, and we also have observed, very severe progressive arthritis following scarlet fever. The suppurative process which precedes the arthritis may be anywhere in the body—in the pleural cavity, the bones, the accessory nasal sinuses, the teeth or the tonsils. A history of rheumatism is not infrequently obtained. It is doubtful if at this age it is really true rheumatism, but rather an unusually acute onset of the arthritis with fever. Hemophilia with hemorrhages into the joint may be followed by severe joint lesions, but these are quite distinct from the condition now under consideration. Nor has this form of arthritis a close connection with syphilis or tuberculosis.

Pathology.—Early in the disease and for a considerable time the joint surfaces and the bones are not involved. The lesion is chiefly in the synovial membrane, joint capsule, ligaments and surrounding structures. The synovial membrane is thickened; its villous processes are hypertrophied and the membrane is hyperemic and edematous. After a time it becomes thickened by the growth of new tissue. The same condition occurs in the capsule. The joint itself may contain fluid; this is usually quite clear. Later, the cartilages may be somewhat eroded at their edges by the hypertrophied villi of the synovial membrane. Very rarely, and only after many years, there may be fibrous or even bony ankylosis. Except for this, the only changes in the bones themselves are atrophic. They show all grades of osteoporosis.

In a certain number of instances, changes in other viscera are found. The spleen and lymphatic glands may be increased to several times their normal size, but they show nothing characteristic. The lesion is merely hyperplasia. Very rarely, without apparent cause, general amyloid degeneration of the viscera is found.

Symptoms.—The onset may be acute with fever and with involvement of the joints almost coincident with the fever, or there may be swelling and articular pain and tenderness with no fever whatever. At other times there may be general symptoms for many weeks before the joints are found to be involved. We have seen one boy who had fever for nearly three months before the involvement of his wrists, which was followed rapidly by that of his ankles and knees. No matter what the mode of onset the joints usually involved are, in order of frequency, those of the carpus and phalanges, the wrists, elbows, ankles, knees, hips and the cervical spine. Rarely other joints such as the sternoclavicular and the maxillary are implicated in the process. The articular lesions are usually symmetrical, but may differ in severity upon the two sides. The joints are swollen and are moderately tender to the touch; on palpation they give a somewhat doughy sensation. They frequently contain fluid but usually not a large amount. The fluid may disappear and

re-accumulate rapidly. The appearance of the fingers is very characteristic, the first interphalangeal joint being the one earliest and most severely affected. The articular involvement causes flexion of the joints to a greater or less extent and this deformity increases with the progress of the disease. The pain is not great, nor is there tenderness upon pressure, but attempts to bring the joints into their normal position by active or passive motion are impossible both on account of pain and the changes in the peri-articular structures. The joints are often covered by fine, shiny skin. There may be no fever whatever, and only the articular swellings. In other circumstances, fever may be a prominent symptom. There may be a persistent elevation of temperature, a degree or two above normal or for weeks there may be daily exacerbations and remissions of several degrees. At times the fever disappears and may be absent for months, but when it has once been a feature of the disease it is likely to return. With the febrile form of arthritis there is usually enlargement of the superficial lymphatic glands, chiefly the inguinal and axillary. The cervical glands may also be involved and not infrequently the epitrochlears. The spleen is often enlarged and rarely the liver also. There may be albuminuria and casts in the urine. With all forms of chronic arthritis the general condition of the child suffers. There is usually a moderate degree of secondary anemia which is most marked in the febrile form. To the form of arthritis with fever and enlargement of the spleen and lymphatic glands, the name "Still's disease" is frequently applied.

An examination with the X-ray shows a thickening of the peri-articular structures, often distention of the joint, and a greater or less degree of osteoporosis. No osteophytes can be demonstrated.

There is a great difference in the rapidity with which crippling of the joints occurs. In one case as much damage may be done in a few weeks as occurs in years in another. Eventually motion in the extremities may be nearly impossible with the joints fixed in positions of extreme deformity.

The course is usually progressive from bad to worse. The crippling becomes greater and greater though the general health may remain fair. Death, in such circumstances, is due to some intercurrent disease, very rarely to amyloid degeneration of the viscera. If the cause of the disease can be removed, the prognosis is good so far as further deformity is concerned. Even when no cause can be discovered, arrest of the disease may occur, and at times recovery is almost complete, but this result is so rare as hardly to be expected.

Treatment.—This should always include a careful search for anything that might act as an etiological factor. Especially should septic processes in the tonsils, in the accessory sinuses and in the teeth be

sought. Unless the cause can be removed, treatment is merely palliative. The patient should be placed under the best hygienic conditions with as much life out of doors as possible. Apparatus should not be worn except to prevent deformity and to assist in walking.

TUBERCULOUS DISEASE OF THE BONES AND JOINTS

The chronic forms of tuberculous bone disease, on account of their insidious onset and the frequency with which they simulate other diseases, more frequently fall, in the early stage at least, into the hands of the physician than into those of the general or orthopedic surgeon. All that will be attempted in this chapter will be to outline in a general way the most important forms—viz., disease of the vertebrae, hip, and knee—dwelling particularly upon the early symptoms and diagnosis. For their fuller discussion, particularly as to the details of treatment, the reader is referred to text-books on general or orthopedic surgery. The causes are the same, and the lesions are very similar in all forms, and will therefore be considered together.

Etiology.—The age at which tuberculosis of the bones most frequently begins, is from the third to the eighth year, it being comparatively rare before the end of the second year. The sexes are affected with about equal frequency. Tuberculous bone disease may occur in a child who has previously been in apparent health, but more often in one who has been reduced by some previous illness, especially one of the infectious diseases; of these, it most frequently follows measles and whooping-cough. Of seventy-one cases in children investigated by Park and Krumwiede, or collected by them, the bacillus was of the human type in sixty-eight and bovine in but three instances.

A family history of tuberculosis is present in a large number, but by no means in a majority, of the cases. Like tuberculosis of the cervical glands, it is rarely preceded by other tuberculous processes, although it may be followed by them. It usually appears as an example of primary infection; but it is quite impossible that such should actually be the case. There has previously been a latent focus of tuberculosis elsewhere in the body. In many cases disease of the bronchial glands has been demonstrated by autopsy. Infection from these or from other tuberculous lymph glands is the most frequent point of origin of infection in cases of bone disease.

Traumatism is often an exciting cause, and it may determine the site of the disease.

Lesions.—The tuberculous joint diseases of childhood are, as a rule, secondary to disease of the bones. Hip-joint disease usually begins in

the head of the femur, and knee-joint disease in one of the condyles; ankle-joint disease in the lower epiphysis of the tibia, etc.

The frequency with which disease is seen in the different locations is shown by the following table, which gives the number of cases of each form applying for treatment at the Hospital for Ruptured and Crippled, New York, during ten years:

Spine	2,145 cases,	or 37.5 per cent.
Hip	1,937 "	" 34.0 " "
Knee	1,222 "	" 21.5 " "
Ankle or tarsus	255 "	" 4.5 " "
Elbow	71 "	" 1.2 " "
Wrist	50 "	" 0.9 " "
Shoulder	24 "	" 0.4 " "
Total	5,704	100.0

The character of the bone disease upon which chronic joint disease depends is generally a primary ostitis, which affects the articular extremities of the long bones, usually beginning near the epiphyseal line; in the short bones it is a central ostitis. The stages in the process are, congestion, swelling, and cell infiltration, followed by caseation, and frequently by softening and suppuration. In the early stage, the bone is slightly enlarged, and on section one or more yellowish foci of disease are seen. The disease may be arrested in this stage, encapsulation of the inflammatory products taking place; or it may continue until there is a more or less extensive breaking down or disintegration of the affected bone. As the disease extends there are involved the periosteum, the articular cartilage, and finally the joint itself. Abscess may form in the joint or in the soft parts surrounding the bone. The process is quite analogous to tuberculous disease of the lung. As the disease advances ligamentous attachments are loosened, and displacement of the parts occurs with the production of deformity, due partly to muscular contraction and partly to the weight of the body. The inflammatory process, with its resulting disintegration, generally goes on to a certain point, where it is arrested. Gradually the broken-down bone substance is separated and thrown off in small particles in the discharge, and a reparative process begins, with the formation of healthy bone. Where joint structures have been destroyed, cure takes place by bony ankylosis. Sometimes the disease finds its way to the surface without involving the joint; at other times the disease may be arrested, and its products become encapsulated within the bone. Inflammation of the joint may occur by a gradual extension of the inflammatory process, or by a sudden perforation of the articular lamella. As a result of extensive disease, all the joint structures may be affected—the synovial membrane,

ligaments, articular cartilages, and the cellular tissue surrounding the joint. The process of disintegration and that of repair are both very chronic and measured by months or years. The entire course of the disease is from one to ten years, three years being about the average duration. In the great proportion of cases but one joint is involved, although it is not infrequent in hospitals to see two, three, and sometimes four of the large joints affected in the same patient.

Secondary Lesions.—Abscesses form in a considerable proportion of the cases, and often burrow a long distance before they reach the surface. Amyloid degeneration of the liver, spleen, and kidney, and sometimes of the intestines, occurs as the result of the prolonged suppuration, chiefly in connection with disease of the hip or spine, occasionally with that of the knee. General or localized tuberculosis, particularly tuberculous meningitis, may develop at any time and prove fatal.

Caries of the Spine—Pott's Disease

This consists in a tuberculous inflammation of the bodies of the vertebrae, usually beginning in the central portion and extending to the periosteum, ligaments, cartilages, and, in fact, to all the contiguous structures. Secondarily it involves the membranes of the cord, the roots of the spinal nerves, and even the cord itself. The number of vertebrae usually affected is from two to five. The gross appearance of the lesion in a well-marked case is shown in the accompanying cut (Fig. 146). After the bodies of the vertebrae have become softened and partially broken down by disease, the pressure from the superincumbent weight of the body causes them to fall together and produces a backward displacement of the spinous processes, giving rise to the deformity known as kyphosis, which in its extreme form is popularly known as "hunchback."

Any part of the vertebral column may be affected; but the disease is most frequent in the dorsal region, as shown by the following statistics from the Hospital for Ruptured and Crippled: Of 2,143 cases, 72.5 per cent affected the dorsal region, 15.3 per cent the lumbar region, and 12.2 per cent the cervical region.

Symptoms.—The onset is gradual, often insidious, and the early symptoms are frequently overlooked or misinterpreted. The case may go on for weeks or even months before the true nature of the disease is recognized, which is often not until deformity has occurred. In nearly all cases, however, the early symptoms are sufficiently characteristic to enable a careful observer to make a diagnosis before the stage of deformity.

The most constant early symptoms are: (1) pains caused by the

irritation of the nerve roots and referred to various parts of the body, following the distribution of the spinal nerves; (2) rigidity of the spine from muscular spasm, this being an attempt to prevent motion at the seat of disease; and (3) the assumption of various postures calculated to relieve pressure upon the diseased vertebral bodies. Sometimes the first symptoms are those of pressure-paralysis; at others they are the local signs of abscess. In addition to the local symptoms mentioned, there is usually disturbed sleep, often accompanied by moaning.

Fig. 146.—Pott's Disease of the Upper Dorsal Region. A vertical section of the spine, showing disintegration of the bodies of the vertebrae and encroachment upon the spinal canal. (From a patient dying in the Hospital for Ruptured and Crippled.)

Cervical Disease.—The pains are often felt above the point of disease, frequently in the form of occipital neuralgia; sometimes they are referred to the front or the side of the neck. They may be so frequent and so severe that the face assumes a constant expression of anxiety or distress. In other cases pain is excited only by an attempt at movement. The muscular spasm most frequently takes the form of slight torticollis, sometimes of slight opisthotonus; sometimes there is simply a fixation of the head by a tonic spasm of all the muscles of the neck; both active and passive motion is resisted, and any movement may be so painful that the child involuntarily steadies his head with his hands. These symptoms come on gradually and are persistent. Sometimes they are overlooked, and the first thing to attract attention is a progressive weakness in the lower extremities, which proves to be the beginning of paraplegia. Occasionally the first marked symptoms are those due to the formation of a retropharyngeal or a retro-esophageal abscess.

The deformity from cervical disease develops much later than when the disease is located elsewhere. Usually the neck appears broadened or thickened in a nearly uniform way, and often the head seems to have settled downward upon the shoulders. In the lower cervical region a kyphosis is not infrequent; but in the middle and upper regions there is more often an anterior prominence, which may be felt in the posterior wall of the pharynx.

Dorsal Disease.—The referred pains are now below the seat of disease, and take the form of intercostal neuralgia or pain in the epigastrium or the abdomen. They are often ascribed to cold, malaria, indi-

gestion, or worms. There is a disposition to assume the prone position while sleeping, and also to lean across a chair or the lap of the nurse. The child walks carefully, holding the spine erect and very stiff, and exhibits great caution in getting into or out of bed, or in rising from a recumbent position. In the beginning there may be a slight lordosis, or forward curve at the seat of disease, instead of the usual kyphosis or backward projection, but the latter soon takes its place, and with it is seen the compensatory lordosis in the lumbar region.

Lumbar Disease.—The first symptoms here are often pain and lameness, referred to one of the lower extremities. This frequently leads to the suspicion that the hip is the seat of disease. In addition to the lameness there may be a tilting of the pelvis to one side, and sometimes quite a distinct lateral curvature of the spine. Referred pains are not so frequent nor so severe as when the upper part of the spine is affected; they may be felt in the groin, in the loin, in the thigh, in the buttock, or in the hypogastrium. The gait and attitude are very characteristic: Throwing the shoulders well back, the patient walks stiffly, with short steps, holding the spine with the greatest care. He rises from the floor awkwardly and with difficulty. Deformity is not usually so early nor so marked as when the disease is dorsal, and often before it is visible there are symptoms due to the formation of psoas abscess—lameness, flexion of one thigh, and a tumor deep in the iliac fossa or at the upper and inner aspect of the thigh; in both locations it has often been mistaken for hernia.

Physical Examination.—Whenever any of the above symptoms are present, the child should be stripped and submitted to a thorough examination, the purpose of which should be to determine, first, the existence of any deformity; secondly, the mobility of the spine; thirdly, the presence of any secondary lesions, such as abscesses or paralysis. The mobility of the spine is best determined by studying the attitude, gait, and posture of the child, and the manner of stooping or rising from the floor. The gait has already been described with the symptoms of lumbar disease. As it has been aptly put, "the child walks with his legs, but not with his back." In stooping, the same disinclination to bend or move the spine is seen. It is often impossible to induce the child to stoop at all, and when he does so, to pick up some object, there is acute flexion at the knee and hip, but as little bending of the spine as possible. In rising from the recumbent position the same thing is seen. The posture and attitude of the child will be modified by the position of the disease, and somewhat by the activity of the process at the time; however, by comparing the movements referred to with those of a healthy child, the great difference will at once be apparent. If the symptoms point to cervical disease, a digital exploration of the pharynx

for deformity or abscess should be made, and the extremities should be examined for paralysis. If the disease is in the lumbar region, deep palpation of the iliac fossa should be made to discover a psoas abscess, and the passive movements of the thigh should be carefully tested to determine whether there is any resistance to extreme extension, this often being present before the psoas tumor. No matter how clearly the lameness may be at the hip, it should be remembered that this often results from disease of the lumbar spine. If the thigh is flexed and freely movable except in extension, the symptoms are probably the result of psoas irritation, for in hip-joint disease the other movements of the joint are also resisted.

The deformity of Pott's disease is often spoken of as "angular" curvature of the spine. While this is a true description of the disease at an advanced stage, there is often in the early stage only a general curve. Later a slight knuckle is seen from the unnatural projection of a single spinous process. This deformity may increase and finally involve five or six vertebrae. It is usually greatest in the upper dorsal region. A slight prominence, which does not disappear on suspending the patient, is always suspicious.

Tenderness upon pressure over the spinous processes and increased sensitiveness to heat and cold are rarely present. Pain may sometimes be produced by downward pressure upon the head or shoulders in the axis of the spine. This symptom is not necessary for diagnosis, and the attempt to elicit it is strongly condemned by Gibney, who has seen serious harm follow such a test.

Course of the Disease.—Caries of the spine is a very chronic disease, its course being measured by months or years, but marked, as in all chronic diseases, by periods of remission and exacerbation. An exacerbation may follow traumatism, and is often accompanied by the formation of an abscess. After the disease has lasted from one to three years, the destructive inflammation usually ceases and repair begins, a cure being finally effected by a process of consolidation of the fragments of the diseased vertebrae, and the production of ankylosis. Relapses are easily excited by traumatism, by improper treatment, or by discontinuing the use of mechanical supports before the disease is quite arrested.

Abscesses.—The frequency with which abscesses occur depends somewhat upon the treatment. Townsend states that of 380 cases, abscess was present in twenty per cent. They are rarely seen earlier than three or four months from the beginning of symptoms, and usually belong to the second year of the disease. They sometimes form with acute symptoms, but more frequently they appear as typical cold abscesses. Those connected with cervical disease are retropharyngeal or

retro-esophageal, or they may open externally, usually just above the clavicle, in front of the sternomastoid muscle. Those with disease of the lower cervical and upper dorsal vertebrae are apt to burrow along the spine, appearing in the lumbar region; rarely they may rupture into the esophagus or the pleural cavity. Those with disease of the lower dorsal or lumbar vertebrae may open just above the iliac crest posteriorly, or burrow anteriorly between the abdominal muscles, but the usual course is for them to follow the psoas muscle, appearing in the groin just above Poupart's ligament or at the upper and inner aspect of the thigh.

Paralysis occurs in about one-half the cases in which the disease affects the lower cervical and upper dorsal vertebrae, but it is rare when the disease is below the middle dorsal region (see Compression Myelitis).

Prognosis.—The actual mortality of Pott's disease is difficult to state, so many of the consequences of the disease being remote and not fully appreciated until adult life is reached. The general mortality from all causes is from ten to twenty per cent. The causes of death are exhaustion from prolonged suppuration, amyloid degeneration, myelitis, general tuberculosis, and tuberculous meningitis. Sudden death occasionally occurs from pressure upon the cord in the upper cervical region, or from the pressure effects of abscesses in the posterior pharynx or in the posterior mediastinum.

The prognosis as to the amount of permanent deformity will depend upon the seat of the disease, the time at which treatment is begun, and upon the thoroughness with which it is carried out. The best results as to deformity are obtained when the disease is below the middle dorsal region. With improved methods of treatment begun early, a large number of these patients recover with an insignificant amount of deformity, and some with none whatever.

Diagnosis.—The spinal deformity resulting from Pott's disease may be confounded with rachitic kyphosis or with rotary lateral curvature. Rachitic curvatures are usually seen in children under eighteen months of age, a time when Pott's disease is rare; there are other signs of rickets present, and instead of rigidity there is usually undue mobility of the spine. What is true of rickets may be said of all curvatures depending upon malnutrition. In young children, especially, the tuberculin test is of considerable assistance in diagnosis.

Rotary lateral curvature is seen about puberty, rarely in young children except in connection with rickets. A slight lateral deviation of the spine, sometimes seen in the early stages of caries, may resemble a case of incipient rotary curvature. The latter is not attended by pain or rigidity, and is most frequent in young girls from eleven to fourteen years of age.

Other abscesses may be mistaken for those dependent upon vertebral caries. This difficulty is likely to exist in the cases attended by very little spinal deformity. These abscesses are most frequently in the iliac fossa or in the lumbar region, and may be due to perinephritis or appendicitis. The latter are more acute than those depending upon bone disease and usually accompanied by fever. Tumors of the vertebrae or of the spinal cord may give rise to symptoms almost identical with those resulting from compression myelitis due to Pott's disease. Both of these are rare (vide Tumors of the Cord).

Treatment.—The treatment of Pott's disease is both general and local, and neither should be neglected. The constitutional treatment should be similar to that employed in other forms of tuberculosis. The local treatment belongs to the domain of orthopedic surgery.

Articular Ostitis of the Hip—Hip-Joint Disease

In early childhood this generally begins as a chronic ostitis in the head of the femur, starting near the epiphyseal line. Exceptionally, and oftener in older children, it begins in the acetabulum. The pathological process, as well as the clinical history, is generally described as consisting of three stages. In the first stage—that of ostitis—the lesions are limited to the bone; in the second stage—that of arthritis—all the joint structures are involved, and in this stage suppuration usually occurs; in the third stage there is breaking down and absorption of the head and sometimes of the neck of the femur, which, with destruction of the ligaments, leads to marked displacement of the parts from muscular contraction. The disease may be arrested in the first or in the second stage, or it may continue through all three stages.

Symptoms.—Clinically, the usual duration of *the first stage* is three or four months; it may last only for a few weeks, it may extend over two or three years, and the disease may be arrested in this stage. The onset is usually very gradual, and the symptoms are often considered of trivial importance until they have continued for some weeks. Generally the first thing noticed is slight lameness, due to stiffness of the joint. In the beginning this may be seen only in the morning, wearing off during the day. It may be accompanied by some tenderness about the hip and a disinclination to walk. A little later the child complains of pain, which is most frequently referred to the front of the knee or the inner aspect of the thigh, but only in rare cases to the hip itself. This is slight at first, but gradually increases in frequency and severity, and soon there are added the "starting pains" at night, which are one of the most characteristic features of early hip disease. These pains are produced by a sudden spasm of the muscles during sleep. The child often cries out

sharply without waking, sometimes wakes with a cry; this is often repeated several times during the night. Soon restlessness and fretfulness during the day are present. The lameness, which at first was slight and occasional, or noticed only in the morning, comes to be a constant symptom, and week by week increases in severity. The evolution of these symptoms may take only a few weeks, but sometimes they come and go in the most inexplicable manner during a period of several months, or even one to two years, before they are fully developed.

Every child with a suspicious lameness, or with pains like those mentioned, should be stripped and submitted to a thorough examination. The first points to be observed on inspection relate to the general contour of the hip; every prominence and depression should be carefully noted. Then the attitude and gait should be studied; and finally all the functions of the joint should be carefully tested, and the limbs measured, to determine the existence of shortening or atrophy. At every step a comparison should be made with the sound limb. The contour of the hip is changed quite uniformly; there is broadening and flattening of the whole gluteal region; the trochanter is unnaturally prominent; the gluteal fold is shortened, and often single instead of double. There is no characteristic position of the limb in this stage. There is atrophy of the thigh and often of the calf In Fig. 147 is shown the appearance of a typical case in the full development of the first stage. In walking, the child favors the diseased side, throwing the weight as much as possible upon the sound limb; but all these symptoms are of much less importance for diagnosis than is an examination of the functions of the joint.

Fig. 147.—Hip-Joint Disease, at the End of the First Stage. Showing muscular atrophy, prominence of the trochanter, flattening of the gluteal region, and a single gluteal fold.

For this purpose the child should be placed upon a table upon his back, and the various movements of the hip—abduction, adduction, flexion, extension, and rotation—should be executed, first with the sound limb and then with the suspected one, the two being carefully compared at every point to determine the degree of motion allowed. It is not necessary that force should be employed or pain inflicted. If the

symptoms have existed for some weeks, there is generally a limitation of motion at the hip in all directions, but first usually in abduction, rotation, or extension. In more advanced cases, no motion whatever may be permitted at the joint, the pelvis tilting with the slightest movement of the femur. This fixation of the hip is due to tonic muscular spasm. Crowding the articular surfaces together, by pressure upon the heel or trochanter, produces pain, which is usually referred to the joint. This test should be carefully made, lest injury be inflicted. Examinations should not be made under ether, since in this way serious injury may be done unconsciously.

Second Stage.—This has been called the stage of arthritis. Its existence may be assumed when the limb takes the position of marked permanent deformity, which is due at this period to muscular action, not to destructive bone changes. The transition from the first to the second stage is in most cases a gradual one, and the line between the two can not be sharply drawn; sometimes, however, it is rapid, and marked by a sharp exacerbation of all the symptoms. This may indicate a sudden perforation of the joint and the rapid development of suppurative arthritis. Such is the usual result when an abscess which has been slowly forming in the bone opens into the joint; or acute joint inflammation may be lighted up without so evident a cause. Sometimes the pus reaches the surface below the capsular ligament, and the joint remains intact. An acute exacerbation is indicated by increased pain, excessive tenderness about the hip, often by inability to walk, or even to bear any weight upon the limb, and frequently by fever. The position assumed by the limb is now fairly characteristic. The foot is generally everted, the thigh slightly flexed and rotated outward, and the limb apparently lengthened. There may be infiltration anywhere about the hip, due to the formation of an abscess. The muscular spasm is so great that the joint is locked—no motion whatever being allowed. Abscesses may form at any point about the hip; they are especially frequent at the upper and outer aspect of the thigh, and may burrow long distances before reaching the surface. The duration of the second stage also is indefinite, but it usually lasts from a few months to a year, or the disease may be arrested in this stage.

Third Stage.—There is now marked deformity, which is the result of muscular contraction after absorption of the head and sometimes the neck of the femur, and destruction of the ligaments. The position of the limb is a very constant one, and resembles that present in dislocation upon the dorsum of the ilium. There is shortening of from one to four inches; the thigh is strongly flexed, adducted, and rotated inward, and the foot is inverted; the trochanter lies against the outer surface of the ilium, and is above Nélaton's line. In this position the joint may be-

come ankylosed. The displacement usually comes on gradually, but it is sometimes so sudden as to be mistaken for a true dislocation, although the latter is exceedingly rare in the course of hip disease.

There is now marked atrophy of all the muscles of the limb, and the thigh may be two or three inches smaller than its fellow. No motion at all is usually allowed at the hip, but this is compensated for to some degree by the exaggerated mobility of the lumbar spine. The spinal curvature—lordosis—is very marked both upon standing and walking. The duration of this stage may be several years. From time to time exacerbations occur, often excited by falls, and accompanied by the formation of new abscesses. In protracted cases, all the soft parts about the hip may be seamed with cicatrices from old sinuses. After the disease has gone on to the third stage, cure can take place only by ankylosis.

Diagnosis.—The important point in the early diagnosis of ostitis of the hip, is the gradual evolution of the symptoms, the most characteristic of which are lameness, "starting pains" at night, and impairment of all the functions of the joint. Mistakes in diagnosis most frequently arise from a failure to obtain a careful history, and from relying too much upon the symptoms of lameness and deformity. The essentially chronic character of the disease should constantly be borne in mind. In the vast majority of cases, with a careful history and a thorough examination, there can be but little doubt as to the diagnosis except at the very outset. The proportion of obscure and irregular cases to those following the regular course is small.

In the early stage, hip-joint disease may be confounded with a strain of the joint, with muscular rheumatism, poliomyelitis, periostitis of the shaft of the femur, phlegmonous inflammation in the neighborhood of the joint, or with caries of the lumbar spine. In the second stage there is even less difficulty in diagnosis, although abscesses resulting from perinephritis or appendicitis have been mistaken for those arising from hip disease. In the third stage, a mistake is almost impossible.

Prognosis.—This is to be considered both with reference to life and limb. The records of the Hospital for Ruptured and Crippled show the mortality of hospital patients with hip disease to be nearly twenty-five per cent. This includes deaths directly or indirectly traceable to the disease. The causes are nearly the same as in caries of the spine—exhaustion from prolonged suppuration, amyloid degeneration, and general tuberculosis or tuberculous meningitis.

Under the most favorable conditions, the disease may be arrested in the first stage, and recovery occur without lameness or any noticeable impairment of the joint functions. This result, however, is not often obtained, because the disease is usually well advanced before it is recog-

nized, or because of the difficulty in the way of carrying out all the details of treatment in the best possible manner. If the disease has advanced to the second stage and suppuration has occurred, there always results some impairment of the joint functions; usually there are decided lameness and marked muscular atrophy, but very little shortening or deformity, provided the limb has been kept in the proper position. If the disease has advanced to the third stage, there are always marked shortening, deformity, and lameness.

Treatment.—The indications for constitutional treatment are the same as in caries of the spine. The purpose of local treatment is to secure constant and complete rest for the diseased parts, and to prevent deformity. It should be in the hands of an orthopedic surgeon.

Articular Ostitis of the Knee—Knee-Joint Disease—White Swelling

Ostitis of the knee usually begins in one of the condyles of the femur, the inner much oftener than the outer one; less frequently it begins in the head of the tibia. The pathological process is very much like that at the hip. There is in the first stage a central ostitis accompanied by infiltration and expansion of the part of the bone affected. The disease may remain limited to the bone, the inflammatory products becoming encapsulated, or softening and breaking down may occur, with the formation of an abscess. Gradually the process extends outward, and the periosteum and the soft parts are involved. The disease may invade the joint itself in a destructive inflammation, or pus may escape externally without seriously involving the joint structures. The degree to which the joint is involved varies much in different cases; there may be only a simple synovitis, a suppurative arthritis, or a destruction of the cartilages and articular ends of the bones, synovial membrane, and ligaments, so that in the advanced stage all traces of a joint structure are lost.

If the process remains limited to the bone, recovery may take place with very little impairment of the joint functions. If suppuration in the joint has taken place, there will be more or less stiffness and fibrous or bony ankylosis. When there is destruction of the ligaments and articular ends of the bones, the limb assumes a characteristic position—the joint is flexed, the tibia is displaced backward and rotated outward, and there is marked over-riding of the femur. Bony ankylosis in this position is often seen.

Symptoms.—The earliest symptoms of disease at the knee are usually a slight stiffness of the joint, with a disposition to flexion and slight lameness. At first these symptoms are noticed only occasionally; finally they become constant and there is pain, which is usually referred to the

knee. In some cases there are "starting pains" at night, although these are less constant and less severe than in hip disease. Swelling is noticed early, as the diseased parts are superficial. At first this is chiefly of the bone itself; the condyle, usually the inner one, is enlarged and elongated, often to a marked degree, before there is any infiltration of the soft parts. Later there is a general fusiform swelling, involving the entire joint and effacing all the normal outlines. Some tenderness upon pressure over the bone affected is present quite early, and there may be atrophy of the muscles of the thigh and calf. The knee is flexed and slightly rotated outward, the position which secures the most complete relaxation of the joint structures. In some cases there is seen the characteristic swelling due to distention of the synovial membrane. Abscesses may form anywhere about the joint; very frequently they burrow beneath the tendon of the quadriceps extensor as far as the middle of the thigh. Gradually the deformity increases until the leg may be flexed at a right angle, and rotated outward over an arc of twenty or thirty degrees.

The course of the disease resembles that of ostitis of the hip and the spine. During periods of remission pain and tenderness often subside for several months so completely as to lead to the supposition that the disease has been arrested. An exacerbation is often excited by a fall or a strain of the joint, or it may follow an attack of acute illness. The disease may then progress rapidly and abscess after abscess form, with extensive destruction of all the joint structures and the production of permanent deformity.

Prognosis.—The danger to life is considerably less than in disease of the hip or spine. Death, however, results from the same causes—exhaustion, amyloid degeneration, and general tuberculosis or tuberculous meningitis.

With an early diagnosis and proper treatment the disease may, in a considerable proportion of cases, remain limited to the bone, and the resulting lameness and deformity be very slight; but otherwise a certain amount of lameness results from the stiffness of the joint. This may be due either to fibrous thickening or to bony ankylosis. Nearly all patients are able to walk without crutches, and if proper treatment has been carried out there is neither marked shortening nor deformity, although there is always great muscular atrophy.

Diagnosis.—The important symptoms for diagnosis are the gradual onset, the early swelling which is due to enlargement of the bone, and the constant lameness and deformity. The disease may be confounded with rheumatism, with synovitis, and even with scurvy. In all these cases the resemblance exists only during the period of exacerbation. A careful history, however, will usually make the diagnosis clear.

Treatment.—The general treatment is the same as in other forms of joint disease. The indications for local treatment are the same as in hip disease.

Tuberculous Osteomyelitis

This disease is rarely seen except in the short tubular bones, most frequently those of the hand and fingers. From this fact it is often called *scrofulous* or *tuberculous dactylitis.* It is described by many writers under the name of *spina ventosa.* Unger gives the following figures showing the frequency with which the different bones were affected: fingers in 43, toes in 3, metacarpus in 41, metatarsus in 14, radius in 2, ulna in 2, tibia in 3, jaw in 3. The first phalanx of the index finger is the bone which is most frequently the seat of disease. In the majority of cases the process is confined to a single bone, although it is not rare to see five or six affected. In such cases the disease is seldom symmetrical. The process is a chronic inflammation, beginning in the center of the bone with the deposit of tuberculous material. The swelling which follows causes an expansion of the bone and thinning of the shaft, until a mere shell may remain. The later changes are inflammation of the periosteum and the soft parts, the formation of abscesses and sinuses, necrosis, the exfoliation of sequestra, etc. The entire disease lasts from one to three years, and causes in most cases marked deformity.

Tuberculous dactylitis is essentially a disease of early childhood, being seen most frequently during the second and third years. The disease frequently appears to be the only tuberculous lesion in the body, but tuberculosis of other parts, especially other bones, may be associated.

Symptoms.—The disease usually begins as a painless enlargement of one of the phalanges, most frequently the first one of the index finger. It may be two or three months before it is of sufficient size to attract much attention. Exceptionally the inflammation is a more active one, and is accompanied by both pain and tenderness. The swelling is quite characteristic; it is smooth, hard, uniform, and generally spindle-shaped, involving the entire phalanx of the affected finger. The appearance of a severe typical case is shown in Fig. 148. Later there is discoloration of the skin, and usually there is suppuration. The abscess generally opens at the side of the finger, and a curdy pus is evacuated. If the opening is enlarged by an incision there is found a cavity partly filled with caseous matter, and dead bone is felt, and perhaps a loose sequestrum. The cavity is surrounded by a thin shell of new bone, which is formed from the periosteum. If no operation is done the discharge continues for weeks or months, other abscesses often form, and finally several small sequestra are exfoliated—sometimes a single large one—which is the shell of the diseased phalanx almost entire.

In some cases the disease is arrested before necrosis occurs, but in the majority this is not so. After the wounds have all healed the finger remains shortened, deformed, and often useless. In some cases the disorganization is so extensive that amputation is necessary.

Diagnosis.—The recognition of dactylitis is usually easy, but as symptoms almost identical may be seen in a syphilitic inflammation, it is often difficult to tell with which of the two forms one has to deal. The tuberculous form is much more frequent and is usually seen in children over two years of age; it may occur in a patient with tuberculous antecedents, or it may be associated with other tuberculous

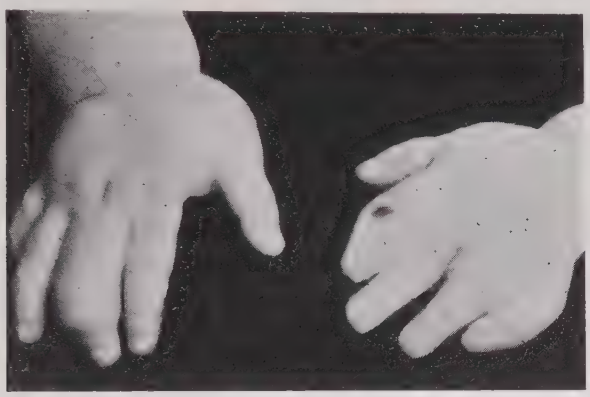


Fig. 148.—Tuberculous Dactylitis.

lesions. Syphilitic dactylitis is distinguished by the fact that it is more often seen in young infants, that the lesion is more frequently multiple, that it is often symmetrical, and that other manifestations of syphilis are generally present. The Wassermann and the tuberculin tests give definite information in nearly all cases.

Treatment.—Painting with iodin and like measures are useless. The diseased part should be kept at rest—if a finger, by the application of a splint. Every means should be taken to build up the patient's general health, as this is the most effective way to influence the local process. The general verdict of surgeons is against early excision as a means of arresting the disease. Abscesses should be opened early and freely, all diseased bone removed, the finger kept in proper position, and the wound treated according to general surgical principles. Under almost any treatment the disease is a protracted one, and rarely lasts less than a year.

CHAPTER V

DISEASES OF THE SKIN

The skin at birth is covered with a whitish sebaceous secretion, the vernix caseosa. The skin itself is of a deep-purplish color, which changes to a bright red over the face and trunk in a few minutes, with the establishment of normal respiration, and in a few hours the whole body has the same tint. This excessive redness slowly fades during the first month, at the end of which time the skin has assumed the pale pink of infancy. On the third or fourth day there may be seen the first signs of physiological icterus; this generally disappears by the end of the second week.

The epidermis which is present at birth soon loosens and is thrown off. This normal desquamation usually begins upon the fourth or fifth day, and is completed in ten days or two weeks. If the skin is frequently oiled and properly bathed, desquamation is scarcely noticeable unless a close examination is made. In some infants, especially those who are delicate and cachectic, it is very much more marked.

Perspiration is rarely present before the end of the fourth month, and is then seen only upon the forehead. In healthy infants it is scarcely noticeable during the first year. Copious perspiration is most frequently a symptom of rickets; less marked perspiration may occur with any general weakness or during acute illness.

CONGENITAL ICHTHYOSIS

Congenital, or more properly fetal, ichthyosis in its severe form is a rare disease, characterized by the formation, usually all over the body, of a thick, horny epidermis resembling parchment. This is divided by fissures or shallow furrows into irregular patches; sometimes these are two or three inches wide, at others as small as a pin's head. In its milder form it is not uncommon. The disease begins in the early months of fetal life, and is an abnormality in the development of the skin, there being an excessive proliferation of the layers of the epidermis.

Symptoms.—In the gravest form of the disease the child often lives but a few hours, and rarely more than a week. The openings of the nostrils and the ears may be occluded by the excessive production of epithelial cells. The eyes are in a condition of ectropion, and there are often deformities of the mouth and other orifices due to the contractions

of the skin. The nails and hair are usually imperfectly developed. The body seems encased in a hard, horny covering, and looks as if it had been varnished or covered with collodion. The skin cracks or splits and the edges curl up, an appearance which has been aptly compared to the skin of a boiled potato.

In the milder form, the duration of life is indefinite, depending upon the degree of development of the disease; but even in such cases there may be seen the deformities at the orifices of the body, and there may also be a continued exfoliation of the epidermis in large irregular patches. After this has separated, the skin beneath appears red and moist, but gradually becomes dry, hard, and shining, slowly contracting until it splits in various directions.

Fig. 149.—Congenital Ichthyosis, Six Weeks Old.

The outlook is unfavorable in all cases; in most of the severe forms death occurs in infancy, but in some of the milder ones, life may be prolonged indefinitely. The "alligator boy" of the "Dime Museum" is an example of this class.

Treatment.—The indications are to keep the skin moist and soft by the use of oils, continuous baths, etc., and to prevent infection by perfect cleanliness. Although a certain amount of improvement usually follows these measures, a cure is not to be expected.

MILIARIA

The term miliaria is applied to an obstruction of the sweat glands, which may occur either with or without inflammation. The non-inflammatory form is known as *sudamina* the inflammatory forms as *miliaria rubra, miliaria vesiculosa* and *miliaria papulosa.*

Sudamina.—In this form there is no inflammation. The sweat ducts, according to Crocker, are blocked by an accumulation of epithelial cells

while no perspiration is going on; and when the process is restored the fluid, being unable to escape, accumulates in the form of tiny vesicles. These appear like small pearly bodies very closely set, and disappear in the course of a few days by absorption. Fresh crops may appear from time to time. Sudamina may be seen in any of the continued fevers or exhausting diseases. It requires no treatment.

Miliaria Rubra.—This condition, also known as *red gum, strophulus*, etc., is a sweat rash, usually seen in young infants as the result of excessive clothing. It is most frequently observed upon the cheeks and neck, often upon the side of the face upon which the infant sleeps, or the side held against the mother's body while nursing, if this is done upon only one breast. The eruption consists of scattered red papules, sometimes with tiny vesicles. Miliaria rubra is an inflammation about the sweat glands, the result of which is a retention of their secretion. There is generally little or no itching. The treatment consists in the removal of the cause, and the application of some absorbent powder, such as boric acid and starch.

Miliaria Papulosa (*Lichen Tropicus, Prickly Heat*, etc.).—This is the most common and most important variety of miliaria. There is in this disease an obstruction of the sweat glands by inflammatory products. The lesion consists in the formation of bright-red papules, which are very closely set, the summits of some of them being surmounted by tiny vesicles, and here and there in severe cases even small pustules may be seen. If not interfered with by scratching, the vesicles dry up without rupture, and are followed by a slight desquamation. Where there is much scratching, an eczematous condition may result. Miliaria papulosa comes out with great rapidity, especially upon the neck, forehead, back, and chest. It is accompanied by an almost intolerable itching and stinging sensation. Over other parts of the body profuse perspiration occurs. The disease is produced by very hot weather and excessive clothing. Although the duration of a single attack is but two or three days, in susceptible patients it may keep recurring for weeks, being exceedingly intractable. Where there is much scratching, the resulting eczema is very troublesome. It is not infrequently followed by furunculosis.

The diagnosis of miliaria rubra and miliaria papulosa is usually easy. They are distinguished from eczema by the suddenness with which they appear, by the associated sweating of other parts of the body, by the transitory character of the eruption, and by the fact that the rash never occurs in circumscribed patches.

Prickly heat is to be prevented by light clothing, frequent bathing, and the plentiful use of a good toilet powder, such as boric acid and starch. The skin should be protected against the irritation of flannel undergarments by the interposition of silk or linen. When the inflam-

mation is at its height, relief is obtained by the application of a calamin and zinc lotion, or by a dilute solution of the acetate of lead; carbolic acid may be added to either, when the itching is intense. In some cases bland powders are preferable to lotions.

SEBORRHEA

Seborrhea is considered by dermatologists generally, as a functional disease of the sebaceous glands; although Unna regards all such cases as parasitic in origin and inflammatory, and classes them as seborrheic eczema. The disease may affect almost any part of the body, and children of any age, but the most frequent form is that which is seen upon the scalp in young infants. This is the most important variety, and the only one which will be here considered.

Seborrhea of the scalp is characterized by the formation upon the vertex, of dirty-yellow crusts, which are soft, greasy, and friable. They are composed of epithelial cells, fat-globules, and granular masses, to which is always added dirt. In neglected cases the hairy scalp is nearly covered by a dense crust, which may be as thick as heavy pasteboard. If the crusts are removed the underlying scalp may be found perfectly healthy, but more frequently, in cases of long standing, it is eczematous. The eczema is set up by the decomposition of the exudation, or by the efforts to remove the crusts by such means as the fine-toothed comb, commonly employed in domestic practice. There is little tendency to spontaneous improvement or recovery, and the condition often lasts for months. Every seborrhea should be treated, for when neglected it furnishes a favorable soil for the development of eczema.

Only local treatment is required. The crusts are first to be softened with oil, and then removed by washing thoroughly with warm water and soap, after which an ointment of resorcin, 2-per-cent strength, or of sulphur, 10-per-cent strength, should be applied. The oil and soap and water are repeated every few days, or as often as the crusts form. In the meantime the scalp is kept covered with the ointment.

ECZEMA

Eczema is the most frequent and altogether the most important disease of the skin in early life. The scope of the present work permits only a discussion of such features and varieties as are peculiar to infants and young children. The eczema of older children does not differ in any essential points from that of adults.

Etiology.—The conditions in infancy which predispose to eczema are, first, that the skin is extremely delicate, and hence more easily affected by external irritants and microörganisms; secondly, its more intense glandular activity. While all children are susceptible, there are certain ones in whom the susceptibility is very marked, and in them the slightest amount of external irritation, or the most trivial disturbance of digestion may produce a severe eruption. Eczema is one of the chief manifestations of the exudative diathesis (Czerny). It is especially prevalent in some families and is not infrequently inherited with the other evidences of the diathesis. Eczema is common in fat, healthy-looking infants, both in those who are nursing and in those who are artificially fed. It rarely occurs in poorly nourished children. Children with eczema are not infrequently subjects of asthma in later life. Eczema may apparently be initiated and is certainly aggravated by overfeeding, whether it be with breast milk or artificial food. The food element which seems to be particularly to blame is the fat, but farinaceous food in excess has also a bad effect. Schloss and Blackfan have shown that there is a susceptibility to animal protein on the part of most patients with eczema, as shown by cutaneous tests with various proteins. Most of the patients are susceptible to egg white and many to cow's milk. A few are susceptible to woman's milk. Some children even with severe eczema are insusceptible. The exact meaning of this susceptibility is not clear.

The exciting causes of eczema may be external or internal. Of the former the most important are heat, cold dry air, and winds—as in the familiar chapping of the face—the use of "hard" water or of strong soaps in bathing. The disease may be due to the irritation of clothing, to want of cleanliness, or to irritating discharges from mucous surfaces, as in the eczema of the upper lip, thighs, or buttocks. It accompanies most of the parasitic skin diseases, particularly pediculosis, scabies and ringworm.

What part is played by microörganisms in the etiology of eczema has not yet been fully determined. As a primary factor they do not seem to be of the first importance. Secondary infection, however, occurs in most cases, and this is important in keeping up the disease.

Simple Chronic Eczema—Eczema Rubrum.—This is the most frequent form of eczema occurring in infants and young children, and is usually seen upon the face. It affects by preference the cheeks, forehead, and scalp, not infrequently the ears and neck, and may occur upon any part of the body. Upon the trunk and extremities the eruption is usually in patches, but in rare cases may cover nearly the entire body. The disease generally begins upon the cheeks with the formation of small red papules; later these coalesce, and there is a moist, red surface exuding

serum. The secretion dries and forms thick, gummy crusts, which may be so hard as to form a mask for the face. From the scratching caused by the almost intolerable itching, the surface bleeds freely, and the dried blood gives to the crusts a dirty-brown color and adds to the distressing appearance. The skin is often much swollen. After the removal of the crusts there is seen, in acute cases, a red, inflamed, granular surface, moist and bleeding readily. When the process is less active, there is redness, thickening, induration, and scaliness of the skin, and marked itching. In the same case these stages may alternate, exacerbations occurring whenever the exciting cause is particularly active. From the cheeks the disease spreads to the forehead, ears, and scalp, and here similar lesions are seen. Upon the trunk and extremities thick crusts rarely form, but the skin is red, thickened, and scaly. The parts most often affected are the forearms, legs, abdomen, and back; occasionally the eruption is general. Eczema of the occipital region of the scalp is usually due to pediculosis.

Swelling of the lymph nodes in the neighborhood of the eruption is a constant feature of eczema of the face and scalp; these may reach the size of a chestnut or walnut, and occasionally they may suppurate. Intense itching is a characteristic feature of all cases of eczema of the face or scalp.

While most children with eczema are well nourished in the beginning, and some remain so during a prolonged attack, the general health of many is undermined. The itching and discomfort cause constant irritability, loss of sleep, and other nervous symptoms which sometimes seriously impair the child's nutrition.

The effects of very extensive eczema resemble in some particulars those of burns of the second degree. There may be fever, delirium, other nervous symptoms and even a fatal termination. We have seen several cases with a generalized eczema in which there developed, without evident cause, exceedingly high temperature, in two cases reaching 109° F., accompanied by symptoms of a most profound intoxication. Most of the infants with such symptoms die, but one child recovered in whom the temperature mentioned was reached. No satisfactory explanation of these severe intoxications has yet been offered.

There are some patients in whom an alternation of eczema and attacks of bronchitis with asthma may occur. During the eczema, the pulmonary symptoms are entirely wanting; but when the eczema is relieved the pulmonary symptoms rapidly develop. In a few patients an alternation of eczema and diarrhea is observed.

Patients with eczema are exceedingly prone to develop attacks of diarrhea and this condition nearly always brings about a marked improvement in the skin, though the diarrhea is often difficult to control.

Eczema of the face is very chronic, easily improved, but cured only with great difficulty. There is a strong tendency to relapse, brought on by neglect of local treatment, by any digestive disturbances, or by overfeeding.

The predisposition to eczema often ceases with the second year; those who have suffered from it almost constantly during infancy may be free from it during the remainder of childhood. This is in part to be explained by the loss of fat in consequence of more active exercise and a diet which is more largely nitrogenous. When the disease continues through the third and fourth years, the associated infantile condition, obesity, is not infrequently present.

Pustular Eczema of the Scalp.—This condition, often called "simple impetigo," is less frequently seen in infants than in children from two to five years old. There are usually present from half a dozen to fifty greenish-yellow crusts matting the hair, usually discrete, but sometimes coalescing to form a mask over half the scalp. There is very little itching, in some cases none at all. The lymph glands are invariably enlarged. This form of eczema is due to infection with pyogenic organisms. The children constantly re-infect themselves, and in this way the disease may be prolonged indefinitely. It is possible, too, that infection may spread to other children.

Intertrigo.—This term is rather indiscriminately applied to any eruption which develops upon two moist surfaces, which are in contact. It is often regarded as a form of eczema. There may be a simple erythema or an eczema resulting from traumatism or the decomposition of secretions. Intertrigo is seen in the folds of the groin, between the scrotum and the thighs, between the buttocks, about the anus, in the axillae, in the neck, or behind the ears. Its essential causes are moisture, friction, want of cleanliness, and sometimes infection. The disease is generally seen in its worst form about the thighs, genitals, and buttocks; it sometimes covers the sacrum and extends down to the middle of the thighs. There is an intense uniform redness, and in some cases the epidermis is denuded over large areas, and the surface is moist. There is no thick crusting and little or no itching. Intertrigo is usually easy to control except in very poorly nourished or marantic children, among whom it is especially frequent.

Diagnosis of Eczema.—This is usually quite an easy matter. In the majority of cases, the disease affects the face or the scalp, and its appearances are typical. Eczema of the body or extremities may be confounded with scabies or syphilis, and occasionally with other forms of skin disease. Scabies resembles eczema in its intense itching and multiform lesions; but in the former, one may often find evidences of its presence in other members of the family; the parts most frequently affected are

the flexures of the wrists, the elbows, the skin between the fingers, the margins of the axillae, the lower part of the abdomen and back, and, in boys, the penis; and by careful examination with a lens some of the characteristic burrows are certain to be discovered.

Syphilis is likely to be confounded with papular eczema of the buttocks. The latter affects the parts near the anus, and the irritation may lead to the development of spots closely resembling mucous patches. The local appearances may at times be indistinguishable from syphilis, and the diagnosis is to be made only by the other symptoms present. In syphilis the characteristic eruption is seen usually upon the face, hands, legs, and sometimes the palms and soles; there is no itching and very little evidence of inflammation; the eruption is copper-colored, and occurs as small circumscribed spots; there are usually present other symptoms, such as the coryza, the syphilitic cachexia, and enlargement of the spleen.

The diagnosis from pediculosis and ring-worm of the scalp, rarely presents any difficulties.

Prognosis.—All cases of chronic eczema are tedious. There is only a slight tendency to spontaneous improvement, and very little to spontaneous recovery during early infancy. About the end of the first year the disease disappears in many children; some relapse after this time, but others are never again troubled with eczema. In a severe case of general eczema the possibility of the development of severe toxic symptoms should not be forgotten. In any given case of eczema, the prognosis depends upon the duration of the disease, its severity, and very much upon the coöperation of the mother or nurse. The results obtained depend not only upon the particular line of treatment adopted, but upon how well it is carried out. Usually it must be continued for several months. Intertrigo is in most cases easily cured, unless the patient is suffering from extreme malnutrition.

Treatment.—A judicious combination of general and local measures is necessary for the best results. Unless disturbances of nutrition can be removed, local treatment will give only temporary relief. External causes also must be investigated.

A thorough investigation into the food is necessary, not only as to its character, but as to quantity and preparation, the manner and frequency of feeding, etc. If the patient is a nursing infant, very fat and well nourished, the amount of food should be reduced by lengthening the interval between feedings and shortening the time which the child is allowed to remain at the breast at one nursing. Plain water, or better, some alkaline water, should be given freely between the nursings. In children fed upon cow's milk the quantity may be too great, or the trouble may be with the sugar, but more frequently with the fat. This

should first be reduced and if no improvement occurs the sugar should also be diminished.

During the latter part of the first and the entire second year, the usual error is that of overfeeding, with in some cases an excessive use of solid food, very often with too much milk. The diet should then be much reduced, and the amount of solid food restricted. The diet which suits most children best is one composed of a moderate amount of milk, beef juice, broth, cooked fruit and green vegetables; eggs and meat must be used with caution. The cereals—rice, wheat or barley—may be added, in small amounts at first. Any form of indigestion which exists is to be managed according to the special indications in each case. When there is a susceptibility to proteins, as shown by cutaneous tests, a reduction or for the time a complete removal from the diet of the protein causing the reaction should be made with children over one year old. In older patients the results are sometimes very striking.

The diet of older children needs to be watched no less closely than that of infants. The general rules laid down elsewhere for feeding after the second year should be observed.

Elimination by the kidneys should be stimulated by the very free use of water, to which may be added an alkaline diuretic—the citrate or acetate of potassium, from ten to twenty grains daily.

Attention to the condition of the bowels is of the greatest importance. To overcome the tendency to constipation is in many cases to cure the eczema. Suggestions under this head will be found in the chapter on Chronic Constipation. The bowels must not only be opened, they must be kept open by the daily use, if necessary, of some of the milder laxatives, such as magnesia, phosphate of sodium, rhubarb, or cascara.

When the disease occurs in flabby, anemic, or poorly-nourished children, iron, arsenic and bitter tonics are required, but rarely cod-liver oil. In other words, the child's general condition should be treated just as if no eczema existed.

The general management of cases is important. The skin must be carefully protected by an ointment whenever the child is in the open air; if the weather is very cold, or there are high winds, children with active eczema should not go out, but be aired indoors. Never should an eczematous surface be washed with plain water, and much less with castile soap and water. When washing is necessary, it may be done with bran water, milk and water, or starch and water, to which borax (a teaspoonful to the quart) may be added. The clothing should not be so excessive as to keep the child constantly in a perspiration. Napkins should not be washed in strong soda solutions, nor, in case of eczema of the buttocks, should they ever be used a second time after being simply dried.

In eczema of the face it is absolutely necessary to prevent the child from scratching the parts. The use of a mask is not always sufficient, nor the wearing of mittens; nor is the local application of anti-pruritic lotions or ointments altogether successful. In severe cases mechanical restraint is absolutely indispensable. The most satisfactory method is to surround the arms at the elbows by pasteboard splints, and hold them in place by bandages. This allows free use of the hands, but makes it impossible for the child to reach the face.

Local Treatment.—Local treatment is always necessary, for not only are the causes sometimes entirely external, but the condition may persist after the original internal cause has been removed. There are several indications to be met by local treatment at different stages in the disease: (1) To remove crusts and other inflammatory products; (2) to allay congestion and acute inflammation; (3) to relieve itching; (4) to protect the delicate new skin which is forming; (5) to prevent infection; (6) to stimulate the skin in the chronic stages of the disease.

Preparatory to the use of any application, the scales, crusts, and other products of inflammation must be softened and removed in order that the diseased surface may be reached. In most cases it is sufficient to soften the crusts by the use of olive oil for twelve or twenty-four hours, and then remove them by soap and warm water. If the crusts are very hard and thick, they can be softened by a poultice. During the stage of acute inflammation only sedative applications should be used, such as a lotion of zinc and calamin.[1] A piece of muslin should be dipped in the solution, and applied to the affected part, being kept in place by a bandage or the skin may be frequently wetted with the lotion which is allowed to dry on. If there is much itching, one per cent of carbolic acid may be added.

Another plan of treatment, where there is much secretion, is to keep the surface covered with equal parts of boric acid and starch or talcum powder. An application which is often successful in allaying the intense burning and itching is black wash. This is applied several times a day in full strength or diluted and allowed to dry on, after which a protective ointment is used.

A soothing application in general eczema is one composed of equal parts of lime-water and sweet-almond oil; sometimes this may be advantageously followed by smearing the body with a thick starch paste and allowing it to dry on.

---

[1] ℞ Pulv. calaminae preparatae..... ℥ij
Zinci oxidi ..... ℥ss.
Glycerinae ..... ℥i
Liquor calcis ..... ℥ij
Aquae rosae ..... ℥viij

As a simple protective ointment, one containing starch, zinc oxid, or bismuth, either alone or in combination, may be used. An excellent formula is zinc oxid ointment with two per cent of salicylic acid.

Later, when the inflammation is less acute and the itching severe, tar in the strength of ten to twenty per cent may be substituted for the salicylic acid.

All ointments used should be spread upon muslin, and kept in close contact with the inflamed part by means of a bandage or mask. Little or nothing is accomplished by simply rubbing the ointment upon the affected part. An ointment containing five or ten per cent of calomel is often the best application for an eczema which is not too extensive.

The methods of treatment above mentioned are especially applicable to eczema of the face and scalp. For pustular eczema of the scalp the best application is the white precipitate ointment, which should be combined with three or four parts of vaseline. This is excellent also for small eczematous patches upon the body, but it is not to be used over a large surface.

In intertrigo, the treatment should have reference to the pathological condition which is present. Cases of simple erythema usually yield promptly to cleanliness and the free use of absorbent antiseptic powders, such as boric acid and starch in equal parts, or calomel two per cent may be used with talcum. If there is an acute dermatitis, the calamin and zinc lotion may be used, and later some protecting ointment. When infection has been added, lotions of resorcin or ichthyol, one-half of one per cent strength, should first be applied, and the skin then covered with one of the powders mentioned; both are to be repeated as often as the parts are wet by urine or soiled by feces. It is important in all cases that the diseased surfaces should be kept separated, which is best done by boric acid and starch. All napkins should be immediately removed when soiled.

In cases of chronic eczema, where the skin remains thickened, red, scaly, and itching, stimulating applications are to be used, such as the tincture of green soap or stronger preparations of tar.

FURUNCULOSIS

A furuncle, or boil, is a circumscribed inflammation of the subcutaneous cellular tissue, usually beginning in a hair follicle, and usually ending in suppuration. When severe, it may result in necrosis of the follicle, which forms the "core," or the necrotic process may extend to the surrounding tissues for a variable distance. The ordinary boil need not be described, as it presents nothing peculiar in early life. The con-

dition, however, which is characteristic of young children is the formation of small ones in great numbers. It is to this more especially that the term furunculosis is applied. The principal location of these small abscesses is, in nearly all cases, the scalp, face, and shoulders, although they may be found upon any part of the body. They are sometimes numbered by hundreds, and appear in crops for a period of several months. In size, they usually vary from a pea to an almond, and they rarely contain a core. Infants are much more often the subjects of this disease than are those who have passed the second year. In the great majority of cases furunculosis is not serious, yet, occurring, as it often does, in infants who are already suffering from extreme malnutrition, whose tissues possess but little resistance, the process may develop into a condition which may prove fatal.

Furunculosis may be seen in children who are in other respects apparently healthy, even robust; but the majority are in a more or less debilitated condition, and often are the subjects of digestive disturbances. The disease is quite frequent in syphilitic infants; but these simple abscesses are to be sharply distinguished from those which result from the breaking down of gummata of the skin. Want of cleanliness of the skin is a factor of some importance in producing the disease. Furunculosis may be associated with eczema. The exciting cause in all cases, as shown by recent investigations, is the entrance of the staphylococcus pyogenes aureus, sometimes with other organisms, into the follicles of the skin.

Treatment.—The general treatment is to be directed toward any disturbance of digestion or nutrition which is present. Tonics are indicated in most cases, but no reliance can be placed upon drugs such as sulphid of calcium or the hypophosphites, in arresting the disease. Local treatment should have for its first object thorough cleanliness of the skin. This is best secured by frequently bathing the parts affected with a 1 to 5,000 solution of bichlorid. Single furuncles may often be aborted by touching them with pure carbolic acid or the application of Bier's cups. In our experience the best plan of treating the multiple small furuncles, is to delay incision until they have pointed, then to incise and empty the follicle completely by compression. Where the abscesses are of large size and upon the scalp, it is wise to make compression by applying a snug bandage for a day. For general furunculosis or the continual recurrence of larger abscesses the use of staphylococcus vaccines is altogether the most effective treatment. While autogenous vaccines are perhaps preferable, the use of stock vaccines seems in most cases to be equally effective. Injections should be repeated every four or five days; beginning with fifty millions, the dose may be increased to one hundred millions, or even more. The beneficial effects in most cases are very striking and the cure permanent.

GANGRENOUS DERMATITIS

This is not a frequent disease, and is seen almost exclusively in infancy. It may be primary or it may follow other diseases, and hence has been described under many different names, viz., *varicella gangrenosa, ecthyma, pemphigus gangrenosa,* etc.

The lesion consists in small, discrete areas of inflammation of the skin, ending in necrosis. In the primary cases there is usually first seen a vesicle, about as large as a pea, with a dusky areola; it increases in size and becomes a pustule. Crusts form which are quite adherent, and on removing them a loss of tissue is seen. The ulcers usually have sharp but not undermined edges, often presenting a "punched-out" appearance. By the coalescence of several smaller ones, ulcers an inch or more in diameter are sometimes formed.

The primary form of gangrenous dermatitis occurs in wretched, poorly-nourished infants, and is most often seen upon the buttocks. In this location it may be mistaken for syphilis. The secondary form is more common, and usually follows varicella, less frequently vaccinia, or impetigo. In such cases the lesion is most often seen upon the upper half of the body, especially upon the neck and chest. It follows the ordinary lesions of varicella and continues usually, in spite of treatment, from one to four weeks, in many cases ending fatally. The disease always occurs in infants of poor vitality, often in those suffering from marasmus, and is seldom seen outside of institutions. It may be accompanied by fever, and other severe constitutional symptoms.

For the production of the disease, two factors are necessary: first, the constitutional condition referred to; and, secondly, the entrance of pyogenic germs, usually the streptococcus pyogenes.

Treatment.—Every means possible should be employed to build up the general health of the infant by fresh air, careful feeding, etc. Locally, strict cleanliness and antiseptic applications are necessary. The best application is a solution of bichlorid (1 to 5,000), or an ointment of ichthyol or white precipitate.

IMPETIGO CONTAGIOSA

Impetigo contagiosa is a disease characterized by the formation of discrete vesiculopustules, occurring most frequently upon the hands and face. Cases are usually seen in groups affecting children in one family or institution. Impetigo may be communicated from one person to another, and spread by auto-inoculation from one part of the body to another.

One rarely has an opportunity to see the disease until vesicles have formed. These are usually from one-fourth to one-half inch in diameter, and are flaccid, never distended. Later, their contents become slightly yellowish; then they rupture and dry, forming thick yellow crusts, which have the appearance of being "stuck on," the surrounding skin being quite healthy. After the crusts fall off, a small red patch remains, which slowly fades. The true skin is not involved, except in poorly nourished, cachectic subjects, as a result of continued local irritation, like scratching. Under such conditions ulceration may occur. Instead of the small vesiculopustules described, bullae from one to two inches in diameter may form, filled first with serum, afterward with sero-pus. Very little inflammation is seen about these patches, and in most cases the intervening skin is normal.

The favorite seat of the eruption is the face, especially about the chin, next the hands, the neck, the feet and legs, the forearms, and the scalp; it is rarely seen upon the abdomen, and never upon the back. There may be only half a dozen vesiculopustules, or from thirty to forty may be present. The smaller ones sometimes coalesce and form others of considerable size. Itching is never a prominent symptom, and in most cases it is absent altogether.

The usual duration of impetigo contagiosa is two or three weeks; it, however, runs no regular course, and by continued auto-inoculation may last much longer than this.

The studies of Gilchrist point to a streptococcus of low virulence as the cause of this disease. European investigators, however, have more often found the staphylococcus pyogenes aureus in the vesicles. Impetigo contagiosa may occur in any child, but is seen most frequently in one who is poorly nourished.

The diagnosis is not often difficult, and is made by the following features, viz., the occurrence of several cases together, the isolated vesiculopustules situated upon the face and hands, the slight itching, and the prompt cure by local measures only. The bullous form, however, is frequently confounded with pemphigus; many cases in which the diagnosis of pemphigus is made are examples of impetigo.

Treatment.—This is simple and usually very effective. The crusts are to be softened and removed by thoroughly washing the part with soap and water or a bichlorid solution, after which the white precipitate ointment, combined with three parts of vaseline, should be applied.

URTICARIA

Urticaria is a frequent disease in early life, and presents some features, particularly in infants and young children, which are quite dif-

ferent from those seen in adults. This is due to the fact that papules and vesicles, and occasionally pustules, are associated with the wheals. As the wheals quickly subside, it frequently happens that the other lesions mentioned are the only ones present. This fact has given rise to considerable confusion in names, and the urticaria of infancy has been called *lichen urticatus, urticaria papulosa, strophulus,* etc. It is now pretty generally agreed that the clinical picture, which is a familiar one, belongs to a single disease, and that this is urticaria.

The initial lesion is the wheal, but on account of the extreme susceptibility of the skin in young children, the process is more intense than in older patients, so that it may result in the formation of an inflammatory papule or a vesicle. In a few hours the wheal may subside, and only the papules or vesicles remain, and without a good history the disease may be a very obscure one. The papules and vesicles occur with greatest frequency upon the hands and feet, particularly the palms and soles.

The more severe form of the disease in poorly nourished children is sometimes accompanied by a pustular eruption, and there may even be deep ulceration (ecthyma). The usual appearance of the eruption is a number of small inflamed red papules whose tops are covered with crusts, the result of scratching. The eruption may be limited to the extremities or it may be general. It is as a rule more severe in regions accessible to scratching.

There is usually severe itching, which leads to loss of sleep, and often in this way the disease affects the general health of the child. The urticaria of older children does not differ essentially from the same disease in adults. The alternation of urticaria and asthma is occasionally met with.

The character of the eruption in urticaria and even its distribution often suggest scabies; and unless one has had an opportunity to witness the development of the lesions, a differential diagnosis may be very difficult, as almost every lesion, except the wheal, may be identical in both diseases. Other cases may resemble varicella.

Urticaria in early life is most frequently the result of some disturbance in the digestive tract. Almost any sort of derangement may produce it, the exciting cause varying with the patient.

Treatment.—The milder forms of urticaria usually respond quickly to treatment; but when it is severe and has existed for several weeks, it is one of the most troublesome and intractable skin diseases of childhood. The treatment is to be directed primarily toward the condition of the digestive organs. Children should be put upon a very simple diet, always excluding sweets, and usually fruits, especially raw fruits. The bowels should be kept open by calomel, a nightly dose of castor oil, or a

morning dose of magnesia. If the urine is excessively acid and scanty, alkaline diuretics should be given.

All local causes of irritation, such as rough flannel underclothing, should be removed. The sleep may be so much disturbed as to require the use of trional or bromid and chloral.

The local irritation and itching may be relieved by a very dilute solution of the subacetate of lead or carbolic acid, or by diluted vinegar, or the fluid extract of hamamelis, or bicarbonate of soda, and water. In severe urticaria almost immediate relief may be obtained by the hypodermic injection of three to eight drops of a 1-1000 solution of epinephrin: the relief often lasts twelve to twenty-four hours. When pustules are present, the white precipitate ointment may be used, combined with four parts of vaseline; in the papular and vesicular forms, an ointment of ichthyol, one-per-cent strength. In many cases the improvement in the general health by the use of tonics, change of air, etc., will accomplish more than any measures directed especially to the relief of the urticaria.

SCABIES

Scabies is a contagious disease due to the burrowing into the skin of the female acarus, with secondary lesions which result from scratching.

The burrowing of the acarus is usually where the skin is thinnest—viz., between the fingers, on the flexor surfaces of the wrist, the axillae, and, in males, the genitals. It is not seen upon the face, except in infancy, when infection may occur from contact with the breasts of the mother. The lesion excited by the acarus is usually a papule or a vesicle, sometimes a pustule. In some cases no evidences of inflammation are present, but in infants and young children they may be marked—pustular eruptions being frequent and often extensive, especially upon the hands and feet. The characteristic burrow is from one-fourth to one-half inch in length, and appears as a fine brown or black line, at the end of which the acarus may be discovered as a small white speck. The burrows are often difficult to find in infants. They are generally to be seen along the ulnar border of the hand and between the fingers. The intensity of the inflammatory lesions varies greatly in different cases; in some they are very few, while in others, particularly in delicate, cachectic, and neglected children, they are sometimes very severe, so that the skin of the affected part is nearly covered with pustules. These secondary lesions are due to infection by the streptococcus or staphylococcus. A pustular eruption upon the hands should always suggest scabies. The lesions which result from scratching may be found on any accessible portion of the body. They are usually at first linear, bloody marks, but after

a time these may not be visible. In little children urticaria is often associated.

The diagnosis of scabies is usually quite easy, as several children in a family are likely to be affected, particularly if they occupy the same bed. The diagnostic features of the eruption are the presence of papules, vesicles, or pustules, especially upon the hands, wrists, and genitals. A careful examination with a lens will usually disclose some of the characteristic burrows, or even the acarus. In infancy, scabies may be easily confounded with the vesicular form of urticaria, unless the development of the lesions has been observed.

Scabies may always be cured, provided sufficient precautions are taken to prevent re-infection. This necessitates boiling or baking, not only the patient's clothes, but all the bedding as well.

Treatment.—This should always be begun by a hot bath, in order to soften the epithelial scales about the burrows. The body should be thoroughly scrubbed with soap and water, preferably with a nail-brush, the bath being continued for at least half an hour. It is well to do this at night. After the bath, the body is anointed with the parasiticide, which should be thoroughly rubbed into the skin, clean clothing applied, and the child put into a perfectly clean bed. In the morning the ointment may be washed off, but none of the clothing previously worn should be put on. This treatment is to be repeated on two or three successive nights, and if thoroughly done it will effect a cure. The ordinary sulphur ointment is too irritating for use in little children, and one of the following may be substituted: β-naphthol, 15 parts; creta preparata, 10 parts; vaseline, 100 parts (Kaposi); or, precipitated sulphur, 1 part; balsam of Peru, 1 part; vaseline, 8 parts; or the simple balsam of Peru may be applied without dilution. After the use of the parasiticide there is generally required, for a few days, some soothing application like those mentioned in the chapter upon Eczema.

TINEA TONSURANS—RING-WORM OF THE SCALP

Ring-worm of the scalp is a very frequent disease in institutions for children, often occurring as an epidemic. According to Crocker, the primary lesion consists in a red papule surrounding a hair, which soon increases to a small circular patch; this spreads at its outer margin, gradually increasing in size until it is from one to two inches in diameter, but rarely larger than this. Sometimes several of the patches coalesce. These affected areas always have rounded borders, and are sharply outlined. Here the hairs are very brittle, and often broken off close to the scalp, so that the area may appear to be bald. Where they have not

fallen off, the hairs have lost their luster. The stumps of the broken hairs point in all directions.

The fungi which produce the disease belong chiefly to the group of small spored fungi or microsporons. Of the several microsporons that have been shown to have etiological significance, the microsporon Audouini is the one of importance in this country. The large-spored fungi (tricophyton crateriforme or tricophyton acuminatum) are responsible for a small proportion of cases. The fungi penetrate the shaft of the hair, both the spores and the mycelium being seen under the microscope. The spores are present in great numbers in the hair, but the mycelium is most abundant in the scales. The amount of inflammation found in the diseased areas varies much in the different cases. There may be only a scaliness of the scalp, or a formation of pustules in the hair follicles, the hairs loosening and falling out in consequence. In young infants, where the hair is scanty and thin, the disease resembles tinea circinata—i. e., it is superficial, and the hair follicles are often not involved. Children of all ages are liable to tinea tonsurans. It flourishes particularly in institutions and among those children who are dirty and generally neglected.

The diagnostic feature of the disease is the presence of scaly patches, with loss of hair. The patches are usually circular, and by examination with a lens the stumps of broken hairs are seen all over the diseased areas. By a microscopical examination the fungus is discovered. In typical cases the diagnosis is easy if the process is at all advanced, but there are many atypical forms and many mild cases where the recognition of the disease is difficult. The symptoms are often masked by the inflammatory conditions present. The disease may be confounded with seborrhea; but in the latter the lesion is diffuse, never sharply defined; there is general thinning of the hair over the scalp, and never the stumpy, broken hairs. Psoriasis has points of resemblance; but it is usually found on other parts of the body, especially the knees and elbows, and upon the scalp the patches are more numerous and smaller. In eczema the loss of hair in circumscribed patches is never seen, nor are the broken stumps.

Tinea tonsurans is always curable, provided the patient can be kept under close surveillance, and treatment thoroughly carried out, but it is particularly obstinate. There is no tendency to spontaneous recovery except toward puberty, when many of the cases recover even without treatment. In a recent case, treatment must usually be continued for several weeks or months, and in chronic cases from six months to one year, with the closest watchfulness.

Treatment.—The great difficulty in treatment is to get the parasiticide deeply enough into the scalp to reach the fungus, since this is often

at the very bottom of the hair follicles. As a first step, the hair should be cut short all over the patch and for at least an inch beyond it; this is necessary in order to get at the diseased part and to detect new foci of infection early—if possible before the fungus has extended deeply into the follicles. The parasiticide should be applied not only upon but around the patch, and the entire scalp should be washed thoroughly two or three times a week. To prevent the disease spreading, all the scales are to be kept softened by the use of carbolic soap. The hair should not be brushed, as this tends to scatter the spores and spread the disease. All patients, while under treatment, should wear a cap of muslin or oiled silk, or one lined with paper, in order to prevent infecting others. In institutions, affected children should invariably be isolated.

To destroy the fungus almost every germicide on the list has been advocated at one time or another, which proves that the disease is a very obstinate one, and that no one application is invariably successful. Cure depends more upon persistent treatment than upon the drugs used. Those which have the sanction of the widest use are the tincture of iodin, the bichlorid, white precipitate and oleate of mercury, β-naphthol, chrysarobin, creosote, carbolic acid and croton oil. As a vehicle for ointments, adeps lanae (lanoline) is greatly to be preferred to vaseline or lard. Epilation is necessary in many cases as an accessory to the application of germicides, particularly in older children. The X-ray has been employed by Sabouraud, Noiré and others. The greatest care should be exercised in its use or permanent baldness may result.

CHAPTER VI

DISEASES OF THE EAR

ACUTE OTITIS

Otitis is a frequent affection during infancy and early childhood, attacks usually occurring in the cold season. Of all the inflammatory conditions which may be met with in early life, there is perhaps none which more frequently gives rise to obscure febrile symptoms than this.

Etiology.—Acute otitis is, as a rule, a secondary disease, and is generally preceded by some infectious process in the rhinopharynx. The usual avenue of infection is the Eustachian tube.

While it is most commonly seen following simple rhinopharyngitis, the most severe forms of otitis follow scarlet fever, epidemic influenza, measles, diphtheria, or pneumonia. The entrance of fluids through the

Eustachian tube from the nasal douche or nasal syringing may cause acute otitis. It sometimes results as an extension of inflammation from meningitis, especially the cerebrospinal form. Otitis is very common in hospital patients, especially poorly nourished infants. In them it is found with little or, more frequently, with no evidences of a rhinopharyngitis.

The microörganisms concerned in the production of acute otitis vary somewhat with the condition of which it is a complication. In the order of frequency there are found the staphylococcus aureus, the pneumococcus, the streptococcus, and the influenza bacillus. Mixed infections are very common. In cases complicating diphtheria, the Klebs-Loeffler bacillus may be found with any of the forms mentioned, or may occur alone. In chronic cases any of the pyogenic organisms may be present, and not very infrequently the tubercle bacillus.

Lesions.—The ordinary course of events in the pathological process is, first, acute hyperemia and swelling of the mucous membrane of the rhinopharynx, which extends into the Eustachian tube, causing obstruction more or less complete. The inflammatory process may be limited to the tube, or it may extend to the mucous membrane lining the middle ear.

There are two varieties of acute inflammation of the middle ear: (1) The catarrhal form, which usually accompanies simple catarrh of the rhinopharynx or complicates measles. This is an inflammation of the mucous membrane merely, and its products are serum and mucus or muco-pus. It is generally confined to the lower part of the tympanic cavity, and is the form most frequently seen in infants. (2) The purulent or phlegmonous form, which affects older children principally. This is a much more serious inflammation, and is often excited by the infectious catarrh of scarlet fever, or diphtheria. In this variety microörganisms find their way into the middle ear in great numbers, and set up an inflammation of a more virulent type, which may involve not only the mucous membrane lining the tympanum, but also the cellular tissue in the upper part of the tympanic cavity. The lining membrane of the mastoid cells is involved in many, if not all, of the cases.

The catarrhal form of inflammation frequently subsides in a few days with proper treatment, the only result being a slight deafness, which is temporary. The phlegmonous form causes a stoppage of the Eustachian tube, rupture or sloughing of the tympanic membrane, and discharge of the products of inflammation, or rarely pus finds an outlet by burrowing between the cartilages. The inflammatory process may extend to the bones, causing necrosis of the ossicles or the bony walls of the tympanum. The remote results are periostitis and necrosis of the petrous bone, pachymeningitis, infectious thrombosis of the lateral sinus,

general purulent meningitis, and cerebral abscess. These will be considered under Complications.

Symptoms.—These are usually few in number, but present great variability as regards their combination and intensity. The two most constant symptoms are pain and fever. In a typical case in an infant, there is generally at the beginning some discharge from the nose, slight congestion of the pharynx and tonsils, and a temperature of 100° to 102° F. There is nothing characteristic about this catarrh. After two or three days the objective symptoms subside, but the infant continues to be restless, worries much of the time, wakes frequently at night with a start, nurses poorly, and the temperature remains elevated, usually from 100° to 103° F. (Fig. 150). The infant seems decidedly ill, and

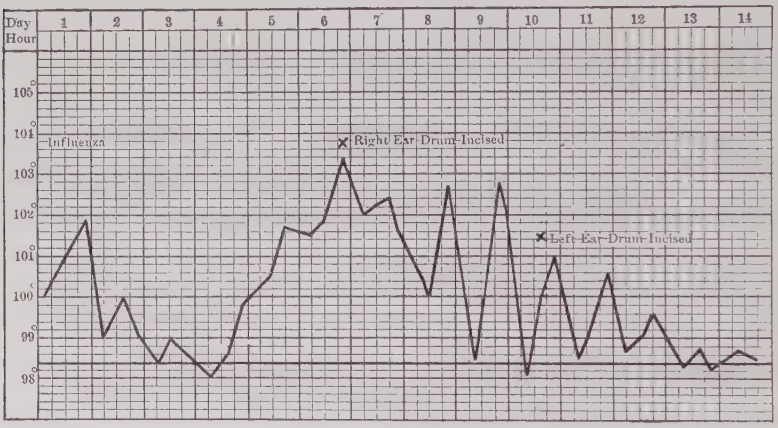


Fig. 150.—Temperature Chart of Acute Otitis Following Influenza, in a Child Three Years Old.

yet no very definite symptoms are present. Rarely there is marked tenderness about the ear, and the child refuses to lie upon the affected side, or shows signs of pain when the ear is touched. After a week or ten days spontaneous rupture of the drum membrane takes place, and subsidence of the constitutional symptoms follows. In some cases there is seen only a high temperature, ranging from 101° to 104° F., which persists for several days without outward evidences of pain or other signs of inflammation, and the discharge is the first symptom which leads the physician to suspect disease of the ear. In other cases there is marked dulness, apathy, anorexia, and sometimes nausea and vomiting, but for several days no evidence of pain; the temperature may be but little elevated. Thus, in most of the attacks seen in infancy, pain is not marked, and it is this fact which so often leads to the obscurity of the symptoms.

In older children the symptoms are more characteristic. Pain is

usually sharp and severe, and is complained of early in the attack. The temperature is nearly always elevated two or three degrees, and occasionally it is 103° or 104° F. (Fig. 151), with severe headache, extreme restlessness, and even delirium or convulsions, so that meningitis may be suspected.

The inflammation does not necessarily go on to suppuration and rupture. There are even more frequently seen, accompanying ordinary head-colds or mild attacks of influenza, cases in which the pain is quite severe for twenty-four or thirty-six hours, and accompanied even by a moderate elevation of temperature, and yet which rapidly subside without further symptoms.

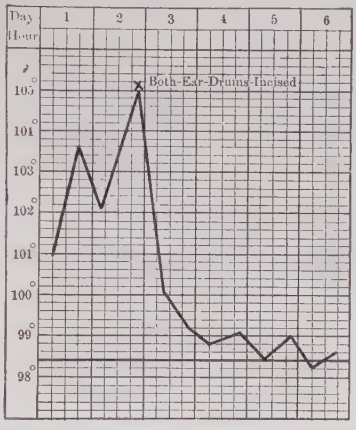


Fig. 151.—Temperature Chart of Acute Otitis Aborted by Early Paracentesis. Boy nine years old; attack followed a mild catarrh; severe pain in both ears began in afternoon of second day. Both drum membranes found acutely congested and bulging; incision followed by free hemorrhage and immediate relief of pain. No suppuration occurred; patient well on fifth day.

In infants suffering from malnutrition or marasmus, otitis often comes on without any objective symptoms, the first thing noticed being the discharge.

Of all the symptoms, fever is the most constant, and is present in all cases except those just mentioned. The usual range of temperature is from 100° to 102° F.; exceptionally it may be from 103° to 105° F. The course of the temperature is irregular. After spontaneous rupture or incision of the drum membrane the temperature usually falls, but often not immediately. Pain is more marked in older children than in infants, because in the latter the drum membrane is not so firm, yields more readily, and ruptures earlier. Tenderness is sometimes elicited by pressure, especially just in front of the external auditory meatus; there may be increased sensitiveness of all parts of the ear and even of the whole side of the head; but no reliance should be placed upon the absence of such symptoms in excluding otitis. Children often complain of noises in the ear. Cerebral symptoms are infrequent, and occur chiefly in cases not receiving proper early treatment; they may indicate meningeal congestion, or, less frequently, localized meningitis or thrombosis.

In secondary otitis, especially when complicating severe scarlet fever, diphtheria, measles, or typhoid fever, all subjective symptoms are frequently wanting; unless the ears are examined the disease may be overlooked until rupture has taken place.

The local appearances in the early stage are marked redness and congestion; later there is distinct bulging. If perforation has taken place, its site may or may not be visible, but its existence may be assumed if bubbles of air are seen deep in the canal, and if, in the absence of a furuncle or marked eczema, much mucus or pus is present, as inflammation of the external canal seldom causes a discharge. In the catarrhal form the discharge is at first sero-mucus and quite profuse; later it is purulent. In the phlegmonous form it is always purulent, and liable to a sudden arrest with an increase in the constitutional symptoms. The pus sometimes burrows between the cartilages and escapes externally behind or at the side of the ear. Rarely it may work its way anteriorly and cause an abscess in the parotid gland.

Diagnosis.—Otitis in infancy is frequently obscure, because the patient is too young to direct attention to the seat of pain, or because the pain is slight or absent. The temperature is almost invariably elevated, and the usual problem presented is to discover a cause for this fever. The examination of the ears with a speculum should be made as a matter of routine in all children with fever, especially those in whom the cause of the fever is not perfectly clear. Otherwise many cases will be overlooked. A leucocytosis of 15,000 to 20,000 is almost invariably found. Local tenderness, deafness, or noises in the ears are significant when present, but are often wanting. Otitis is so common a cause of high temperature in infants during the cold season, that one should always have it in mind.

Complications and Sequelae.—Remote consequences are most likely to be seen in cases following scarlet fever, probably because of their severity, particularly when early treatment has been neglected.

Mastoiditis.—This is the most frequent complication of acute otitis. In infancy the mastoid process is small and contains but a single cavity, the mastoid antrum, which communicates directly with the vault of the tympanum. It is probable that in every severe case of acute suppurative otitis there is some pus in the antrum. This is usually discharged into the middle ear after the tympanic membrane is incised or ruptures spontaneously. The principal cause of mastoid involvement is want of proper early treatment in acute otitis, particularly the practice of allowing these cases to take their natural course instead of securing early drainage by incision of the drum membrane.

The important symptoms of acute mastoiditis are fever, mastoid tenderness, and swelling. If mastoiditis develops rapidly after acute otitis the temperature may be high—103° to 105° F., and the leucocytosis is somewhat greater; if it develops gradually and appears late the temperature may be scarcely above 100° F. Abrupt cessation of an ear discharge should always arouse suspicion. It is always difficult to de-

termine the presence of a slight amount of mastoid tenderness, but persistent tenderness of one side only is significant. It is often most marked close behind the auricle just over the antrum. Care should be observed in ascertaining tenderness to make pressure only over the mastoid. When there is eczema or furunculosis of the canal pushing forward the auricle causes pain. The early swelling is due to edema from periostitis; later there may be an accumulation of pus beneath the periosteum. Post-auricular abscess causes a very characteristic swelling, the ear standing out from the head. It is usually due to spontaneous rupture through the outer bony wall just over the antrum; it may occur when there has been no discharge from the ear; but mastoiditis practically never occurs as a primary hematogenous infection and examination of the drum membrane will reveal unmistakable evidences of an otitis media. It is a frequent result of severe cases of acute mastoiditis not operated upon, especially in young children.

The characteristic otoscopic appearances of acute mastoiditis are, bulging of Shrapnell's membrane and drooping of the upper posterior wall of the external auditory canal due to edema.

Meningitis.—This is very rare in infants, but is more common in older children. There may be a localized pachymeningitis with the formation of pus—an epidural abscess—or, less frequently, general purulent meningitis. It may be secondary to other lesions, such as thrombosis of the lateral sinus, or the rupture of a cerebral abscess, but is usually due to infection through the roof of the tympanum, or along the internal auditory meatus. Meningitis may occur either with acute or chronic cases. Its symptoms are those of a severe acute meningitis; its duration is short; its termination almost invariably in death.

Cerebral Abscess.—This is due to a direct extension of the infection from the bone, veins, or dura mater. In about two-thirds of the cases the abscess is in the temporosphenoidal lobe. The next most frequent seat is the lateral lobe of the cerebellum. Körner states that disease of the mastoid and middle ear leads to cerebral abscess, and disease of the labyrinth to cerebellar abscess. Abscesses may be complicated by thrombosis or by meningitis. They are often latent until just before death, which more frequently occurs from the development of purulent meningitis than from any other cause. They are rare except in otitis of long standing. (See Cerebral Abscess.)

Thrombosis of the lateral sinus may be simple or septic. In the former there is occlusion of the vessel by a fibrinous clot; in the latter there are in addition, microörganisms.

Simple thrombosis causes no important symptoms. Septic thrombosis is relatively infrequent and causes very marked and severe symptoms. It follows operation upon the mastoid, or occurs as a complication

of mastoiditis quite apart from operation. The temperature is usually of a high and widely fluctuating type, and there may also be chills with older children, but this cannot be depended on as evidence of thrombosis in infants or young children. In some cases the constitutional symptoms, except fever, may not at first be severe, but may suddenly become very grave. Marked cerebral symptoms often develop rapidly, and death may follow in from twelve to twenty-four hours. At autopsy there may be found a soft broken-down clot in the sinus, which may extend into the jugular. It may be followed by secondary lesions of a general pyemia, or by localized or general meningitis. Blood cultures usually give positive information, but it is often necessary to make several before organisms are found.

The labyrinth is infrequently involved, although cases are recorded by Pye, Phillips, and others, in which the necrosis and discharge of the entire labyrinth has occurred after scarlet fever. In most of these cases the deafness was complete, and in several vertigo was present.

Facial paralysis rarely occurs in the acute cases, but accompanies a considerable proportion of the chronic ones. It is especially seen in the tuberculous variety. It is due to an extension of the inflammatory process from the bone to the seventh nerve, where it passes through the canal. The symptoms are those of ordinary peripheral facial palsy. The prognosis is good for recovery in the non-tuberculous variety.

Treatment.—Something may be done in the way of prophylaxis. It is of the first importance to secure a normal condition of the mucous membrane of the rhinopharynx by the removal of enlarged tonsils, adenoids, etc. The occasional attacks of otitis accompanying these conditions are pretty sure to be followed by more serious trouble unless they are relieved. Repeated attacks of otitis media in childhood are responsible for fully eighty per cent of the cases of chronic catarrhal deafness in adult life. Whether during attacks of measles or scarlet fever, much can be done to prevent otitis, is still a mooted question. We believe the risks of infection of the middle ear when judicious nasal syringing is employed are less than when nothing is done to cleanse the rhinopharynx.

The medical treatment of acute otitis aims at the relief of pain and arrest of the inflammation. If the case is seen in the early stage the introduction of a few drops of a solution of epinephrin into the nostrils and ears and repeated every two or three hours, will sometimes abort an attack. Carbolic acid in olive oil in a strength of ten per cent has an undoubted effect in allaying inflammation if applied in the early stages. This may be aided by free catharsis and the application of dry heat. Laudanum should not be dropped into the ear as is so often done in domestic practice; but there is no objection to a few drops of a four-per-cent solution of cocain, which may relieve intense pain. If the child

is not soon comfortable, an opiate should be given which may not only relieve pain, but may have a favorable influence upon the inflammation.

A continuance of pain in spite of these measures, with an increasing temperature, calls for operative interference. But a more reliable guide is the appearance of the drum membrane. If in addition to these symptoms there is mastoid tenderness immediate paracentesis of the drum membrane is imperative. An early incision is usually followed by a discharge of blood only; but tension is relieved, pain disappears, and the inflammation often quickly subsides without the formation of pus. (See Fig. 151.) Much suffering is thereby avoided; the wound rapidly heals, and much less damage is done than by allowing the disease to go on to a spontaneous rupture. Later incision may be required either for the relief of pain or for the evacuation of pus to prevent, if possible, the disease from spreading to the bony parts. The advantages of early paracentesis in acute otitis can hardly be overstated. Properly performed, it is free from risk, causes little or no shock, and should be advised in many cases even in which the indications are not so clear as those above described. Incision of the drum membrane should be favored in cases of doubt rather than waiting for more definite indications with the attendant risks of delay.

In the secondary otitis of scarlet fever, measles, and diphtheria, the indications for paracentesis are usually to be derived from the appearance of the drum membrane alone, other symptoms being absent or masked by the primary disease.

After incision or spontaneous rupture of the drum membrane, to prevent the wound from closing and to cleanse the parts, the ear should be syringed every two or three hours with a warm saline solution, or a saturated solution of boric acid. A bulb ear-syringe of soft rubber or a fountain syringe may be used. The external auditory canal should be carefully dried after irrigation to prevent maceration and the development of eczema.

In most acute cases the discharge ceases in from one to three weeks; should it continue longer, some measures for checking it may be used. The use of a few drops of a 1 to 3,000 solution of bichlorid in sixty-five per cent alcohol after syringing is of some value. It should be used with a medicine dropper. When the discharge has become fetid, syringing once a day with a solution of peroxid of hydrogen (1 to 2) is often useful. A persistent discharge often depends upon the fact that the child's general condition is poor, and improvement in this is more important than any variation in local treatment.

When symptoms pointing to acute mastoiditis are present, early free incision of the drum membrane is indicated, and a mastoid ice-bag should be applied intermittently for twenty-four to thirty-six hours.

In addition, in older children, the artificial leech may be placed over the antrum or the mastoid tip. With these measures the inflammation often subsides. Regarding operation upon the mastoid, our belief is that it is now performed too frequently and with insufficient indications, especially in infancy and very early childhood. The operation is a serious one, and at this age its immediate risks are considerable. We have known of a number of deaths directly connected with it, and of others occurring at a later period, where the child was worn out by the long after-treatment, dying perhaps from some intercurrent disease or from exhaustion. On the other hand, the dangers to which very young patients are exposed who are not operated upon have been exaggerated. In our experience, meningitis, sinus thrombosis, and cerebral abscess do not occur in anything like the proportion of cases that the surgeons would have us believe.[1]

While fully appreciating the value of the operation, and being quite sure that lives are often saved by its timely performance, we would insist that it be done only with very positive and clear indications. In infants, localized tenderness is difficult to determine; and fever after acute otitis may be due to many other conditions. In very young patients we should therefore insist upon other symptoms before deciding to operate. The risks of waiting for clearer indications are much less than those attendant on unnecessary operation. Often the cause of the temperature is found in the lungs; and not very infrequently a moderate pulmonary congestion or bronchitis becomes a pneumonia as a consequence of the prolonged anesthesia necessary for the operation. With infants therefore in case of any doubt, as to diagnosis or the progress

[1] The records of the New York Foundling Hospital, with a resident and constantly changing population of about 800 infants and young children, showed 573 cases of acute otitis in five years (1900 to 1904, inclusive). During this period there were three extensive epidemics of measles with a total of 1,034 cases; 166 cases of scarlet fever; 578 cases of diphtheria; and 1,505 cases of pneumonia. With the 573 cases of otitis, acute mastoiditis was recognized and recorded in but 17 patients. It is not improbable that other mastoid inflammations were overlooked. In this institution, however, nearly every fatal case comes to autopsy, and if an unrecognized mastoiditis had led to a fatal result the autopsy records should show it. In the five-year period, 900 autopsies were made. There was no instance recorded of abscess of the brain following otitis. There were but two examples of acute meningitis following otitis with mastoiditis; but there were 14 cases of acute meningitis secondary to other conditions—pneumonia, 10; to pericarditis, 2; to empyema, 1; to diphtheria, 1. During the period mentioned there were 11 mastoid operations performed in the hospital, with 6 recoveries and 5 deaths, all from causes directly connected with the operation.

If mastoiditis follows the otitis which complicates the acute infectious diseases of early childhood as often as has been claimed, we must admit that a very large proportion of the patients may get well without operation.

of the case, one should invariably decide against operation, or at least for postponement. With older children, however, conditions are somewhat different; diagnosis is easier and the operative risk much less.

The treatment of chronic otitis and of the associated conditions is largely surgical, and belongs to the specialist; but it is extremely important that the general practitioner should be familiar with their symptoms, and realize the danger from these neglected cases, not only to the function of hearing, but also to life itself. The essential thing in treatment is that the operation should be thorough enough to secure free drainage, and to permit thorough cleansing of the parts. Too much can not be said against the expectant treatment of these cases, or against the practice of prolonged poulticing.

SECTION IX

THE SPECIFIC INFECTIOUS DISEASES

A more accurate knowledge of the causative agents of the various infectious diseases has made necessary a revision of the opinions once held regarding the manner in which they are communicated. It was formerly believed that most of the common contagious diseases were air-borne infections. Smallpox and scarlet fever especially were cited as examples of diseases which could be conveyed by air currents at a considerable distance from the body. It was believed that these and other contagious diseases were frequently carried by a third person. It is now pretty definitely established that such contagion is possible only for a very short distance, probably but a few feet from the patient, and that communication through a third person is an extremely rare occurrence. In the spread of contagious diseases, articles of clothing, toys, books, furniture and other objects which had been in contact with the patient were once regarded as frequent sources of infection. While it cannot be denied that these are sometimes the vehicles of contagion, this mode of spreading these diseases is certainly infrequent.

Infection, as a rule, is acquired either by contact with or close proximity to a person suffering from a contagious disease. By contact there may be actual transfer of the organism causing the disease. By proximity the specific poison of the disease which is discharged from an infected person, usually in the form of minute droplets by coughing or sneezing, may be inhaled. In this way whooping-cough, epidemic catarrh and measles in the early stage are probably most frequently communicated. Measles and scarlet fever are often spread in the later stages by the discharges from mouth, nose, eyes, ears or glands.

There are two very important sources of infection which are constantly overlooked. The first is the unrecognized case, which escapes notice, in scarlet fever, because of its mild character; and in tuberculosis, because the early stage is so prolonged. The second source is the group of persons known as "carriers." To the latter are very often traced epidemics of typhoid fever and diphtheria; rarely, epidemics of cerebrospinal meningitis and acute poliomyelitis. Carriers are persons who harbor the organisms of infection, usually as the result of a previous attack, sometimes because they have been in close contact with the dis-

ease, but are not themselves at the time suffering from it. The recognition and segregation of these carriers constitute one of the most difficult and important problems in connection with the prevention of communicable diseases.

Infection may take place through the inhalation of dust particles which contain the specific organism of the disease. The bacilli of tuberculosis, diphtheria and typhoid may survive drying and become a part of the dust of the room. While rarely present in the upper air of the room, they may be found in places where dust settles, as on floors, window-sills, etc. Infection of older children or adults by actual inhalation of these organisms with dust is probably very uncommon; but small children, playing much on the floor, may easily acquire infection from dust upon hands, toys, etc., most often through the mouth.

There are certain disease organisms that die so quickly after being discharged from the body that infection by dust is most improbable. Examples of this are the B. influenzae, the meningococcus and the gonococcus.

Epidemic catarrh spreads so rapidly in epidemics that the evidence is stronger in this disease than in any other that it may at times be airborne; but it is more frequently spread through contact or near proximity to infected persons through coughing, sneezing, etc., or from handkerchiefs, clothes, drinking utensils, etc., which have been in contact with patients.

General Care.—In most of the contagious diseases discussed in the following pages the infectious agent is confined to the discharges from the patient's mouth, nose, throat, eyes, ears, sputum or glands. If the spread of these diseases is to be prevented, this poison should be destroyed as soon as it leaves the body. The physician who is in charge of a patient with an infectious disease has a responsibility, not only to the patient and those in immediate contact with him, but to the community. As the same general directions should be followed with all severe communicable diseases, they may well be outlined in this introductory chapter.

The Sick-room.—One with good light and air, so situated as to be easily shut off from the rest of the house or apartment, should be chosen. An open fire and an adjoining bath-room are very desirable. Carpets, rugs, upholstered furniture and all hangings should be removed. Only the simplest and most necessary furniture should be left behind and such books or toys as can be destroyed. An abundant supply of hot water should be provided for, a large slop jar, and plenty of old muslin and absorbent cotton to be used in place of handkerchiefs for discharges, and a supply of paper bags, in which these can be placed for removal. Free ventilation should be secured, and windows should be screened against flies and mosquitoes. The sick-room should be kept

scrupulously clean; especially should all dust be wiped up daily from floors, window-ledges, and railings, with a cloth which has been wrung from a 1-1000 bichlorid solution. The cloths used should be kept in the same solution. The bed linen should be frequently changed, and kept clean. In the room should be a large bowl of carbolic acid, 1 to 40, or some similar solution for cleansing the hands. There is no objection to the hanging of sheets moistened in carbolic, bichlorid, or other disinfectant solutions before the door, but neither this nor hanging them about in the sick-room is to be regarded as having any value in disinfecting the air of the room. They create a false sense of security, and often lead to the neglect of thorough cleanliness.

The *nurse* should wear a washable cap and gown, which she should remove on leaving the room. Rubber gloves are an added protection in severe infections. The nurse should not eat in the sick-room.

The *physician*, before entering the sick-room, should remove his coat and don a cap and gown, kept hanging outside the sick-room for his special use. He should carefully wash his face and hands before leaving the room.

The *patient* being the source of infection, special care should be taken with everything which comes in contact with him. The outer clothing, worn when he was taken ill, should be exposed to sunlight for at least one day and thoroughly brushed in the open air. Underclothing should be boiled for ten minutes and placed in a 5-per-cent solution of carbolic acid. Bed-linen should be soaked in the carbolic solution and boiled in soapsuds before going to the general wash. Handkerchiefs, if used at all, should be treated in the same way. If there is much sputum it should be received in paper cups, which should be burned, or in vessels containing 5-per-cent solution of carbolic acid. All discharges from the mouth, nose, eyes and ears should be collected on old muslin or absorbent cotton, thrown into paper bags and burned. Handkerchiefs should not be used for this purpose. Special disinfection of discharges from the bowels is not needed in the diseases treated in this Section, except in the care of typhoid cases. All remnants of food should be burned. All dishes, knives, forks, spoons, etc., should be boiled in soapsuds and used only by the patient. At the termination of quarantine the patient should receive a complete and thorough bath, including the hair, with soap and water, and entirely clean clothing put on in an adjoining room. Especial care should be given to cleanliness of the mouth and teeth.

The *room subsequent to the illness* should receive the most thorough cleaning. Floors, woodwork and furniture should be thoroughly scrubbed with soap and hot water, walls should be wiped down with damp cloths wrung from 1-1000 bichlorid solution. After severe infec-

tions like scarlet fever and diphtheria, repapering or repainting should be done. Toys and books used in the sick-room should be destroyed or sent to hospitals where similar infections are treated. The mattress and blankets should be sent to a steam disinfecting place, if one is available; if not, they should be exposed for two or three days to sunlight and beaten in the open air, to remove all dust. All washable bedding should be treated as heretofore mentioned. Not only the sick-room but the adjoining room much used by attendants should receive special cleaning. Fumigation will be quite unnecessary if the above directions have been thoroughly carried out. Its value has always been problematical; it is now rapidly being abandoned by health authorities. Its efficacy is in no way to be compared to the special cleanliness heretofore emphasized.

CHAPTER I

SCARLET FEVER

(*Scarlatina*)

Scarlet fever is an acute, contagious, self-limited disease, one attack usually protecting the individual through life. The period of incubation is usually from two to five days; that of invasion, from twelve to twenty-four hours; that of eruption, from four to six days; that of desquamation, from three to six weeks. The disease may be communicated at any time from the first symptom of invasion and even during the existence of purulent discharges from the nose or other mucous or serous membranes. It is usually ushered in by vomiting, fever, and sore throat, and is characterized by an erythematous rash appearing first upon the neck and spreading rapidly over the entire body. Its chief complications are otitis, adenitis, and membranous inflammations of the pharynx, which frequently extend to the nose, rarely to the larynx. The most important sequelae are otitis and nephritis. The constancy of the throat infection in scarlet fever strongly points to the pharynx as the point of entry of the infection.

Etiology.—Analogy leads to the belief that scarlet fever is due to a microörganism, but as yet its nature has not been discovered. The complications are usually associated with the development of a streptococcus. Some have gone so far as to claim that a streptococcus is the cause of the disease. From present knowledge, however, it appears rather to play the rôle of a secondary or accompanying infection, for the development of which the mucous membranes of a person suffering from

scarlet fever seem to afford most favorable conditions. To the streptococcus may be ascribed the membranous inflammations of the tonsils and pharynx, the otitis, the inflammation of the lymph nodes and the cellular tissue of the neck, and probably also the nephritis, endocarditis, pneumonia, and joint lesions. In many of the above conditions the streptococcus is associated with other pyogenic germs, and in some cases with the diphtheria bacillus.

Predisposition.—The susceptibility of children to the scarlatinal poison is much less than to that of measles; still, it is much greater than that of adults. Billington (New York) records observations made in twenty-six families living in tenements where little or no attempt at isolation was made. In these families there occurred forty-three cases of scarlet fever; but forty-seven other children, although unprotected by previous attacks and constantly exposed, did not contract the disease.

Johannessen reports that of 185 children under fifteen years who were exposed, twenty-eight per cent contracted the disease; while of 314 adults, only five per cent contracted the disease. It may be stated that, approximately, not more than one-half of the children exposed take the disease. The susceptibility is slight in early infancy, but it increases until about the fifth year, after which it steadily diminishes. Both sexes are equally liable to scarlet fever. Epidemics are more frequent in the fall and winter than in summer, and cases occurring in the cold months are apt to be more severe. Whitelegge, in 6,000 cases, found the highest mortality in the month of October; and in Caiger's report of 1,008 cases this was also the month showing the greatest mortality.

Incubation.—Of 113 cases [1] in which the period of incubation could be accurately determined, it was as follows:

24 hours or less	6 cases.	8 days	2 cases.
2 days	15 "	9 "	5 "
3 "	28 "	11 "	1 case.
4 "	25 "	14 "	1 "
5 "	6 "	21 "	1 "
6 "	15 "		
7 "	8 "		113 cases.

Thus in eighty-seven per cent of these it was between two and six days, and in sixty-six per cent between two and four days. Speaking generally if, after exposure, a week passes without symptoms, the chances of infection are very small. A short incubation is more frequently seen in severe than in mild cases.

[1] Part of these are from personal observation, but the great majority are isolated cases scattered through medical literature, occurring under circumstances which made it possible to determine the exact length of the incubation period.

Mode of Infection.—The chief source of infection is the patient himself. It is the mild and unrecognized cases which act as carriers to which the spread of the disease is very frequently due. It is somewhat doubtful whether the poison of scarlet fever can be conveyed by the breath, but infection is chiefly by discharges from the mucous membranes involved. Whether it can be conveyed by the scales during desquamation or by the excretions of the patient—urine, feces and perspiration—is a question of grave doubt. It has not been demonstrated. Infection may take place from the carpets or furniture of the sick-room and from the clothing of the patient. Toys or books may be carriers of the disease. A bouquet of flowers sent from a sick-room to an institution has been known to be a vehicle of infection. Cats, dogs and other domestic animals in rare instances have conveyed the disease. Scarlet fever is sometimes spread by milk. The simultaneous occurrence of a considerable number of cases in a community should lead one to suspect the milk supply. All of these sources of infection are relatively infrequent.

The transmission of the disease through a third person is not frequent, but numerous instances of it are on record. The persons most likely to carry it are the nurse and the physician, the latter rarely unless there has been very direct contact with the patient, and when the interval before seeing the second child is short. The transmission of the disease by one who, although living in the house, does not come in contact with the patient, is extremely improbable.

Duration of the Infective Period.—There is no evidence to show that the disease is communicable during the period of incubation. It is slightly contagious from the beginning of invasion, before the rash appears. Infection appears to be most active at the height of the febrile period—from the third to the fifth day.

In simple cases, the average duration of the contagious period may be placed at five weeks, or until discharges from mucous membranes of the nose and throat, the ears and glandular sinuses have ceased. The infectious nature of these discharges has not been sufficiently recognized. One case is recorded in which scarlatina was communicated through a purulent nasal discharge after eleven weeks; another in which the opening of a post-scarlatinal empyema in a surgical ward was followed by an outbreak of scarlet fever.

In winter especially, a chronic pharyngeal catarrh may long contain the infective agent. Ashby found, on careful investigation, that from two to four per cent of patients discharged from a scarlet-fever hospital subsequently conveyed the disease. There is particular danger from a child who has recently had the disease sleeping with other children. Line records a case in which the disease was contracted in this way after

fourteen weeks. It is impossible to say that at any specified time absolute safety exists. All patients before being discharged from a hospital or released from quarantine in private practice, should be carefully examined as to the condition of the mucous membranes, and quarantine continued as long as catarrhal inflammations are present. The poison of scarlatina clings more tenaciously to clothing, upholstery, and apartments than that of any other infectious disease, possibly excepting tuberculosis.

Lesions.—The only characteristic lesions of scarlatina are those of the skin and the mucous membranes of the mouth and throat. The skin is the seat of an acute dermatitis of variable depth and intensity. There is first acute hyperemia, followed by an exudation of serum and cells in the corium, especially about the blood-vessels and hair follicles. There results a death of the epidermis which is thrown off in the desquamation. The mucous membrane of the mouth, tongue, and throat is the seat of a catarrhal, membranous, or gangrenous inflammation which rarely invades the larynx, but very frequently the middle ear and nose. The entire esophagus is often the seat of an intense congestion. From the ear the infection may extend to the mastoid cells, the meninges, or the brain, and from the nose to the accessory sinuses, particularly the antrum of Highmore. All the lymph nodes about the neck may be involved, the infection ending in cell-hyperplasia, suppuration, or necrosis. The cellular tissue of this neighborhood may also become infiltrated, this being followed sometimes by suppuration and occasionally by gangrene.

The most constant change throughout the body, according to Pearce, is hyperplasia of the lymphoid tissue, which is seen everywhere. The other lesions are degenerations due to the scarlatinal poison alone, or in conjunction with the various forms of secondary infection, or to the latter alone. The most important are: fatty degeneration of the heart; areas of focal necrosis in the liver; acute degeneration of the kidney or acute diffuse nephritis; proliferation of the cells of the Malpighian bodies of the spleen; bronchopneumonia, gangrene, or abscess of the lung; pleurisy, which is often purulent; endocarditis, pericarditis; abscesses in the cellular tissue and inflammation of the joints. These visceral changes will be considered more fully under Complications.

Symptoms.—*Invasion.*—As a rule, the invasion of scarlet fever is abrupt, the symptoms at the onset usually being directly in proportion to the severity of the attack. In the majority of cases there is vomiting, a rapid rise in temperature, and soreness of the throat. Often the vomiting is repeated; it is frequently forcible, and without nausea. In severe cases the rise in temperature is very rapid, to 104° or 105° F.;

in the mildest cases it may not be above 101° F. A child may complain of soreness of the throat, or the throat symptoms may be entirely objective. In most severe cases there is a uniform erythematous blush covering the pharynx, tonsils, and fauces, but on the hard palate it appears as minute red points. The appearance of this is usually coincident with the rise in temperature. Occasionally membranous patches may be seen upon the tonsils the first day, but generally not before the third or fourth day. In mild cases the throat shows only a very moderate congestion. Severe cases are sometimes ushered in by convulsions, especially in very young children. Diarrhea is not uncommon in summer. There is general prostration, which is directly proportionate to the height of the fever.

Eruption.—This usually appears from twelve to thirty-six hours after the first symptoms of invasion; exceptionally, not until the third or even the fifth day. A later appearance than this is somewhat doubtful, for the rash not infrequently recedes and reappears, having been overlooked in the first instance. In 108 cases tabulated the duration of the rash was as follows:

Two days or less	5 cases
Three to seven days	81 "
Eight to eleven days	16 "
Over eleven days	4 "
Recurring	2 "

These figures are confirmed by the observations of most writers, that the rash lasts from three to seven days. The full development of the rash is generally seen in from twelve to twenty-four hours from its first appearance, and not infrequently the whole body is covered in the course of four or five hours. Its first appearance is almost invariably upon the neck and chest. Its color is red rather than scarlet, and on close inspection it is seen to be made up of very minute points upon a reddish ground giving the appearance of a uniform blush; or the background may be wanting and only the punctate eruption shows. These points are the papillae of the skin and hair follicles. The rash usually covers the entire body except the face. Even in cases with intense eruption the central part of the face usually escapes, though elsewhere the eruption may be as bright as upon the body. There is often a peculiar pallor about the mouth which is characteristic. The appearance of the eruption in dark-skinned races is much modified and often difficult of recognition. In the negro the palms and soles may be the only places where the eruption can be distinguished. Here may be seen a bright red blush or a fine papular eruption.

Variations in the eruption are very frequent and very puzzling.

They occur especially in the mild and in the most severe cases.

In the mild cases the rash is not seen upon the face; it is often faint upon the body, and may be present only upon certain parts; when the rash is faint or scanty it is usually most marked in the groins and axillae, or over the buttocks and back of the thighs; it may last only one day, and sometimes may be so slight as to escape notice altogether. The eruption may be absent in some very mild cases, in certain others where the throat symptoms are severe, and in malignant cases. In the very severe cases many irregularities are seen, both as to the time of the appearance of the eruption and its character. Sometimes it occurs as large, irregular patches; again, it is macular, closely resembling the rash of measles. Not infrequently an eruption of fine vesicles is seen, especially on the chest, axillae and abdomen. It is seen both in mild and severe cases. A well-developed bright rash indicates strong heart action, and a sudden recession of the rash is a sign of heart failure. Often a rash which is faint and doubtful in character may be brought out fully by a hot bath.

With the eruption at its height, there is intense itching or burning of the skin, and in severe cases considerable swelling, chiefly noticeable upon the hands and face.

Desquamation.—Shortly after the rash has faded, about the eighth day, there begins an exfoliation of the dead epidermis, known as desquamation. This is even more characteristic of the disease than is the rash. It is usually first seen upon the neck and chest, where it appears as fine flakes. The desquamation of the trunk is completed in from one to three weeks. If baths and inunctions are being used, it may be scarcely perceptible. It continues longest where the epidermis is thickest—viz., upon the hands and feet—and here it lasts from four to seven weeks, and not infrequently eight weeks. The appearance of the fingers and toes during desquamation is characteristic. The finger tips usually peel first, and the new epidermis is pink and fresh-looking, while that which has not yet separated is of a dull gray color and loosened at the margin. Occasionally the epidermis of a considerable part of a finger may be loosened at once, so that a partial cast may be thrown off like the finger of a glove. Sometimes the patient comes under observation for the first time during desquamation, the history of the early symptoms being doubtful or absent. Such desquamation as has been described, occurring both upon the hands and feet, may be regarded as conclusive evidence of scarlet fever.

1. *The Mild Cases.*—The symptoms may be so slight as to be entirely overlooked, nothing being noticed until desquamation occurs. Usually, however, there is a rather abrupt invasion, with vomiting and a temperature from 100° to 103° F. The tonsils and pharynx are congested,

while the palate shows a punctate redness somewhat like the cutaneous eruption. The papillae of the tip and borders of the tongue are enlarged. Nearly always within twenty-four hours the rash makes its appearance, generally first upon the neck and chest. Very often it is not seen upon the face, but is abundant on the rest of the body. The rash fades on the third or fourth day, and has disappeared by the fifth day. There is very little prostration, the child often being with difficulty kept in bed.

The highest temperature is coincident with the full eruption, and is usually seen during the first thirty-six hours of the disease. It gradually falls to normal by the third or fourth day. Some examples are shown in Fig. 152. In the mildest cases the temperature may never be above 100° F.

Desquamation is often faint over the body, but is usually unmistak-

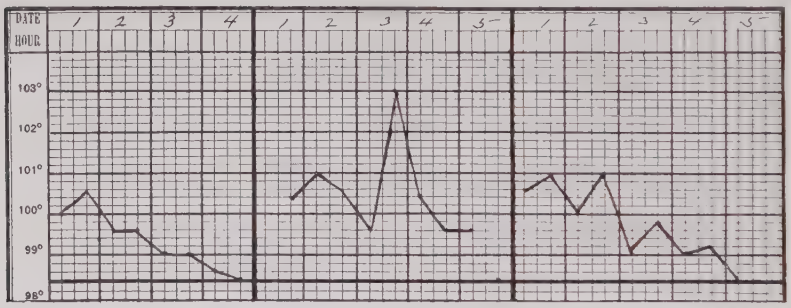


Fig. 152.—Mild Scarlet Fever. Three cases occurring successively in the same family. Diagnosis not made until the third case developed, at which time the first one was found to be desquamating in a typical manner.

able over the hands and feet, always being most marked where the eruption has been most intense.

The mild cases are usually uncomplicated, but the possibility of otitis and of late nephritis should always be kept in mind, as these may occur even with the mildest attacks. The difficulties in diagnosis in mild attacks of scarlet fever are often great. It should be remembered that these cases are just as contagious as severe ones, and that from a mild attack a severe one is often contracted. It is frequently by these mild cases that this disease is spread in schools. In dispensaries, patients desquamating from scarlet fever are often seen who had been attending school regularly up to the time when they were brought for treatment for nephritis.

2. *Cases of Moderate Severity.*—The onset is sudden with vomiting, which is usually repeated, rarely with convulsions. The temperature rises rapidly, and by the end of the first twenty-four hours has reached 104° or 105° F. The rash generally appears within the first twenty-

four hours, and its intensity is usually in direct proportion to the severity of the attack. Appearing first upon the neck or chest, it extends rapidly, covering the entire trunk and extremities, often in a few hours. It is generally typical in appearance, being made up of minute points, but giving the appearance of a uniform blush, which has been compared to a boiled lobster. Little change takes place in the rash for four or five days. After this it fades quite rapidly, and disappears by the sixth or seventh day.

The throat resembles that of the mild form, except that the redness is more intense and there is slight swelling of the tonsils, fauces, and uvula, and often pain upon swallowing. Occasionally small yellowish patches are seen upon the tonsils by the second or third day, but these can be wiped off and are not distinctly membranous. There is usually a moderate discharge of a sero-purulent character from the nose. The lymph glands at the angle of the jaw are swollen and quite tender. The tongue may be coated in the center and show bright red points at its borders and tip, or it may be quite red and show everywhere the prominent papillae—the "strawberry tongue"; while not exclusively seen in scarlatina, this is of considerable diagnostic value. It is rarely seen before the third day, and may continue several days or even weeks.

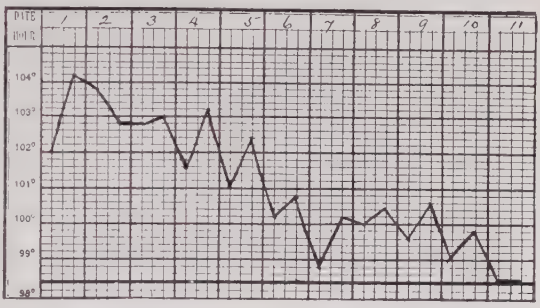


Fig. 153.—Typical Temperature Curve of Uncomplicated Scarlet Fever of Moderate Severity. Girl three years old.

During the height of the fever, restlessness, thirst, and, not infrequently, slight delirium are seen. The temperature usually reaches the maximum by the second day, and falls gradually, but even in uncomplicated cases the fever often lasts from ten to fourteen days (Fig. 153). The pulse in the early part of the disease is rapid, its frequency being usually out of proportion to the height of the temperature. There is much prostration, frequently followed by quite a marked degree of anemia.

This form of the disease rarely proves fatal apart from complications. The complications seen most frequently in this form of scarlet fever are adenitis, otitis, and pneumonia. Nephritis is the only common sequel.

3. *The Severe Cases.*—The severe type of scarlet fever usually declares itself from the beginning. The incubation is short, and the full rash may be seen within a few hours after the initial symptoms. It is

usually intense and covers the entire body, even including the face. In other cases the eruption is delayed, often scanty, and may disappear in a few hours. The disease assumes one of two fairly distinct types; one is characterized by the severity of the general toxemia, the other by the predominance of the throat symptoms. In the first group the toxemia is shown by the height of the temperature, the severity of the nervous symptoms, and the profound cardiac depression. The temperature quickly rises often to 105° or 106° F., and usually remains steadily high until the death of the patient. The nervous symptoms are great prostration and delirium, which is sometimes active, but more often low and muttering. The pulse is very rapid, 160 to 180 being not uncommon; it is weak, compressible, often irregular, and the muscular sounds of the heart are feeble. The urine is scanty and almost invariably albuminous.

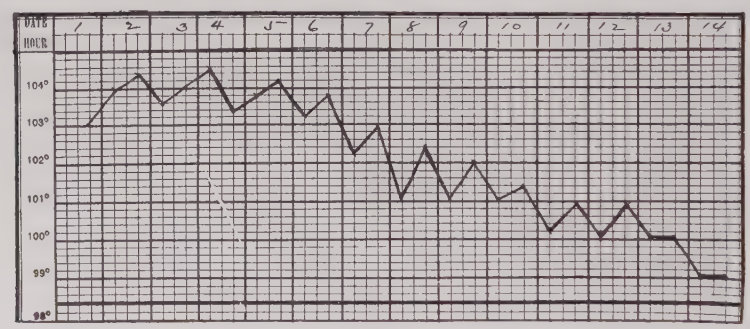


FIG. 154.—TYPICAL TEMPERATURE CURVE OF SEVERE SCARLET FEVER ENDING IN RECOVERY. Prolonged course due to severe throat symptoms lasting from second to sixth day, otherwise uncomplicated; boy twelve years old.

Hemorrhages from the mouth, the nose, or other mucous membranes are occasionally seen. The duration of the disease in this form is generally from five to seven days. Exceptionally the symptoms develop with greater intensity, and death follows in three or four days. A shorter duration than this, the so-called malignant scarlet fever, is rare.

In the second group with predominant throat symptoms, the first three or four days may show nothing more than cases of the moderate type. Membranous patches appear upon the tonsils and spread to the soft palate, uvula, and pharynx, sometimes to the nose and through the Eustachian tube to the ear, very rarely involving the larynx. The mucous membrane of the mouth is intensely congested, and often partly covered by membrane; there are sordes on the lips and teeth, and there may be superficial ulcers, which bleed readily. The glands of the neck swell rapidly, often to a great size, and the cellular tissue about them is infiltrated. The head is thrown back to relieve the dyspnea which the pressure from this swelling occasions. There is an abundant dis-

charge from the nose and mouth; the breath is very offensive. The general symptoms are those of a severe septicemia. The temperature is steadily high, usually between 103° and 105° F., for about a week, after which in cases ending in recovery it slowly falls unless complications develop (Figs. 154, 156, 157); but even in uncomplicated cases the fever sometimes continues for three weeks. In fatal cases the temperature may be steadily high till death (Fig. 155), or it may fluctuate widely. The pulse is rapid, weak, and irregular. There is complete anorexia. There is low delirium or apathy, and sometimes all the symptoms of the typhoid condition are present.

Signs of a bronchopneumonia may be found in the chest, and by

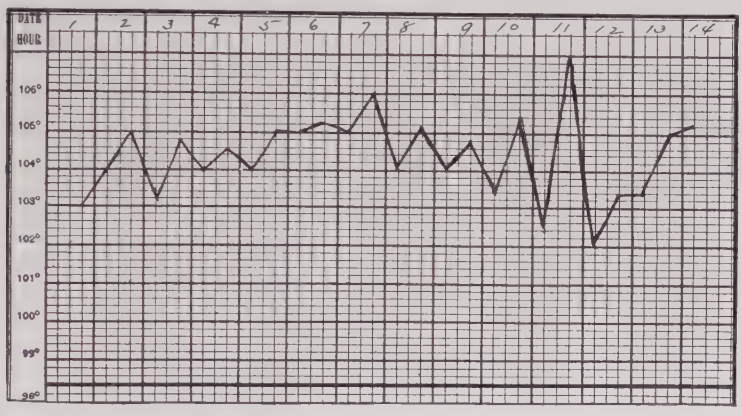


Fig. 155.—Severe Scarlet Fever, Septic Type; Death on Fourteenth Day. Intense angina; otitis; nephritis; necrotic inflammation of cervical lymph glands; girl seven years old; death from heart failure.

the end of the first week or early in the second, acute otitis often develops. The urine is rarely free from albumin, but the amount present is not usually great; there may be hyaline and epithelial casts, and sometimes blood. In some cases the throat symptoms predominate; in others, those of general sepsis, but more frequently the two are combined and are directly proportionate to each other. In still other cases, instead of the membranous inflammation of the throat, it may be of a gangrenous character, and extensive sloughing may take place in the pharynx or the cellular tissue of the neck, sometimes exposing or even opening the great vessels.

The duration of the symptoms in cases with severe angina is from seven to fourteen days. There is increasing prostration and finally a septic stupor, with death from exhaustion, from heart failure, or from some complication—bronchopneumonia, pleurisy, nephritis, hemorrhages following sloughing, pericarditis, or endocarditis. In cases which

recover, the acute symptoms nearly always continue for a full month; and after escaping the dangers of sepsis and the early complications, the child has still to run the gauntlet of all the late complications—nephritis, pneumonia, endocarditis, pyemia, etc. A case may prove fatal as late as the end of the seventh week; nearly all such results are due to nephritis or to its complications.

4. *Surgical Scarlet Fever.*—The existence of a special form of scarlet fever occurring in patients with recent wounds or those who have been subjected to surgical operations, while stoutly maintained by several writers, has been vigorously denied by others. The question is one difficult of solution on account of the close similarity at times existing between the symptoms of scarlet fever and sepsis, and the necessity of deciding in an undoubted case whether the infection with scarlet fever was dependent upon or coincident with the wound.

Hamilton, from a study of 174 reported cases, reached the conclusion that proof of the existence of a special form of scarlet fever rests upon the reports of cases, usually meager, and careful analysis of these would lead one to consider them rather as septic, than as scarlatinal infections; that when there was undoubted evidence of scarlet fever, there was no proof that it was in any way due to the coincident wound, and that there is as yet no convincing proof in the literature that surgical scarlet fever is anything more than scarlet fever in the wounded. On the other hand, there have been observed clinically cases which seem to admit of no other reasonable explanation than that an abrasion of the skin, a recent wound, or even possibly a varicella vesicle, may be the point of entry of the scarlatinal infection, instead of the more usual portal, the pharynx.

Relapses, Recurrences, and Second Attacks.—As a rule, one attack of scarlatina gives immunity through life. The exceptions are very few, but are well authenticated. We have seen but once an undoubted instance of a second attack in the same individual.

Relapses or recurrences within a brief period after the first attack are more frequent. There are to be excluded the cases of pseudo-relapses in which the rash, having temporarily subsided for two or three days, reappears; also those where the rash varies in intensity from time to time; and, lastly, the cases in which, occurring late in the disease, it is due to septicemia or pyemia. They are comparable to the relapses of typhoid fever. They occur most frequently during desquamation, between the seventh and twenty-fourth days. There may be not only a new eruption, but a rise of temperature, sore throat, and vomiting, just as in the initial attack. These recurrences are sometimes shorter and milder than the first attack, but this is by no means uniform, since Körner mentions eight cases where the second attack proved fatal.

In considering the subject of second attacks, the liability to errors in diagnosis must be borne in mind and only cases included which have presented typical symptoms.

Special Symptoms, Complications, and Sequelae.—*Temperature.*—The temperature curve of this disease is quite characteristic. There is usually seen an abrupt rise, the maximum being reached on the second day; there follows a period of variable duration, generally lasting, according to the severity of the case, from two to five days, in which the fluctuations are very narrow; then a gradual decline to normal, which is reached in the milder cases in about a week; in those which are more severe, in about two weeks. This typical curve (Figs. 151 and 152) is seen in the great proportion of uncomplicated cases which end in recovery. Deviations from it, therefore, are important as indicating that some complication exists. The explanation is usually to be found in the development of otitis, adenitis, nephritis, pneumonia, etc. Severe throat symptoms prolong the temperature but do not usually modify its course. In very severe cases ending fatally the high temperature is prolonged. In any case, a rise after the third day is unfavorable.

Throat.—Three distinct forms of angina are seen in scarlatina: simple or erythematous, membranous, and gangrenous.

1. Erythematous Angina.—This can hardly be ranked as a complication, as it is nearly as constant as the scarlatinal rash. Usually there is only the intense general blush over the entire pharynx with the fine red points upon the hard palate; but there may be seen upon the tonsils grayish-yellow spots resembling those of follicular tonsillitis, which can be wiped off, leaving a clean surface. This simple angina is at its height with the maximum temperature, and fades as the temperature falls.

2. Membranous Angina.—These cases were formerly classed as scarlatinal diphtheria. Cultures, however, have shown that the great majority of these inflammations are not true diphtheria, but are due to the streptococcus.

The lesions of this form of angina are considered in the chapter on Membranous Tonsillitis. Usually on the second or third day of the disease an exudation appears upon the tonsils, and in the milder cases it covers only the tonsils. In the most severe form it may be seen within twnty-four hours of the onset, sometimes before the eruption appears. Beginning upon the tonsils, the membrane rapidly spreads to the entire pharynx, the mucous membrane of the nose, the mouth, the Eustachian tube, and even to the middle ear. In color it may be gray, greenish, or almost black. The infiltration of the cellular tissue of the neck and the enlarged lymph glands produce great external swelling, which may extend like a collar from ear to ear. The breath has a foul odor, the

nasal discharge is thin and fetid, and nasal respiration is obstructed, so that the mouth is open constantly. It is surprising that the larynx is so seldom invaded.

These local changes are accompanied by constitutional symptoms of great severity, which are due to a general streptococcus septicemia; bronchopneumonia and nephritis are very frequent, otitis is almost constant, and suppuration of the lymphatic glands is not uncommon. The eruption is often irregular and late in appearing.

The frequency with which diphtheria coexists with scarlatina varies greatly. In hospital practice the proportion often runs as high as thirty or forty per cent. In private practice it is much lower. The streptococcus angina is usually seen at the height of the disease; true diphtheria may occur at any time, even during convalescence. The only positive means of differentiation is by cultures, which should invariably be made from the throat of every patient admitted to a scarlet-fever hospital, and of every case in private practice showing any exudate upon the tonsils.

3. Gangrenous Angina.—This is seen only in the worst cases of scarlet fever. The process may be gangrenous from the outset, or preceded by a membranous inflammation. It is sometimes insidious in its development. There is a fetid odor to the breath, an irritating discharge from the nose and mouth, with very great glandular swelling. The tonsils are gray or grayish-black in color, and large masses of necrotic tissue may be removed with the forceps from the tonsils, uvula, fauces, or pharynx, and sometimes sloughing occurs in the cellular tissue of the neck. Blood-vessels of considerable size are sometimes opened, and serious or even fatal hemorrhage may result. The constitutional symptoms are those of great asthenia, prostration, and profound cachexia, followed almost invariably by a fatal termination.

Lymph Nodes.—These are swollen in all cases accompanied by severe angina. The inflammation may be simply an acute hyperplasia, or it may go on to suppuration and necrosis. Abscess does not often occur at the height of the disease, but the early swelling may almost completely subside only to recur, and suppuration may take place even as late as the fifth or six week of the disease. It may be confined to the glands or be complicated by suppuration in the cellular tissue of the neck.

Cellulitis of the Neck.—This usually occurs toward the end of the first week, and is associated with grave throat symptoms. Rapid and extensive infiltration occurs, the skin becomes tense and brawny, the head is held back, and there may be considerable dyspnea. The infiltration may be only in the neighborhood of the lymph glands or it may be diffuse. Unless relieved by early incision, the diffuse form may result in suppuration and extensive sloughing, which may be deep

enough to lay bare the large vessels of the neck. This is a complication of the gravest possible import. Death may occur from septicemia before or after sloughing or from hemorrhage due to opening by ulceration of the external carotid or some of its branches; or there may be associated thrombosis of the jugular vein, leading to thrombosis of the lateral sinus, meningitis, or pyemia.

Ears.—The otitis is due to direct extension of the infection from the rhinopharynx. It is the most frequent complication of scarlatina, and in doubtful cases may have some diagnostic importance. As a rule, the younger the child the greater the liability to otitis. It is more frequent in winter than at other seasons, and is closely connected with the severity of the throat symptoms. Of 4,397 cases reported by Finlayson,

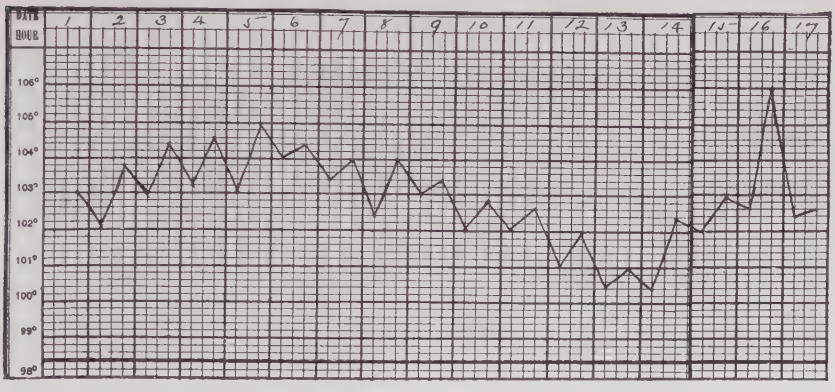


Fig. 156.—Severe Scarlet Fever; Otitis; Mastoiditis; Death. Typical symptoms and temperature curve until fourteenth day; secondary rise of temperature from otitis; double paracentesis on the fifteenth day; mastoid operation on the sixteenth day; death twelve hours later from septicemia; boy five years old.

otitis occurred in 10 per cent, and of 1,008 cases reported by Caiger, in 13 per cent. In Burkhardt's statistics the proportion was as high as 33 per cent. Of cases accompanied by severe throat symptoms otitis is present in fully 75 per cent.

As a rule, both ears are affected. Otitis is most frequent early in the second week, but may occur at any time, even during convalescence. When it develops at the height of the disease there are in some cases no new symptoms; in others there is pain and deafness and a rise in the temperature, which may fall after paracentesis or rupture of the drum membrane, or there may be extension to the mastoid (Fig. 156). The otitis is often overlooked unless the ears are regularly examined. The form of inflammation may be catarrhal or purulent, the latter being often accompanied by necrotic changes.

Bezold makes the following report upon 185 cases showing the dis-

astrous consequences of scarlatinal otitis: "In 30 there was entire destruction of the membrana tympani; in 59 the perforation comprised two-thirds or more of the membrane; in 15 there was total loss of hearing on one side, and in 6 of the cases upon both sides; in 77 of the cases the hearing distance for low voice was less than twenty inches."

As a cause of permanent deafness and deaf-mutism, no disease of childhood compares in importance with scarlet fever. May has collected statistics of 5,613 deaf-mutes, of whom 532 owed their condition to otitis following scarlet fever.

Kidneys.—Albuminuria accompanies nearly all the severe cases of scarlet fever. In many this is simply the ordinary febrile albuminuria due to acute degeneration of the kidneys. In those with severe throat complications, and in nearly all the septic cases, there is an acute diffuse nephritis; the interstitial changes may be very marked and the kidneys

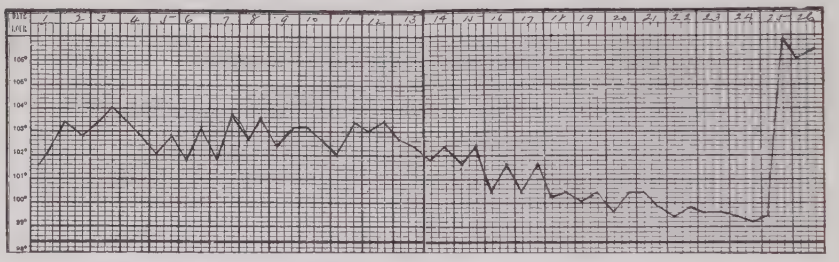


Fig. 157.–Scarlet Fever of Moderate Severity Followed by Fatal Nephritis. Early symptoms typical and uncomplicated; twenty-first day vomiting; twenty-fifth day uremic convulsions; death twenty-sixth day. No dropsy; urine never below 10 ounces in twenty-four hours; girl ten years old.

contain minute abscesses. This occurs at the height of the febrile process and is rarely accompanied by dropsy; but albumin, casts, and even blood may be found in the urine. The most severe and the most characteristic renal complication, and that generally designated as *post-scarlatinal nephritis*, is a diffuse nephritis, with changes in the glomeruli as the most striking feature. It usually develops during the third or fourth week of the disease, and may follow mild as well as severe cases (Fig. 157). The onset may be gradual, with dropsy and urinary changes, usually accompanied by a slight rise of temperature; or it may be abrupt, without dropsy but with convulsions, suppression of urine, and very high temperature.

The characteristic urine is of a reddish or smoky color and scanty. It contains a large amount of albumen, often sufficient to render the urine solid upon boiling. Under the microscope there are seen red blood-cells, pus cells, epithelial cells, and casts of every variety. Death may take place from acute uremia, or the attack may be followed by

permanent damage to the kidneys. It is more fully described with the Diseases of the Kidney.

Joints.—Acute articular rheumatism may occur coincidently with the development of the scarlatinal rash, and occasionally during convalescence in patients who have a predisposition to that disease. Acute swelling of the joints is sometimes of pyemic origin. In pyemic arthritis the large joints are usually involved and the lesions are apt to be multiple. Joint disease may occur as a sequel of scarlet fever, when it is secondary to disease of the bone or to periarticular abscesses opening into the joint.

The foregoing include but a small proportion of the joint complications seen in scarlet fever. The most frequent and most characteristic form of inflammation is *scarlatinal synoritis*, often improperly called *scarlatinal rheumatism*. It occurs in different epidemics with varying frequency. Carslaw (Glasgow), in 533 cases of scarlet fever, met with synovitis in 60 patients. It is seldom seen in children under three years of age, and is most frequent after five years. It may occur in mild as well as in severe cases. According to Ashby, synovitis develops toward the end of the first or the beginning of the second week. The symptoms are generally mild, and are followed by prompt recovery. Suppuration is rare. Any of the joints may be attacked, but those of the wrist, hand, elbow, or knee are most frequently affected. The symptoms are redness, moderate pain, swelling, which is usually due to synovial distention, and sometimes a slight rise in temperature. The duration is generally but three or four days, and in most cases there is spontaneous recovery. Besides these milder cases there occurs a much more severe form, which may develop later, even during convalescence. It is not very acute, but is accompanied by fever, and both the fever and swelling may continue for many weeks. Recovery may be complete or some joint disability may remain and chronic arthritis may follow.

Lungs.—The pulmonary complications of scarlet fever are neither so frequent nor so important as those of measles. Bronchopneumonia is usually found at autopsy in septic cases where death has occurred later than the third or fourth day, but it is not generally recognizable so early by physical signs.

In septic cases pleuropneumonia sometimes occurs early in the disease and at other times late, generally associated with nephritis, but occasionally without it. It is not infrequently a direct cause of death. Empyema may follow pleuropneumonia or occur with pyemia or nephritis, but with the latter, simple serous pleurisy is more common. Edema of the lungs occurs chiefly with nephritis, in which it is the most common cause of death.

Heart.—Cardiac murmurs are frequent at the height of the disease;

in fact they are heard in almost all severe cases. Endocarditis and pericarditis are oftenest seen in septic cases, and with post-scarlatinal nephritis. Endocarditis may be simple or malignant, and may lead to embolism during convalescence. Some degenerative changes in the cardiac muscle are probably present in all the severe cases. Acute dilatation may result, which is sometimes a cause of death.

Blood.—In all cases there is a rapidly progressing anemia that lasts into convalescence. The reduction in the red cells in an average case is about one million. The chief interest, however, attaches to the number and character of the white cells. In mild cases there may be only a moderate increase in their number, usually to from 10,000 to 14,000. It is in cases of moderate severity that the characteristic changes are found. In these there is a decided leucocytosis which appears early, attains its maximum about the fourth day, and gradually declines until the normal is reached, which may not be until the third, fourth, or fifth week. The maximum is usually about 30,000 to 35,000; although it may be as high as 75,000. During the first week the polymorphonuclear neutrophiles form from 90 to 95 per cent of these cells; the eosinophiles as well as the lymphocytes are diminished. After the fifth or sixth day, there is a rapid increase in the eosinophiles which attain their maximum —sometimes 20 per cent of the total leucocytes—between the fourteenth and twenty-first days. After the third week they gradually diminish. Exceptionally there is found in convalescence a relative lymphocytosis, which may be as high as 50 per cent. Complications, nephritis excepted, usually cause actual as well as relative increase in the polymorphonuclear neutrophiles. In malignant and rapidly fatal cases there is usually a very small proportion of eosinophiles, and little if any leucocytosis, though exceptionally it may be high. Much has recently been written regarding the so-called "inclusion bodies" which are found in the leucocytes in this disease. It seems clear that they are not specific and that their presence is not diagnostic of scarlet fever. They are regularly found in all but the mildest cases before the fourth day; but they are found also in other conditions, e. g., pneumonia, sepsis and erysipelas.

Digestive System.—Functional disturbances are very frequent, and, in fact, are seen in most of the cases, but organic changes are rare. Vomiting is the mode of onset in the majority of cases, but rarely continues throughout the attack. Diarrhea may be associated with it under both conditions. The tongue is nearly always coated, and clears off in quite a characteristic way, which, with the prominent papillae, gives rise to the "strawberry" appearance. Catarrhal stomatitis is a very frequent complication, and in many cases of severe membranous angina the same process is seen in the buccal cavity.

Nervous System.—Nervous complications and sequelae are seen less frequently with scarlatina than with most of the infectious diseases of such severity. Convulsions are frequent at the outset, and generally indicate a severe attack, though not invariably so. Occurring late in the disease, they are usually due to uremia. Meningitis may occur as a complication of otitis, in pyemic cases, and sometimes with post-scarlatinal nephritis. Paralysis from peripheral neuritis is rarely seen. Hemiplegia sometimes occurs from meningeal hemorrhage, or from embolism secondary to endocarditis and associated with nephritis. Chorea was noted as a sequel in only three of 533 cases reported by Carslaw. In a report of 187 cases of epilepsy, Wildermuth states that it followed scarlet fever in 12 cases. Insanity has been occasionally observed, the usual form being acute mania, with complete recovery in a few weeks or months.

Gangrene.—Cases of symmetrical gangrene after scarlet fever have been reported. The parts generally affected are the buttocks, thighs, and arms, but it may occur almost anywhere. The pathology of these cases is obscure. The process usually begins in several places simultaneously, or in rapid succession, and advances steadily till death occurs.

Other Infectious Diseases.—Diphtheria is most frequently seen, and may be present even when there is no distinct membrane.

Scarlatina may also be complicated by measles, varicella, or facial erysipelas, and occasionally by variola or typhoid fever. The symptoms are often an irregular commingling of those belonging to the two diseases. They may begin simultaneously, or more frequently one develops as the other is subsiding.

Diagnosis.—The characteristic symptoms of scarlet fever are the abrupt onset, usually with vomiting, the marked elevation of temperature, the erythematous condition of the throat, the punctate eruption on the hard palate, with the appearance of the rash within twenty-four hours, and later the characteristic appearance of the tongue. The difficulties of diagnosis usually depend upon irregularities in the eruption. The variations are seen in the mildest and in the most severe cases. In the former the rash may be of short duration, often less than a day, and in consequence easily overlooked; or it may be present only upon certain parts of the body instead of being diffuse. In every doubtful case the groins, axillae, and loins should be closely scrutinized for a punctate eruption. In very severe attacks the rash may appear late or recede after being fully out, or it may be hemorrhagic or in irregular blotches. In any case, too much stress should not be placed upon the rash alone.

Until we have some exact means of laboratory diagnosis as in typhoid fever, malaria, and diphtheria, an absolute diagnosis will in

certain cases be impossible. Sometimes the diagnosis remains doubtful until the end, although occasionally confirmatory evidence may be obtained even in convalescence. Thus, a patient may desquamate in a manner so typical as to leave no doubt as to the nature of the preceding illness; again, the occurrence of a characteristic sequel, such as nephritis in the third or fourth week, may testify strongly for scarlatina as the primary disease; and, finally, the outbreak of undoubted cases among children who have been in contact with the patient is practically conclusive, always provided other sources of infection can be excluded. Desquamation, however, follows so many other eruptions that when slight or irregular, one should not rely upon it as an evidence of scarlet fever, but only upon a typical exfoliation upon the hands and feet. It is a point of some practical importance not to oil the skin of a patient when awaiting desquamation for diagnosis, as this alters very much the characteristic appearances. In some puzzling epidemics the length of the incubation may be of material assistance in the diagnosis; when this is regularly more than a week, one may be pretty sure that he is not dealing with scarlet fever.

Scarlet fever with severe throat symptoms and doubtful eruption can be distinguished from diphtheria only by cultures. Measles is distinguished by the length of the invasion, the catarrhal symptoms, and the slowly spreading eruption, but most of all by Koplik's spots. Much more difficult is it to distinguish between mild scarlatina and rubella. In rubella the important thing is that, although the rash may be well marked, often covering the body, the constitutional symptoms are few or entirely absent. In scarlet fever with an eruption of the same intensity there is almost invariably a considerable elevation of temperature, usually 102° or 103° F., and a bright-red throat.

There are so many skin eruptions which may resemble that of scarlet fever, that it is always hazardous to make the diagnosis of this disease from the eruption alone. This is especially true of sporadic cases occurring in infants; there is seen at this age a great variety of eruptions, usually associated with digestive disturbances, which closely simulate a scarlatinal rash; but most of them are of short duration. A scarlatiniform erythema is occasionally seen after diphtheria antitoxin, also in influenza, typhoid fever, pneumonia, and varicella, which may cause them to be mistaken for scarlet fever, or may lead to the conclusion that both diseases are present. The same is the case with the septic erythema occurring in surgical patients. Belladonna, quinin, and occasionally antipyrin, the salicylates and aspirin may produce eruptions more or less closely resembling that of scarlet fever. This is also true of some cases of urticaria and other forms of skin disease. Eruptions resembling scarlet fever may also arise from irritation due

to clothing, to heat, to the local application of irritants to the chest, such as camphorated oil, etc. There is little doubt that many of the cases reported as relapsing scarlatina are really examples of recurring erythema, particularly as some of the latter are followed by a desquamation which is very similar to that after scarlatina.

Prognosis.—There is no disease in which it is more difficult to foretell the outcome than in scarlet fever. Cases apparently of the mildest type not infrequently develop serious symptoms and even complications that could not be foreseen. Symptoms indicating a bad prognosis are, very high temperature, especially one which continues to rise for the first three or four days, and severe nervous and throat symptoms. The most common cause of death is the disease itself, the scarlatinal toxemia. From this cause more than half the deaths occur. Next are the complications, cardiac, pulmonary, renal, otitic, mastoid and cerebral, given in the order of their frequency. The mortality of scarlet fever varies much in different epidemics. In some, nearly all the cases are of a mild type, and the mortality may be as low as 3 or 4 per cent; in others, a severe or malignant type prevails, and it may be as high as 40 per cent. The disease is, as a rule, more fatal in the youngest infants, becoming less so as age advances.

The following are the mortality records from various European sources:

Ashby, Manchester Hospital	681	cases;	mortality,	12.2	per	cent.
Koren, a single epidemic	426	"	"	14.0	"	"
Bendz, Copenhagen	22,036	"	"	12.2	"	"
Ollivier, three Paris hospitals for five years	893	"	"	14.0	"	"
Fleischmann, five epidemics	1,356	"	"	10.0	"	"

The general mortality of the disease may therefore be assumed to be from 12 to 14 per cent; it is, however, much higher than this among young children, as shown by the following figures:

New York Infant Asylum	116	cases	under	5	years;	mortality,	20	per cent.
Ashby, Manchester Hospital	259	"	"	5	"	"	23	" "
Bendz	not stated	"	"	5	"	"	13	" "
Heubner	136	cases	"	7	"	"	30	" "
Fleischmann	not stated	"	"	4	"	"	43	" "

Under five years of age the average mortality from scarlet fever is, therefore, between 20 and 30 per cent.

Prophylaxis.—Even the mildest cases should be isolated for four weeks. If complications exist, such as otitis, rhinitis, pharyngitis, empyema, or suppurating glands, the quarantine should be continued until these conditions are cured. Patients should not be allowed to mingle with other children for at least a month after all symptoms have sub-

sided, and should be forbidden to sleep with other children for three months. Children in the family who have not been exposed to the disease should be immediately sent away; and those who have been exposed, separately quarantined for at least a week.

After recovery, the patient, before he is released from quarantine, should have one thorough bath, the entire body, including the hair and scalp, being scrubbed with soap and water, and every particle of clothing changed.

The nurse should be quarantined with the patient, and should not mingle with other members of the family until a complete change of clothing has been made and hands and face thoroughly washed. The nurse and all others in close contact with a severe case should use frequently an antiseptic gargle and a nasal spray. The care of the room during and after the attack has been considered in the introductory pages of this Section.

Schools are hot-beds for the spread of scarlet fever. The greatest sources of danger are the mild or walking cases in which the disease has not been recognized, and the clothing of patients who have had a severe form of the disease. As a rule, a child should be kept from school six weeks from the beginning of the attack, and the certificate of a physician should be required before readmission. Other children in the household should not be allowed to attend schools of any kind during the period of active symptoms; they should be kept at home on the average for a month. This precaution is necessary, first, because they might carry the disease from the patient at home; secondly, because otherwise they might themselves attend school while suffering from the disease in a very mild form or during the period of invasion. When the sick child is completely isolated, the danger from the first source is very slight. During severe epidemics it frequently becomes necessary to close all schools.

In general, it is to be remembered that the danger is first from the patient, secondly from the room, and thirdly from the nurse. Special attention should always be given to the complete and immediate isolation of the first case which appears in an institution or community, which should apply to mild as well as severe forms of the disease.

Treatment.—There is as yet no specific for scarlet fever. The physician's duty in the average case consists in (1) establishing proper quarantine and the carrying out of adequate means of disinfection; (2) the hygienic care of the patient; (3) directing the diet; (4) watching for complications, especially otitis and nephritis. It should be borne in mind that otitis is rarely accompanied by pain or tenderness, and is recognized only by an examination of the ears; also that severe and fatal nephritis may follow mild as well as severe cases.

Mild attacks require no medicine. Children should be kept in bed at least a week after the fever has subsided, and upon a diet chiefly of milk and farinaceous food with plenty of water for a period of three weeks. This is an important matter in the prevention of nephritis. During the height of the eruption, the intense itching of the skin may be allayed by sponging with a bicarbonate of soda solution, or by inunctions with vaseline, or by the free use of rice or talcum powder. Plenty of fresh air should always be secured in the sick-room. As soon as the fever and rash have disappeared, daily warm baths with soap and water should be used, after which the entire body should be anointed with vaseline, with the purpose of facilitating desquamation.

The temperature does not usually require interference when it only occasionally rises to 104° or 104.5° F. But if there is hyperpyrexia, or a temperature which ranges from 104° to 105.5° F. or over, antipyretic measures are called for. Hydrotherapy is much safer and more certain than drugs. Sometimes sponging is sufficient, but in the great proportion of cases the pack or bath is required. The use of water in the reduction of temperature is especially indicated in septic cases with typhoid symptoms, and in those with pronounced cerebral symptoms. The temperature of the water employed will depend upon the duration of its application. It is generally better to use prolonged sponging or bathing with tepid water than water at a lower temperature for a shorter period.

The nervous symptoms are frequently better controlled by ice to the head and by cold sponging than by medication. Antipyretic drugs may occasionally be useful to control restlessness and promote sleep, and in mild cases to effect a moderate reduction in temperature. Phenacetin is usually to be preferred.

As soon as the pulse becomes weak or rapid and irregular, or the first sound of the heart feeble, stimulants should be given, no matter at what stage of the disease. In septic, or malignant cases, or in those accompanied by severe angina, adenitis, or cellulitis, stimulants should be used freely. Digitalis is especially valuable when the pulse is weak and the tension low. It may be given alone or combined with caffein; one minim of the fluid extract of digitalis, and gr. $\frac{1}{2}$ of caffein being the initial doses for a child of five years.

The erythematous sore throat requires no treatment except the use of a bland gargle. If there is a profuse nasal discharge, gentle nasal syringing with a warm saline or boric-acid solution may be used with the hope of preventing infection of the middle ear. The local treatment of the throat is the same as that of other cases of severe angina.

Milder forms of adenitis require no local treatment. When severe, the glands should be covered with ichthyol, and an ice-bag applied

continuously. Poulticing almost invariably does harm. If an abscess forms, early incision should be made.

The ears of patients with severe throat symptoms should be examined daily in order that there may be no delay in performing paracentesis should this become necessary. Any unusual rise in temperature should direct attention to the ears. The indications for the operation are the same as in other severe forms of otitis.

The physician should be constantly on the watch for the development of nephritis, not only during the febrile period, but also during convalescence. Repeated examinations of the urine are absolutely necessary. These are facilitated by having a rack of test tubes and the ordinary reagents for detecting albumin in the sick-room, so that the physician may himself examine daily a fresh specimen of urine. The nurse should be instructed to measure and record accurately the twenty-four hours' urine throughout the attack. The treatment of scarlatinal nephritis has been considered in the chapter devoted to Diseases of the Kidney. Diffuse cellulitis of the neck calls for free, early incision as the only means of preventing extensive sloughing.

On the assumption that streptococci, though not the cause of the disease, are still responsible for most of the serious complications of scarlet fever, sera prepared by means of several different strains of this organism have been produced and extensively used but without any uniform or striking success. One has been produced by Moser (Vienna), concerning whose effects there is much more favorable evidence. Escherich, Bokay, and other reliable Continental observers in their reports have declared that its effects are not less striking than those obtained from diphtheria antitoxin. It must be given in very large doses, from 100 to 200 c. c. The value of streptococcus serum has not yet been demonstrated in this country.

Transfusion and the intramuscular injection of blood or of blood serum from patients convalescent from scarlet fever have been employed by Zingher (New York) and others in very severe forms of scarlatinal toxemia. The beneficial results which have followed have in some instances been so striking that they can hardly be considered accidental. In desperate cases this should be tried whenever practicable. Considerable amounts of blood must be used, from 100 to 300 c. c., according to the age of the patient. Some benefit also under similar conditions seems to follow the injection of normal blood from healthy persons.

During convalescence, the urine should be frequently examined. Antiseptic gargles and a nasal spray should be used as long as a purulent discharge from the nose or pharynx continues.

CHAPTER II

MEASLES

(*Rubeola, Morbilli*)

Measles is an epidemic contagious disease, more widely prevalent than any other eruptive fever; very few persons reach adult life without contracting it. One attack usually confers immunity. It is highly contagious even from the beginning of the invasion, and spreads with great rapidity from the patient to all susceptible persons exposed. The infectious agent, however, does not cling to clothing or apartments as does that of scarlet fever. Measles has a usual incubation period of from eleven to fourteen days; a gradual invasion of three or four days with symptoms of an acute coryza, and a maculopapular eruption which appears first upon the face and spreads slowly over the body, and which lasts from four to six days. This is followed by a fine bran-like desquamation, which is complete in about a week. The mortality is low, except among infants and delicate children, in whom it may reach 30 or even 40 per cent. In institutions for infants and young children no epidemic disease is more to be dreaded than measles, not only on account of its severity, but from the frequency with which, in such subjects, it is complicated by bronchopneumonia.

Etiology.—Little is as yet known of the essential cause of measles. Anderson and Goldberger, and since them a number of others, have succeeded in inoculating monkeys with the blood and also with the nasal and buccal secretions from patients with measles and have produced a disease attended by fever, eruption and respiratory symptoms which are believed to be identical with measles in the human subject. They have successfully carried the strain of infection by blood inoculations through six generations of monkeys. The virus obtained by Anderson and Goldberger passes through the Berkfeld filter, resists drying and freezing for twenty-four hours, but is destroyed by a temperature of 55° C. Blood from patients with measles was found to be infective at least twenty-four hours before the eruption and for twenty-four hours after its appearance. Later than this its infectivity is much lessened and soon disappears. The secretions from the mouth and nose were infective for the monkey only when collected during the stage of eruption. The experiments suggest a rapid loss of infectivity with the beginning of convalescence. Attempts to convey the disease to animals by inoculating with scales from desquamating patients were unsuccessful.

Clinical observations indicate that the virus of measles is more readily diffused than that of most communicable diseases; also that its viability is less than most pathogenic organisms. Only a short exposure is required to communicate the disease.

Predisposition.—Infants under six months do not readily contract measles, but all other children are extremely susceptible. In an epidemic reported by Smith and Dabney, 110 unprotected children, between the ages of eight and eighteen years, were exposed and only two escaped. In one institution epidemic observed by us there were 62 children over two years of age; five were protected by a previous attack and escaped; of the remaining 57 children, 55 took the disease. There were also in the institution 113 children under two years old; of this number 78 per cent took the disease; but, although a number were exposed, not one child under six months old contracted measles. We have, however, seen in one instance a typical attack of measles in an infant of seven months, the disease having been contracted in this case from the mother. The age of the persons affected depends much upon the length of time since the last outbreak of the disease. In an epidemic occurring in the Island of Guernsey, where the disease had not prevailed for many years, all ages were affected, the youngest being twelve days old, and the oldest, a man and wife, each aged eighty years. Instances have been reported by Somer, Gautier and others in which the eruption of measles has either been present at birth or has developed within a few hours after birth, when the mother was suffering from the disease at the time.

Except, then, in early infancy, the probabilities are very strong that every child exposed to measles will contract the disease. Occasionally, however, one is seen who seems insusceptible, no matter how close the exposure.

Epidemics of measles are more frequent and more severe during the winter and spring months. They are least frequent and mildest during the summer and autumn months.

Incubation.—In 144 cases,[1] in which the period of incubation could be definitely traced, it was as follows:

Incubation of less than nine days	3	cases.
" " nine or ten days	22	"
" " eleven to fourteen days	95	"
" " fifteen to seventeen days	19	"
" " eighteen to twenty-two days	5	"

Thus in 66 per cent of the cases the incubation was between eleven and fourteen days, and in only one case was it less than a week. The con-

[1] About twenty-five of these are taken from our own records; the remainder are mainly isolated cases, scattered through medical literature. The incubation is reckoned from the time of exposure to the beginning of catarrhal symptoms.

stancy of the incubation period is strikingly shown in some epidemics. Thus in the one reported by Smith and Dabney in an institution in Virginia, exactly eleven days after the rash appeared in the first case, the disease developed in twenty children—no cases having occurred in the interval.

Duration of the Infective Period.—This is much shorter than in scarlet fever, and the average duration may be placed at two and a half weeks. The minimum period of isolation should be ten days after the appearance of the eruption. It should be extended if there persist discharges from the nose and throat or a cough. Haig-Brown discharged fifty-eight cases on or before the twenty-ninth day of the disease, and in no instance was measles spread by these children. Ransom, however, records one instance in which it was communicated thirty-one days after the appearance of the rash.

Measles is highly contagious from the very beginning of the catarrhal symptoms. A case occurred under our observation in which a child conveyed the disease four days before the rash appeared; and many such have been observed. An instance is known to us where, of thirteen little girls at a children's party, only one (protected by a previous attack) escaped measles; the source of infection was a child who showed no rash until the following day. The period during which the disease is most contagious is still a matter of dispute, the general belief being that it is coincident with the most severe catarrhal symptoms and the beginning of the eruption.

With the fading of the eruption and the subsidence of the catarrh, the communicability of measles diminishes rapidly. It is generally proportionate to the severity of the catarrhal symptoms, and when these are protracted it is probable that the disease may be communicated for a much longer period than in the usual case.

Mode of Infection.—Measles is usually spread by direct exposure to an affected person. The infectious agent is chiefly disseminated by the minute droplets which are given off during coughing and sneezing, probably also by the discharges from any affected mucous membrane. Proximity to a patient seems necessary to contagion, but not actual contact. Infection from the scales during desquamation probably does not occur. It is very infrequent that measles is conveyed through the medium of clothing, furniture, or a third person. Though a good many instances are on record in which the disease has been carried by a third person, this, after all, very rarely happens and we think never unless the contact both with the sick and the well child is very close and the interval short.

Lesions.—The only constant lesions of measles are those of the skin and the mucous membranes, chiefly of the respiratory tract. According

to Neumann, the process in the skin is of an inflammatory character, but is more superficial than in scarlet fever. There is congestion, accompanied by an exudation of round cells about the small blood-vessels, and also about the sweat and sebaceous glands, and the papillae. To this exudation and the edema, the swelling of the skin is due. It occurs everywhere, but is especially noticeable upon the face.

The changes in the mucous membranes are quite as much a part of the disease as are those of the skin. There is a catarrhal inflammation affecting the conjunctivae, nose, pharynx, larynx, trachea, and large bronchi, which varies in intensity with the severity of the attack. In the most severe forms in infants and in young children, this inflammation extends with great uniformity to the small bronchi, and usually to the air vesicles, causing bronchopneumonia. In severe cases, the lesion in the pharynx and larynx also, instead of being catarrhal, may be membranous; the larynx being much more frequently involved, and the ears much less so, than in scarlet fever. Freeman has described areas of focal necrosis in the liver similar to those found in diphtheria: they were present in four of twelve cases examined. The lesions of the lungs and of other organs will be more fully considered under the heading of Complications.

The bacteria which are associated with the lesions of the respiratory tract are the staphylococcus and the streptococcus, separately or together, and either form may be associated with the pneumococcus (see Bacteriology of Bronchopneumonia). Measles produces conditions in the mucous membranes of the respiratory tract which are especially favorable for the development of these bacteria. They are present in the mouth in great numbers; they may cause pneumonia, otitis, and other local inflammations, and the pneumococcus or streptococcus may produce a general septicemia.

Symptoms.—*Invasion.*—As a rule, the invasion of measles is gradual, both the fever and catarrhal symptoms increasing steadily up to the appearance of the eruption. The characteristic symptoms of the invasion are those of a severe coryza—suffusion of the eyes, increased lachrymation, photophobia, sneezing, and a discharge from the nose. The hoarse, hard cough indicates that the catarrhal process has involved the larynx and trachea, as well as the visible mucous membranes. Frequently the patient complains of some soreness of the throat, and on inspection there is seen moderate congestion of the tonsils, fauces, and pharynx. On the hard palate are frequently seen small red spots. Much more characteristic are the minute white spots upon the mucous membrane of the cheeks, known as Koplik's sign (see Diagnosis). The constitutional symptoms are indefinite, and may be met with in almost any disease. These are dulness, headache, pains in the back, and the

usual symptoms of malaise; there is rarely vomiting or diarrhea. Drowsiness is a frequent symptom, and is regarded by the laity as characteristic.

The exceptional cases in which the invasion is abrupt are puzzling. There may be a sudden accession of fever with vomiting, and even convulsions, as in a case lately under our observation. Not infrequently, when the disease prevails epidemically, the invasion is sudden, with high fever and pulmonary symptoms which are so severe as to mask everything else until the rash makes its appearance, the case up to that time being often regarded as one of primary pneumonia or of grippe. The duration of the stage of invasion—i. e., from the beginning of the catarrh until the eruption—in 270 cases which we have analyzed was as follows:

1 day or less	35 cases.	6 days	20 cases.
2 days	47 "	7 "	6 "
3 "	64 "	8 "	2 "
4 "	64 "	9 "	2 "
5 "	29 "	10 "	1 case.

From this table it will be seen that the length of the period of invasion varies considerably—more, we think, in infants and very young children (most of these were under three years old) than in those who are older. In the greater number of cases it lasts from two to four days.

Eruption.—The rash usually appears on the third, fourth, or fifth day of the disease—in the largest number upon the fourth day. As a rule, it is first seen on the back or behind the ears, on the neck, or at the roots of the hair over the forehead. It appears as small, dark-red spots, which are at first few, scattered, and not elevated, resembling flea-bites. In twenty-four hours the macules are much more numerous, and many of them have become papules. They frequently group themselves in crescentic forms. They are usually separated by areas of normal skin, but where the rash is intense they are frequently coalescent. From the time of its first appearance to the full development of the rash on the face, is usually about thirty-six hours, but may be from one to three days. With a full eruption there is seen considerable swelling of the face, especially about the eyes, and the features are sometimes scarcely recognizable. On the second day of the rash it begins to appear upon the neck beneath the chin, the upper part of the chest and back; on the third day the trunk is covered, and scattered spots are seen upon the extremities. The rash appears last upon the lower extremities, and by the time it is fully out upon them it has usually begun to fade from the face. In mild cases it remains discrete, but in severe ones it is

frequently confluent upon the face and upon the extensor surfaces of the extremities. As a rule, it covers the entire body, even the palms and soles.

The eruption fades slowly in the order of its appearance, and there is left behind, in typical cases, a slight brownish staining of the skin which often remains for a week or more. The duration of the rash is from one to six days, the average being four days.

There are many cases in which the rash does not follow the typical course described: (1) Instead of spreading gradually, the entire body may be covered in a few hours. (2) The rash may be hemorrhagic. This condition was present in about five per cent of our cases. The whole eruption may be hemorrhagic, or it may be so only upon certain parts—usually the abdomen or extremities. In such circumstances small petechial spots take the place of the macules—the "black measles" of the older writers. It is in most cases a bad, but by no means a fatal symptom. We have seen it in several cases that were not especially severe. (3) The rash may be very faint, and of short duration, being scarcely elevated at all. (4) It may consist of very minute papules, closely resembling the rash of scarlet fever. It is to be remembered, however, that the irregular eruptions of scarlet fever much more frequently resemble measles than *vice versa*. (5) It may be very scanty, and late in its appearance; particularly in cases of great severity and hyperpyrexia—the so-called malignant cases. (6) Temporary recession of the eruption may occur at any time during the height of the disease, and is usually due to heart failure. A recurrence of the eruption after it has run its usual course is something which we have never seen; although such cases have been reported, they must be regarded as very exceptional.

During the first two days of the eruption, the local and constitutional symptoms increase in severity, both usually reaching their maximum at the time of the full development of the rash upon the face. The skin is swollen, and the seat of intense itching and burning. The eyes are very red and sensitive to light, and there is swelling of the conjunctivae with an abundant production of mucus or muco-pus, causing the lids to adhere. There is pain on swallowing, also swelling of the glands at the angle of the jaw or in the postcervical region. The cough is frequent and very annoying. There is complete anorexia, and often diarrhea. The tongue is coated, and may show at its margin enlarged papillae, somewhat resembling the "strawberry" appearance of scarlet fever. As the rash fades the temperature declines rapidly, often reaching the normal in two or three days. The catarrhal symptoms now subside, and soon the patient is convalescent. Within a day or two after the fever has ceased, the rash disappears.

Desquamation.—This begins almost as soon as the rash has subsided, and is first noticed on the face and neck, where the eruption first appeared. The nature of the desquamation is invariably fine, branny scales, never in large patches, as in scarlet fever. It is often quite indistinct and may be overlooked. Its usual duration is from five to ten days. It may, however, be prolonged for two weeks. The amount of desquamation varies considerably in the different cases. It is most marked in those in which there has been an intense eruption. There is frequently noticed at this time an odor about the patient which is quite characteristic of measles. During this stage the cough often persists and the eyes remain weak and very sensitive to light, but in other respects the patient usually feels perfectly well.

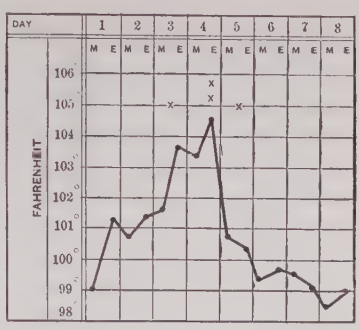


Fig. 158.—Temperature Curve in Uncomplicated Measles, Showing the Gradual Rise and Critical Fall. Patient ten years old; × = first eruption; ⨯ = full eruption on the face.

1. *The Mild Cases.*—The mildest cases are distinguished by low temperature, which at the height of the eruption usually reaches 102° or 103° F., but rarely lasts more than four days. The eruption is often scanty, and is never confluent. The swelling, itching, and other cutaneous symptoms are wanting, as is also the intense red color of the skin. The rash is frequently obscure, and, without the other symptoms, hardly sufficient for diagnosis. The catarrhal symptoms are more uniform than the rash, but these are very mild as compared with the usual form. The duration of the rash is shorter, desquamation is scarcely perceptible, and there are no complications.

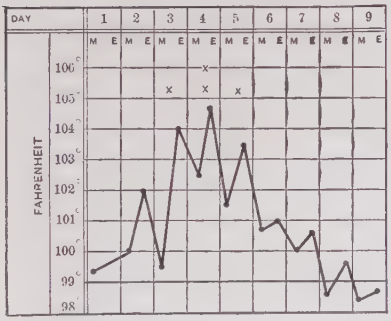


Fig. 159.—Typical Curve in Uncomplicated Measles, with Gradual Rise and Gradual Fall. Patient three years old.

2. *The Cases of Moderate Severity.*—The course of measles is much more regular in children over three years old than in infancy. In the former, the symptoms of invasion come on gradually, and the temperature rises steadily until the appearance of the eruption, which is in most cases on the third or fourth day of the disease. Figs. 158 and 159 represent the typical temperature curve in average uncomplicated cases. Such a curve was seen in 44 per cent of 173 cases in which careful

observations were made. Sometimes the decline in the fever is very rapid, almost a crisis, as in Fig. 158, but more often it falls gradually, as in Fig. 159. In such cases the duration of the fever is from five to nine days, the average being about a week. The other symptoms follow very closely the course of the fever. The maximum temperature is nearly always coincident with the full rash upon the face, at this time usually being in uncomplicated cases from 103° to 104° F. in older children, and 104° to 105° F. in infants and young children.

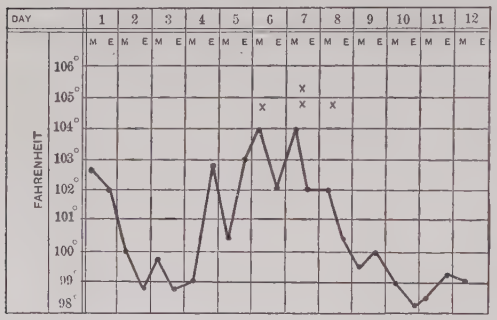


FIG. 160.—A NOT INFREQUENT TEMPERATURE CURVE IN MEASLES, SHOWING ABRUPT INVASION, BUT SUBSEQUENT COURSE TYPICAL. Uncomplicated case; patient nine months old.

A not very uncommon temperature curve is that of Fig. 160, where the onset of the disease is marked by a sudden rise to 102° or even 104° F., with a fall nearly or quite to normal on the second day, after which the fever rises gradually, as in the first group. This curve was seen in five per cent of our cases.

3. *The Severe Cases.*—In Fig. 161 is shown a type of the disease which is more frequent in infants than in older children, the important features being the late eruption and the continuance of the high fever for several days after the rash has begun to fade. Such a prolonged

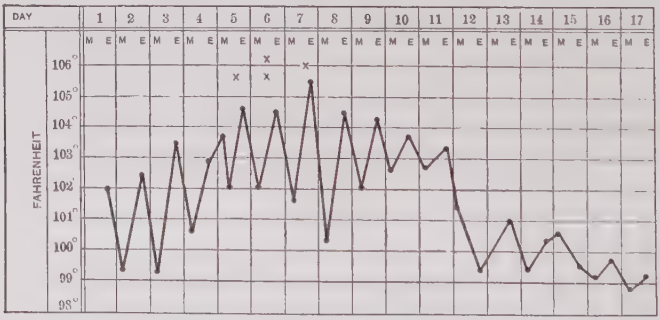


FIG. 161.—MEASLES WITH PROLONGED INVASION. Continuance of high temperature after full eruption due to severe bronchitis and diarrhea; child two years old.

course and so high a temperature are almost invariably due to some complication, usually bronchopneumonia. When the pneumonia goes on to the production of areas of consolidation, the fever usually continues for three and sometimes for four weeks, even though terminating in recovery.

Figs. 162 and 163 illustrate two types of the disease which are often seen when measles is complicated by pneumonia. In cases like that shown in Fig. 162 the onset is abrupt with high temperature, prostration, and pulmonary symptoms not unlike those of primary pneumonia. A temperature curve resembling this was seen in 28 of 173 cases. The rash is often late in appearance; it is faint and altogether irregular; it may recede after the first day and reappear after an interval of one or two days. The catarrhal symptoms are not marked, but the whole force of the disease seems to be expended upon the lungs. The diagnosis of these cases presents great difficulties, and very often it would not be made but for the fact that there are other cases of measles in the family or the institution. This form is usually seen in infants, and it is usually fatal.

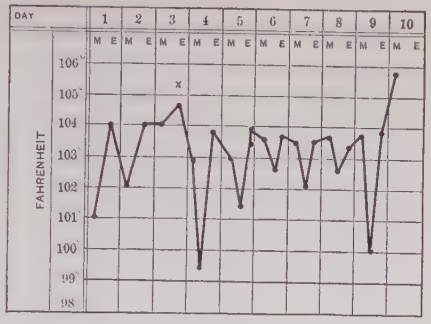


Fig. 162.—Fatal Attack of Measles, Complicated by Bronchopneumonia. Very severe symptoms from the onset; patient eighteen months old; death on tenth day.

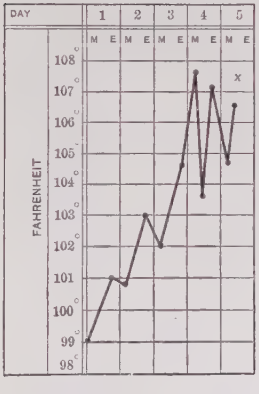


Fig. 163.—Fatal Attack of Measles Complicated by Bronchopneumonia. Early invasion mild, but rapid development of severe symptoms on fourth day; rash on last day; patient eight months old.

In other cases marked by a sudden severe onset, the system seems to be overpowered by the poison of the disease itself. There is profound depression, and hyperpyrexia, and the patient may die from toxemia with cerebral symptoms before the appearance of the rash or just as it is beginning to show itself. Sometimes the pulmonary symptoms are entirely wanting; at others the rash, if it appears, is hemorrhagic.

In still another group of cases the onset is not violent, and for the first two days the attack may appear to be of only average severity; but there may then develop, often quite suddenly, pulmonary symptoms of such intensity as to cause death within twenty-four hours. The eruption, if seen at all, is faint and not characteristic (Fig. 163).

A secondary rise in the temperature after it has once fallen to normal was seen in 8 of 173 cases, being due to the development of otitis, ileocolitis, or pneumonia.

Complications and Sequelae.—The most frequent and most important

complication of measles is bronchopneumonia, and next to this are ileocolitis, otitis, and membranous laryngitis. Most of the others are infrequent; all complications are relatively infrequent in children over four years old.

Lungs.—The greatest danger in measles arises from pulmonary complications, and the frequency is greatest in children under two years of age. In two institution epidemics, embracing about 300 cases, nearly all in children under three years old, bronchopneumonia occurred in about 40 per cent of the cases. Of those who had pneumonia, 70 per cent died. Fortunately, such a record as this is never seen outside of institutions for young children. Of 2,477 cases, embracing several epidemics of measles among children of all ages, pneumonia occurred in 10 per cent. Our own experience in the post-mortem room fully bears out the statement of Henoch, that a certain amount of pneumonia is found in almost every fatal case. Pneumonia is more frequent and its mortality is higher in spring and winter epidemics than in those occurring at other seasons. It may develop at any time from the beginning of invasion until convalescence, but it most frequently begins about the time of full eruption.

Lobar pneumonia, although rare, occasionally occurs as a complication in children over three years old. In some epidemics many of the cases of pneumonia are complicated by severe pleurisy, which adds much to the danger from the disease. This form is frequently followed by empyema. Pneumonia is always to be suspected when the temperature continues high after the full appearance of the rash.

Bronchitis of the large tubes, always accompanied by tracheitis, is seen in every case of measles, possibly excepting a few of the very mildest. This is so constant a feature as hardly to be ranked as a complication. In nearly all of the severe cases the bronchitis extends to the medium-sized and smaller tubes.

Larynx.—A mild catarrhal laryngitis accompanies almost every case of measles. Severe catarrhal laryngitis is present in about ten per cent of the cases; it may give symptoms which closely resemble those of membranous laryngitis, and the two are no doubt often confused.

Membranous laryngitis is especially seen in the epidemics of institutions. As a cause of death in older children it ranks next to pneumonia. When it develops at the height of the disease, it is sometimes due to the streptococcus; but when it develops at a later period, it is usually due to the diphtheria bacillus. The streptococcus inflammation is in most cases associated with similar changes in the pharynx or tonsils, but not always. True diphtheria, occurring as a complication of measles, not infrequently begins in the larynx. The streptococcus inflammation may be as serious in this connection as is true diphtheria.

from the probability, which amounts almost to a certainty, of the development of bronchopneumonia. No complication is more to be dreaded than this. The diagnosis between the two forms may sometimes be made by the time of development, but only with certainty by cultures. We once saw in measles, where no false membrane was present in the rest of the larynx, a necrotic inflammation with almost entire destruction of the vocal cords—a condition which may be compared to that seen in the tonsils or epiglottis in scarlatina.

Throat.—A catarrhal angina is part of the disease, and is as characteristic of measles as is the eruption upon the skin. There is acute congestion and swelling of the tonsils, uvula, palate, and pharynx. In a certain proportion of cases, very much less frequently than in scarlatina, the development of membranous patches is seen upon the tonsils and adjacent mucous membranes. These occur in two or three per cent of the cases. They are to be regarded in the same light as similar conditions complicating scarlet fever, with this difference, that in measles there is much greater likelihood of the extension of the disease to the larynx, while extension to the nose and ears is much less probable. True diphtheria, however, may complicate measles, and cases of membranous inflammation of the tonsils or pharynx developing late in measles are usually due to the Klebs-Loeffler bacillus.

Although in most cases the inflammations of the pharynx and tonsils which accompany measles are not serious when they are due to the streptococcus, they are sometimes quite as severe as any that accompany scarlet fever. They may cause death from general sepsis apart from any affection of the larynx.

Digestive System.—Gastric disorders are not more common than in other febrile diseases; but diarrhea is very frequent, and in summer it may be even more serious than the pulmonary complications. All forms of diarrhea are seen, from that which results from simple indigestion to the severe types of ileocolitis. This complication is most often seen in children under two years old. The most severe intestinal symptoms are not usually seen at the height of the primary fever; but, beginning at this time, they often increase in severity, and are most marked in the second and third weeks of the disease.

Catarrhal stomatitis is present in almost every case of measles; less frequently the herpetic form is seen. Ulcerative stomatitis is not uncommon, particularly in institutions. One of the worst complications of measles, but fortunately a rare one, is gangrenous stomatitis, or noma. This usually occurs in inmates of institutions, or in children with bad surroundings who were previously in wretched condition. It is nearly always fatal.

Gangrenous inflammations of other parts of the body are some-

times seen after measles, especially of the ear, the vulva, or the prepuce.

Nervous System.—Convulsions are seldom seen at the onset of measles. During the progress of the disease they are not so rare, and may occur in connection with otitis, meningitis, or severe bronchopneumonia—chiefly in infants.

Meningitis is rare, but either the simple or the tuberculous form may occur, more often, however, as a sequel than as a complication. Mental disturbance, usually of a temporary character, occasionally follows measles. In the epidemic of 108 cases reported by Smith and Dabney, insanity was noted three times, all the cases terminating in recovery. Epilepsy and chorea are rare sequelae.

Ears.—Otitis is a frequent complication in some epidemics; in others it is seldom seen. In one hospital epidemic it was noted in 14 per cent of the cases. This epidemic occurred in early spring and affected very young children, both of which circumstances are favorable for the development of otitis. Usually both ears are affected, but the otitis of measles is, as a rule, less serious than that of scarlet fever.

Eyes.—Simple catarrhal conjunctivitis accompanies nearly every case of measles. In the severe form there is a mucopurulent catarrh, which may attain any degree of severity. In neglected cases, and among children who are poorly nourished, especially in asylums, the disease is apt to extend to the cornea. Chronic conjunctivitis often persists after measles, particularly in the class of children just mentioned.

Lymph Nodes.—Swelling of the lymphatic glands of the neck is frequent, but not generally severe, and rarely terminates in suppuration. Chronic enlargement may continue for months, and sometimes the glands may become tuberculous. Similar changes and similar consequences may occur in the glands of the tracheobronchial group.

Kidneys.—The infrequency of renal complications in measles is in striking contrast to scarlet fever. Transient febrile albuminuria is not uncommon, but a serious degree of nephritis, either clinically or at autopsy, we have never seen, and literature furnishes but few cases.

Heart.—Both endocarditis and pericarditis have occurred in the course of measles, but they belong to the rare complications. The same may be said of changes in the muscular walls of the heart.

Skin.—As complications, erysipelas, furunculosis, impetigo, and pemphigus have been noted; but all are rare.

Hemorrhages.—Associated with the hemorrhagic type of the eruption, severe and even fatal hemorrhages may occur from the mucous membranes, and the latter are sometimes seen without the hemorrhagic eruption.

Blood.—In cases which have been studied early in the stage of incubation a lymphocytic leucocytosis has been observed. This is succeeded by a

leucopenia in which there is a reduction in the lymphocytes both actual and relative. This condition is marked one or two days before the eruption—sometimes even earlier—and continues during the height of the disease. A decided leucocytosis during this time or later points to a complication.

Other Infectious Diseases.—Measles in institutions is often complicated by diphtheria. Scarlet fever or varicella occasionally occurs during measles, though it is rare that the two eruptions are exactly simultaneous. Epidemics of measles and whooping-cough frequently occur together or follow each other. The relation of measles to tuberculosis seems to be particularly close. In some cases general or pulmonary tuberculosis follows directly in the wake of measles, which seems to furnish, especially in the lungs, conditions which are favorable for the development of latent tuberculosis. As a late manifestation the most common one is tuberculosis of the bones, occurring as hip-joint disease, caries of the spine, etc. An attack of measles in a child with latent tuberculous antecedents should, therefore, always be looked upon with apprehension.

Diagnosis.—A sign of the greatest diagnostic value is the buccal eruption. Although it appears that this was described many years ago by Flindt, of Denmark, it is to Koplik that the credit belongs for its independent discovery and for the appreciation of its diagnostic significance. The unit of the eruption is a bluish-white speck upon a red ground; only a few of these are present for the first twenty-four or thirty-six hours; after this the mucous membrane may be fairly peppered with them. Often they are not seen except by careful search, for which strong sunlight is necessary; artificial light is not satisfactory. The spots are best seen on the inside of the cheeks opposite the molar teeth, and in most cases only there; but they may be present on almost any part of the buccal mucous membrane. Their diagnostic value is due to the fact that they are nearly always present, that they are not found in other diseases, and that they usually appear two or three days before the skin eruption. This generally disappears at the time of full eruption.

We have records of an epidemic of 187 cases in an institution in which careful notes were made regarding this buccal eruption: it was unmistakably present in 169 cases, absent in 8, doubtful in 10. In 78 cases, fever, rash, and Koplik's sign were all present at the first observation. In 54 patients the sign was noted one day before the rash; in 25, two days before; in 4, three days before; in 3, four days before; and in 2, five days before. In 2 the spots were not seen until after the skin eruption; in one case they were present without any eruption. As this patient had been exposed and had a prolonged fever, it seems fair

to regard the case as one of measles. In only one case was the buccal eruption seen before any elevation of temperature.

These facts, amply confirmed by other observations, indicate that Koplik's sign is of value in enabling us to make a diagnosis from one to three days before it is possible by the skin eruption, also in furnishing a means of distinguishing measles from the other eruptive fevers, as well as from rashes due to drugs, antitoxin, etc.

Other important symptoms are the coryza, the gradual rise in temperature, and the eruption which appears first upon the neck and face, and slowly extends over the body. Cases which present the greatest difficulties in diagnosis are usually the very severe ones and those in infants.

Prognosis.—This depends upon the age and previous condition of the patient, the character of the epidemic, and the season of the year. Except in children under three years of age, the deaths from measles are few; but in institutions containing young children, no epidemic disease is so fatal.

The general mortality of the disease is from 4 to 6 per cent; but in epidemics in institutions for young children it has, in our experience, ranged from 15 to 35 per cent. The following table gives the figures of an epidemic in one institution:

From six to twelve months	42 cases;	mortality,	33 per cent.
" one to two years	51 "	"	50 " "
" two to three years	27 "	"	30 " "
" three to four years	20 "	"	14 " "
" four to five years	3 "	"	0 " "

In any single case the important symptoms for prognosis are the temperature and the character of the eruption. An initial temperature above 103° F., or one which remains high until the eruption appears, is a bad symptom. So also is one which rises after a full eruption, or which does not fall as the rash fades. The following table shows the highest temperature and mortality in 161 hospital cases:

Highest temperature not over 102° F.	6 cases;	mortality,	0 per cent.
" " 102° to 103.5° F.	14 "	"	7 " "
" " 104° " 104.5° F.	49 "	"	16 " "
" " 105° " 105.5° F.	65 "	"	40 " "
" " 106° F. or over..	27 "	"	80 " "

A favorable eruption is one of a bright color, covering the body, remaining discrete, and spreading gradually. It is unfavorable for the eruption to appear late, to be very faint, scanty, or hemorrhagic, or to recede suddenly, as this is usually due to a weak heart.

Of 51 fatal cases, the cause of death was bronchopneumonia in 45, ileocolitis in 4, and membranous laryngitis in 2. More than half the

deaths occurred during the second week, the earliest being upon the fifth day of the disease.

The ultimate result of an attack of measles may not be evident for some time. Cases in which the temperature persists for two or three weeks without assignable cause after the disease is apparently over, should be watched with the greatest solicitude. The explanation of this is most frequently to be found in the lungs, although the physical signs are often obscure. The condition may be either pneumonia or pulmonary tuberculosis. Even though the attack of measles may not have been in itself severe, seeds are often sown the full fruits of which are not seen until long afterward. Chronic glandular enlargements which may or may not be tuberculous, chronic bronchitis, chronic laryngitis, subacute or chronic nasal catarrh, hypertrophy of the tonsils, and adenoid growths of the pharynx—all are frequent sequelae.

Prophylaxis.—Measles is often regarded by the laity as so mild a disease that its prevention is thought to be of little importance, and no effort is made to limit its extension. The great probability that every person at some time in his life will have the disease, is no justification of unnecessary exposure. Although in older children measles is usually mild, this is not so in infants, who should be carefully protected from exposure. Special care should also be taken to avoid the exposure of delicate children or those with a strong tendency to pulmonary disease or to tuberculosis. In institutions it is of the utmost importance to secure prompt and complete isolation of the first case which appears.

The disease being nearly always spread by the patient, it follows that while early isolation is more important, there is not required the same thorough disinfection of apartments which should follow every case of scarlet fever. In an institution, the ward or cottage from which a case has been removed should be quarantined for at least sixteen days after the appearance of the last case, and absolute security can not be said to exist until the end of three weeks. The same rule should be applied in private families where children who have been exposed should be quarantined apart from the patient, but not sent away. In ordinary circumstances the quarantine of a case of measles should be placed at two and a half weeks, or ten days from the beginning of the eruption. It should be continued longer if there is otitis, or a nasal discharge.

Thorough cleansing and disinfection of the sick-room should be done before it is again occupied by children, and it should remain vacant at least two weeks. Children should be kept from all schools while the disease is in their homes, chiefly because they are otherwise liable to spread the disease while suffering from the early symptoms of invasion.

Treatment.—Measles is a self-limited disease, and there are no known

measures by which it can be aborted, its course shortened, or its severity lessened. The indications are therefore to treat serious symptoms as they arise, and, as far as possible, to prevent complications, which are the principal cause of death.

While the bed should be screened to protect the sensitive eyes of the patient it is not desirable to exclude sunlight from the sick-room. Every child with measles should be put to bed and kept there with light covering during the entire febrile period. There can be no possible advantage in causing a child to swelter by thick covering, under the delusion that the disease may be modified thereby. The food should be light, fluid, and given at regular intervals. If the conjunctivitis is severe, iced cloths should be applied to the eyes, which should be kept clean by the frequent use of a solution of boric acid, the lids being prevented from adhering by the application of vaseline or some simple ointment. The intense itching and burning of the skin may be relieved by inunctions of plain or carbolized vaseline, or by bathing with a solution of bicarbonate of soda. The cough, when distressing, may be allayed by small doses of opium, either in the form of codein or the brown mixture. The restlessness, headache, and the general discomfort which accompany the height of the fever may be relieved by an occasional dose of phenacetin. As soon as the rash has subsided, a daily warm bath should be given, followed by inunctions to facilitate desquamation.

The important indications to be met in the severe cases are very high temperature, cardiac depression, and nervous symptoms—dulness, stupor, sometimes coma, or convulsions. In some of the cases there is in addition dyspnea and cyanosis, showing severe acute pulmonary congestion. For the nervous symptoms and high temperature, nothing is so reliable as the cold bath or pack and the nearly continuous use of ice to the head. We do not think there is any evidence that the use of cold increases the liability to pneumonia; but cold extremities, feeble pulse, and cyanosis, when associated with high temperature, call for the hot mustard bath, although ice should still be applied to the head. The indications for stimulants and the methods of using them are the same as in bronchopneumonia, which is usually present in cases requiring them.

To diminish the chances of pneumonia, it is necessary that every patient should be kept in bed during the attack, and care exercised to avoid exposure. But still more important is it in hospitals and institutions where most of the cases of pneumonia occur, to allow the patients plenty of air space, never crowding them together in small wards. If possible, cases complicated by pneumonia should be separated from simple cases. The pneumococcus and the streptococcus are found in the mouth in such numbers that systematic disinfection of the mouth may prove of some value.

The danger of diphtheria as a complication may be greatly lessened if during epidemics of measles in institutions every case receives an immunizing dose of diphtheria antitoxin.

The bronchitis and bronchopneumonia of measles should be managed as when they occur as primary diseases, since the coexistence of measles furnishes no new indications. The same is true of the diarrhea, conjunctivitis, otitis, membranous laryngitis, pharyngitis, and tonsillitis. Should cultures show the presence of the diphtheria bacillus, the case should be treated like one of diphtheria.

During convalescence the eyes should be used very carefully for at least several weeks. Should the cough and slight fever persist, with or without physical signs in the chest, the patient should, if possible, be sent away to a warm, dry, elevated district, as the development of tuberculosis is always to be feared. Cod-liver oil should be given continuously throughout the succeeding cool-season, and iron and other tonics according to indications. The cough itself should be treated as when it follows an ordinary bronchitis, creosote being more generally useful than any other drug.

CHAPTER III

RUBELLA

(*German Measles; Rötheln*)

Rubella is a contagious eruptive fever which is rarely seen except when prevailing epidemically. It is characterized by a short invasion, with mild, indefinite symptoms, usually lasting but a few hours, and by an eruption which is generally well marked but of variable appearance. The constitutional symptoms are very mild, and the disease rarely proves fatal, not often being even serious. For a long time rubella was confounded with measles and scarlet fever, as the eruption sometimes resembles one and sometimes the other disease. Its identity is now fully established, and, as Strümpell well says, its existence is doubted only by those who have never seen it.

Rubella is not a simple affection of the skin; it prevails independently either of measles or of scarlet fever; its incubation, eruption, invasion, and symptoms differ materially from those of both these diseases; it attacks indiscriminately and with equal severity those who have had measles and scarlet fever and those who have not, nor does it protect in any degree against either of them; it never produces anything but

rubella in those exposed to its contagion; it occurs but once in the same individual.

Etiology.—Rubella is beyond question contagious, but is decidedly less so than either measles or scarlet fever; so that some observers have doubted its contagion altogether. It can be communicated at any time during its course, but is especially contagious during the early stage. Epidemics usually prevail in the winter or spring. As in the other eruptive fevers, a striking immunity is seen in infants under six months old; but, with this exception, all ages are liable to the disease.

The incubation of rubella varies considerably; the usual period is from fourteen to twenty-one days, although the limits are from ten to twenty-two days.

Symptoms.—*Invasion.*—This is rarely more than half a day, and in many cases no prodromata whatever are noticed, the rash being the first thing to attract attention. In a few cases there are mild catarrhal symptoms, with general malaise and slight fever. At other times there may be vomiting, convulsions, delirium, epistaxis, rigors, headache, or dizziness; but all are to be regarded as very exceptional.

Eruption.—Frequently a child wakes in the morning covered with the rash, no symptoms having been previously noticed. It generally appears first upon the face, and spreads rapidly to the whole body, the lower extremities being last covered. Less than a day is usually required for its full development. Exceptionally the eruption comes first upon the chest and back, and sometimes nearly the whole body is covered almost at once. The rash is occasionally observed in the roof of the mouth before it is visible on the face. In a considerable number of cases the entire body is not covered; but the rash is more constantly seen upon the face than upon any other part of the body.

Its character is subject to considerable variation. The eruption is most frequently composed of very small maculopapules; they are of a pale-red color, and vary in size from a pin's head to a pea. The spots are usually discrete, but may cover the greater part of the body. On the face it is frequently confluent, and often appears here as large, irregular blotches of a red color. From this description the rash will be seen to resemble that of measles more than that of any other disease. Very often, however, there is a fairly uniform red blush which bears a close resemblance to the rash of scarlet fever; but even in such cases there will nearly always be found upon some part of the body, usually the wrists, fingers, or forehead, some typical maculopapules. Between these two extremes all variations are seen. The color of the eruption is sometimes dark red, and rarely it has been noted to be hemorrhagic. The degree of elevation above the surface is also variable; sometimes this is so marked as to give to the skin a "shotty" feel, while in others the

elevation is scarcely perceptible. The duration of the eruption is usually three days. Occasionally it lasts only two days, and it may last but one; it is rare for it to remain as long as four days. It fades in the order of its appearance, and more rapidly than the eruption of measles. A slight brown pigmentation of the skin sometimes remains for a few days after the rash.

The highest temperature is coincident with the full eruption; this does not usually exceed 101°, and often it is only 100° F. As a rule, the temperature continues but two days, falling as the eruption fades. Very often the fall to normal is abrupt. Rarely more severe cases are seen in which the fever lasts for two or three days, being 101° or 102° F. during the invasion, and rising to 103° F. or more during the full eruption. The other symptoms are in most cases even less marked than the fever. Occasionally catarrhal symptoms resembling a mild attack of measles are present, or a sore throat suggesting mild scarlet fever; but more frequently all these symptoms are absent. The eruption is usually out of all proportion to the other signs of disease.

Swelling of the post-cervical glands is one of the most constant features of rubella. In most epidemics it is seen in nearly all cases; but as a symptom for differential diagnosis it is not of great importance, as it is not uncommon in measles and scarlet fever. The glandular swelling is most marked at the height of the disease; it is never very great, and subsides slowly without suppuration. Vomiting and diarrhea are rare. Swelling and itching of the skin are usually present and sometimes marked. There is no leucocytosis in this disease.

Forchheimer has described an eruption on the mucous membrane of the throat, or "enanthem," which he believes to be characteristic. It consists of minute, bright, rosy-red points, seen on the uvula and soft palate, rarely on the hard palate. It is present only during the first twenty-four hours.

Desquamation.—This is exceedingly variable. It is sometimes entirely wanting; writers who have observed some fairly typical epidemics have stated that it did not occur. In most cases, however, some desquamation is present, though it may be so slight as to be discovered only by a close examination. It is usually in the form of fine scales over the body and extremities. In a few cases it is more pronounced, and may be in larger flakes or patches.

Prognosis.—There are few diseases so free from danger as rubella. Complications and sequelae are very seldom seen, and when present are usually of the mildest character.

Diagnosis.—The principal interest attaching to rubella is in its diagnosis. This is a matter of extreme difficulty, and often it is an impossibility. The characteristic thing about the disease is a well-marked erup-

tion with very few other symptoms. Cases so closely resemble mild scarlet fever that the differentiation by symptoms may be impossible; it must be made by the circumstances in which the disease occurs, especially a prevailing epidemic. Scarlet fever with a low temperature and abundant rash should always be regarded with suspicion; also an abundant rash with little or no desquamation. The longer period of incubation in rubella is often of much assistance. Koplik's sign furnishes a valuable means of distinguishing measles from rubella. The difficulties in diagnosis can be appreciated only by one who has seen epidemics of measles and scarlet fever in institutions, and has watched the exceedingly mild course of undoubted cases of these diseases which have there occurred.

It is always hazardous to make the diagnosis of rubella unless the disease is prevailing epidemically. Sporadic cases in which this diagnosis is made are, we believe, almost invariably instances of mild measles or scarlet fever. The first cases of rubella in an epidemic are usually overlooked. The continued absence in succeeding cases of the characteristic symptoms and complications of measles or scarlet fever should suggest to the physician that he is probably dealing with rubella.

Treatment.—None whatever is required for the disease excepting isolation, which should be complete until the diagnosis is positively determined; after this it is hardly necessary. The individual symptoms and complications are to be treated as they arise.

CHAPTER IV

VARICELLA

(*Chicken-pox*)

Varicella is an acute, contagious disease, characterized by a cutaneous eruption of papules and vesicles and by mild constitutional symptoms, serious complications and sequelae being very rare. Although long confounded with varioloid, its existence as a distinct disease has been generally admitted for many years.

Etiology.—It is well established that the contagium of the disease is contained in the vesicles, as it may be communicated by inoculation with their contents. The specific poison, however, has not yet been isolated. Varicella is contracted by exposure to another case or through the medium of a third person. It affects children of all ages, one attack being as a rule protective. It is very contagious, resembling measles in this

respect. The period of incubation is quite uniformly from fourteen to sixteen days.

Symptoms.—Slight fever and general indisposition may be noticed for twenty-four hours before the appearance of the eruption, but in most cases the eruption is the first symptom. It usually appears first upon the face or trunk, as small, red, widely scattered papules. The papules in most cases come in crops, new ones continuing to appear for three or four days, even upon the same part of the body. The earlier ones have generally begun to dry up by the time the later ones appear, so that all stages of the eruption may be present at one time in the same region, this being one of the diagnostic features. The papules are at first very small, but gradually increase in size, and are surrounded by an areola from one-fourth to half an inch in width. Many of them go no further than this stage, but the majority become vesicular. The vesicles are usually flat, and vary a good deal in size—the largest being about one-fourth of an inch in diameter. The process of drying up generally begins at the center; this causes a slight depression, giving the vesicle a somewhat umbilicated appearance. The areola is most distinct at the time of the fully formed vesicle, and fades as the latter dries. Crusts now form, which fall off in from five to twenty days, depending upon the depth to which the skin has been involved. In the majority of cases no mark is left, but after the most severe attacks, when the true skin has been involved, scars remain, and occasionally there is quite deep pitting. Such marks are few in number, and are most likely to occur upon the face.

Sometimes, especially upon hands and feet, the vesicle appears without having been preceded by a papule; often there is no areola, and the vesicle resembles a drop of water upon healthy skin. In most cases pustules are not seen, but they may develop in consequence of irritation or infection, the result of scratching, or in children who are poorly nourished. Under these circumstances deeper ulceration may occur, lasting for weeks. In rare cases there may be a necrotic inflammation about the site of the pock, a condition to which is sometimes given the name *varicella gangrenosa*. It is not peculiar to varicella, and is described elsewhere under the head of Gangrenous Dermatitis.

The pocks are usually most abundant over the back and shoulders. In mild cases only twenty or thirty may be found upon the entire body, but in severe cases the skin in certain regions may be nearly covered. The eruption is never confluent. The pocks are usually seen on the hairy scalp, and often on the mucous membrane of the mouth or pharynx —a point of some diagnostic value. In the latter situation the appearance is first as a tiny vesicle, and later as a superficial ulcer resembling that of herpetic stomatitis. Marfan and Halle have described cases of varicella of the larynx. Croupy symptoms were present, and in one

case which proved fatal from pneumonia a tiny ulcer was found on the vocal cords.

The temperature is highest when the eruption is most rapidly appearing, this usually being the second or third day. In an average case it reaches only 101° or 102° F., and lasts but two days; in severe cases it may rise to 104° or 105° F., and lasts for four or five days. It falls gradually to normal as the rash fades. The other symptoms are mild and not characteristic.

Complications.—The most important complication is erysipelas, which develops about the pocks, particularly when they are deep and attended with some ulceration. We have known of several fatal cases from this cause. Adenitis, either simple or suppurative, and abscesses in the cellular tissue, are occasionally seen. Nephritis is very infrequent, but a number of cases are recorded. It may occur at the height of the disease, but more often at a later period, like the nephritis of scarlet fever. Varicella is quite frequently complicated by other infectious diseases. We have seen coincident scarlet fever in a number of cases. Severe nervous lesions occasionally follow varicella, the most frequent being encephalitis. We have seen transverse myelitis develop in a boy of seven after an attack of varicella.

Diagnosis.—The diagnosis of varicella is usually easy, provided the following points are kept in mind: first, that the eruption comes out slowly and in crops, so that papules, vesicles, and crusts may be seen upon the skin in close proximity; secondly, that the umbilication is due only to the mode of drying up of the vesicle, which begins at the center; thirdly, the appearance of the pocks upon the mucous membranes, and the history of exposure. It is distinguished from urticaria and other forms of skin disease by the presence of fever and often by the lesions in the mouth. Cutaneous inoculations from fresh vesicles, as first practiced by Kling, apparently protect against varicella. At the site of inoculation small localized lesions are produced, but there are no general symptoms.

Treatment.—Although it is usually a trivial disease, isolation of cases of varicella should be enforced in schools and in institutions containing many infants. In the home, unless other children are delicate or in poor condition, quarantine is unnecessary. The disease may probably be conveyed as long as the crusts are present, hence isolation should be maintained until they have fallen off. In most cases constitutional symptoms of the disease are so mild as to require no treatment.

Locally, the itching, when annoying, may be allayed by sponging with a solution of bicarbonate of soda, a one-per-cent solution of carbolic acid or the use of carbolized vaseline. When the crusts have formed, this ointment or vaseline containing two per cent ichthyol should be

applied. Care is necessary to keep the skin clean, and, in the case of infants, to prevent scratching. In severe cases the urine should invariably be examined.

CHAPTER V

VACCINIA—VACCINATION

Vaccinia (cowpox) is a febrile disease induced in man by inoculation with the virus obtained either directly from the cow (bovine virus) or from a person who has been inoculated (humanized virus). The disease is not contagious in the ordinary sense of the term, but is communicated by inoculation either accidental or intentional.

The protection against smallpox which vaccination affords is one of the best attested facts in medicine. Its effect when systematically practiced is graphically shown in the accompanying chart (Fig. 164). It is the imperative duty of the physician to see to it that every young infant is vaccinated.

Re-vaccination.—Regarding the duration of the protective power of a single vaccination, positive statements are impossible. Nearly all writers are agreed that vaccination should be done in infancy, again at puberty, and a third time at about the age of twenty or twenty-five. Many also insist upon re-vaccination at about the seventh year. It is a safe rule when smallpox is prevalent to vaccinate every person who has not been successfully vaccinated within five years.

Choice of Lymph.—The substitution of bovine for humanized virus is now well-nigh universal. It has precluded the possibility of transmitting syphilis and greatly lessened the chances of other forms of infection. A still further advance was made by the introduction of "glycerinated" lymph. As now prepared, the lymph is taken from the calves under the most rigid aseptic precautions and emulsified with glycerin. The few saprophytic bacteria present soon die, so that when properly prepared the glycerinated virus is practically sterile. It should not be distributed until it has been carefully tested for pathogenic organisms of all kinds, particularly the tetanus bacillus. It is preserved and distributed in capillary tubes hermetically sealed; these are much safer than quills or ivory points, which may easily become contaminated by handling. After the lymph has been taken, the calves are killed in order to make certain that they were free from disease. The practical advantages of glycerinated lymph are so great that it has been officially adopted by the Governments of the United States, Great Britain, Germany, and many other countries.

Noguchi has succeeded in cultivating vaccine virus *in vitro*. It can readily be produced in indefinite quantities; so that we may soon hope to be supplied with virus in pure culture, free from all possibility of bacterial contamination from animal sources. Experience with its use indicates that it is quite as effective as the ordinary bovine virus.

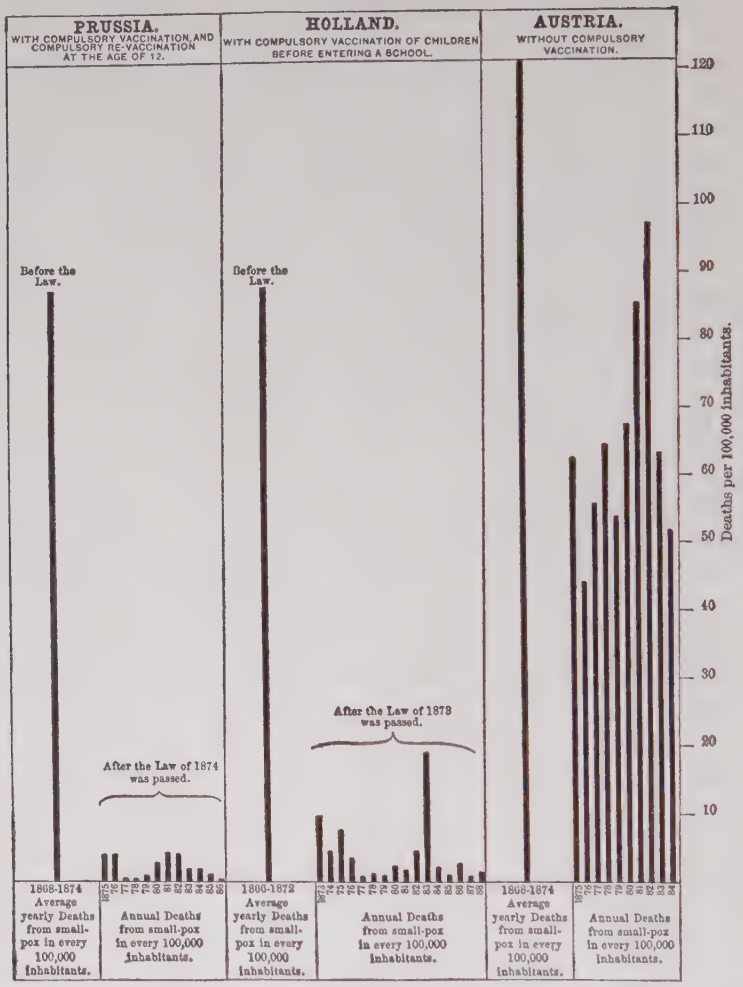

PRUSSIA.
WITH COMPULSORY VACCINATION AND COMPULSORY RE-VACCINATION AT THE AGE OF 12.
HOLLAND.
WITH COMPULSORY VACCINATION OF CHILDREN BEFORE ENTERING A SCHOOL.
AUSTRIA.
WITHOUT COMPULSORY VACCINATION.
Before the Law.
After the Law of 1874 was passed.
Before the Law.
After the Law of 1873 was passed.
Deaths per 100,000 inhabitants.
120
110
100
90
80
70
60
50
40
30
20
10
1868-1874 Average yearly Deaths from small-pox in every 100,000 inhabitants.
Annual Deaths from small-pox in every 100,000 inhabitants.
1866-1872 Average yearly Deaths from small-pox in every 100,000 inhabitants.
Annual Deaths from small-pox in every 100,000 inhabitants.
1868-1874 Average yearly Deaths from small-pox in every 100,000 inhabitants.
Annual Deaths from small-pox in every 100,000 inhabitants.

FIG. 164.—TABLE SHOWING THE PROTECTIVE POWER OF VACCINATION. (Carsten.)

Time for Vaccinating.—In selecting a time for vaccination, the child's age and general health must be taken into consideration. It is pretty well established that the constitutional disturbance is much less in infancy than in later childhood; and there is besides in infancy less chance of accidental infection of the vaccine wound. Between the ages

of two and six months seems the best general time for vaccination. In delicate infants or in those whose nutrition is a matter of great difficulty, those who are syphilitic, those suffering from eczema or any other form of active skin disease, vaccination should be deferred until the child is in good condition, unless he is likely to be exposed to smallpox.

Methods of Vaccinating.—In our experience it is preferable to vaccinate in a single place rather than to make two or three inoculations. Either the leg or the arm may be chosen; in young infants it is usually easier to protect the vaccine sore upon the leg than upon the arm; in children old enough to run about, the arm is to be preferred, as being more easily kept at rest. The point selected for inoculation should be either the outer aspect of the left calf, about the junction of the middle with the upper third of the leg, or, if the arm is chosen, the insertion of the left deltoid. Vaccination should be regarded as a minor surgical operation and the hands of the physician, as well as the arm of the patient, should be washed with soap and water, dried, and the skin then washed with alcohol.

The New York Health Department supplies with each tube of lymph, a sterilized needle and a rubber bulb. A single scratch not more than one-fourth of an inch long is made with the needle just deep enough to draw blood; or a minute scarification may be made not over one-eighth of an inch in diameter. The ends of the capillary tube are broken off, and the lymph blown out of the tube upon the scratched surface and rubbed in for a full minute. The wound should not be covered until dry; a sterilized bandage should then be applied. The limb should not be washed for twenty-four hours.

The Normal Course of Vaccinia.—The course of a proper vaccination-pock is quite uniform, and one which does not follow this course should not be considered protective. The wound heals and nothing is noticed until the third or fourth day, when a red papule makes its appearance. Usually in twenty-four hours more a small vesicle appears which enlarges until the sixth or seventh day, reaching its full development about the ninth day. Its shape and size depend somewhat upon the extent of the scarification (Figs. 165-169). The vesicle is usually from one-fourth to one-half inch in diameter; it is of a pearly-gray color and has a depressed center. During the next two days an areola forms about the vesicle extending from it a variable distance, usually for one or two inches into the healthy skin. Its size depends upon the intensity of the infection. This areola is normally of a bright-red color and accompanied by some induration. It is generally at its height about the ninth day. The vesicle usually dries down to a firm, dark crust which remains from one to three weeks and falls off, leaving a bluish scar which fades to white, becoming somewhat honey-combed. When the process is at its

FIG. 165.—Fifth day.

FIG. 166.—Seventh day.

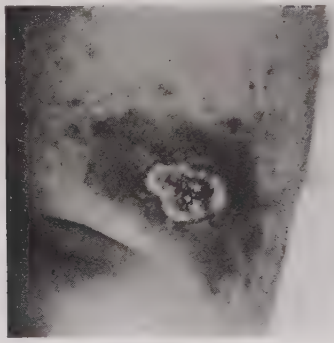


FIG. 167.—Ninth day.

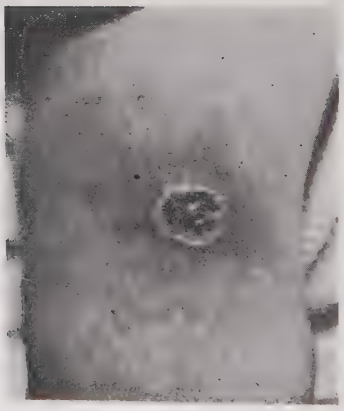


FIG. 168.—Eleventh day.

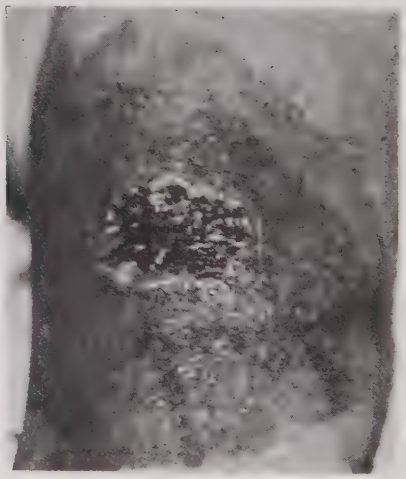


FIG. 169.—Tenth day.

FIGS. 165–169.—VACCINE VESICLES. (Two-thirds natural size.)

FIGS. 165, 166, 167, and 168 show typical appearance of vesicle at the different stages when a very small scarification is made.

FIG. 169 shows the effect of a larger scarification with a more intense areola. The amount of inflammation is excessive but not unusual.

height some constitutional disturbance is usually present; there may be loss of appetite, fretfulness, and general indisposition, and the temperature is usually elevated from one to three degrees. The lymph nodes in the groin or axilla may be tender and swollen. These symptoms generally last for three or four days.

If in a young infant the first inoculation is unsuccessful, at least three trials should be made with good virus, and in the event of further failure, after a year vaccination should be repeated. A failure to inoculate does not mean insusceptibility to smallpox, as is often popularly believed, but most frequently arises from the fact that the virus is inert. We have known one case in which the seventh, and another in which the thirteenth, inoculation was successful after previous failures; occasionally there are seen children who can not be inoculated at all.

Fig. 170. — Generalized Vaccinia. Boy eight years old.

Constitutional symptoms, as previously stated, may be absent in very young infants; but in others there is quite constantly present a fever which runs a fairly regular course. It usually begins on the fourth or fifth day, is remittent in type, and rises gradually, reaching its highest point with the full development of the vesicle. At this time even without complications it may touch 104° or 105° F. The duration of the fever in cases running the usual course is four or five days. Accompanying the fever there may be anorexia, restlessness, loss of sleep, slight indigestion, and other symptoms of a general indisposition.

Both the local and the general symptoms are sometimes more severe. This may depend upon the susceptibility of the child, even though the lymph is pure and the vaccination properly done. The original vesicle may be much larger than usual, and small secondary vesicles may form in the neighborhood. In very rare instances a generalized eruption of true vaccine vesicles occurs with fever and other general symptoms of corresponding severity (Fig. 170). Single vesicles may be produced on distant parts of the body as a result of auto-inoculation, usually by scratching. When eczema of the face is present, inoculation is not infrequently carried thither. Most of the very sore arms and legs, however, are due

to infection from pyogenic bacteria accidentally introduced at the time of vaccination but more often subsequently. In the milder cases the swelling and other evidences of local inflammation are more marked than in a normal vaccination; a drop or two of pus forms beneath the crust, and when the latter comes away an excavation is left which heals in two or three weeks. Or, the inflammation may extend more deeply into the connective tissue, to be followed by more extensive suppuration or sloughing, leaving an ugly ulcer an inch or more in diameter which slowly fills by granulation in from five to eight weeks. Sometimes the period of incubation is unduly prolonged, so that the vesicle does not form until the twelfth or fourteenth day, although its subsequent course may be quite normal. In other cases the incubation is very much shorter than usual, and the vesicle may appear as early as the fourth or even the third day.

Much has been written about the so-called "raspberry excrescence" which not very infrequently takes the place of a proper vesicle. It is of a dark-red color, elevated, smooth or slightly granular, not sensitive, having no areola and no constitutional symptoms. It generally persists for two or three weeks, and slowly disappears, leaving no scar. It is usually the result of virus of feeble activity, and if it gives any protection it is very slight. Such cases should always be re-vaccinated, and in our experience re-vaccination is usually successful.

Complications and Sequelae.—Post-vaccine eruptions are many and of great variety. The most frequent is a general roseola, usually occurring at the height of the local process. Other eruptions seen are urticaria, and, rarely, purpura. Complications are chiefly from accidental infection. Syphilis and tuberculosis are excluded by the modern method of procuring the lymph. Tetanus in rare instances has followed vaccination. It may result either from introduction of the bacilli with the vaccine lymph but more often from subsequent accidental infection of the wound or sore. Cases of the first mentioned variety are extremely rare. By proper legal restrictions regarding the production of vaccine virus they should be entirely eliminated. Its production should never be permitted in a district in which tetanus is endemic; and each quantity of lymph sent out should be tested for tetanus. In the great majority of the reported cases in which tetanus has followed vaccination the evidence is strong that infection occurred subsequent to vaccination, owing to want of proper care or insufficient protection of the vaccinated part. It should not be forgotten that vaccination produces an open wound, which may become infected like any other wound. The most common form of local infection is cellulitis, which may terminate in suppuration or sloughing at the site of vaccination, and sometimes may cause suppuration of the neighboring lymph nodes. Erysipelas may develop at any time before

the wound is entirely healed; it is usually due to neglect of proper precautions in the care of the vaccine sore.

The mortality of vaccination is stated by Voigt, from careful statistics drawn from German sources, to have been 35 in 2,275,000 cases, including both primary and secondary vaccinations. Of the deaths, 19 were due to erysipelas, 8 to gangrene, 2 to cellulitis, 3 to "blood poisoning," and 3 to other causes. Nearly all the deaths from vaccination are from causes which are preventable.

Treatment.—The whole purpose of treatment is to prevent infection. The first essentials are a clean limb, pure virus, and a sterile needle; the next, to allow thorough drying of the wound before the clothing touches it. After this no treatment is necessary until the vesicle forms. Then the important thing is to prevent scratching and the irritation by the clothing. All vaccine shields are objectionable. For an infant nothing is better than the sterilized gauze bandage, which can be kept in place by sewing to the stocking or to the sleeve of the shirt. Any constriction of the limb is injurious. For older children the simplest dressing is a pad of sterile gauze fastened to the limb by two pieces of adhesive plaster. Should the vesicle rupture and discharge serum, it should be kept clean and dry by dusting daily with boric acid. When the local symptoms are at all severe the limb should be kept at rest. An infected vaccination wound, like any other infected wound, requires careful surgical treatment; disastrous results often follow the use of poultices and other applications much in vogue in domestic practice.

CHAPTER VI

PERTUSSIS

(*Whooping-Cough*)

Pertussis is a contagious disease which prevails epidemically and, in all large cities, endemically. Although it may affect persons of any age, it is generally seen in young children. While in later childhood pertussis may be ranked as one of the milder infectious diseases, in infancy it is one of the most fatal. Its principal complications are bronchopneumonia and convulsions. Pertussis is characterized by catarrhal and nervous symptoms. The catarrh affects the mucous membrane of the respiratory tract, and is probably due to a specific form of infection. It is accompanied by a hyperesthetic condition of this mucous membrane. The most prominent nervous manifestation is a peculiar

spasmodic cough which occurs in paroxysms, and from which the disease takes its name. The cough is no doubt of reflex origin, from an irritation which has been located by different writers in various parts of the respiratory tract. In addition to these conditions, there is present in pertussis a marked irritability of the nervous system, which in infancy often shows itself by convulsions. Whooping-cough is a disease whose importance is too often passed over lightly. In New York State it causes more deaths than scarlet fever and nearly as many as does typhoid fever.

Etiology.—Present evidence points to the Bordet-Gengou bacillus as the specific organism of pertussis. It is a small Gram-negative bacillus which in many points resembles the influenza bacillus. It is difficult to obtain the organism from the respiratory secretion unless the plug of mucus brought up after the paroxysm of coughing is secured, as it develops chiefly in the lower respiratory tract. It is found only in the early stage of the disease, rarely later than a week after the whoop begins. Smears are unreliable for diagnosis; only cultures are to be depended upon. In practically all cases there is mixed infection, certainly after the first week or two. There may be associated the pneumococcus, the B. influenzae, the staphylococcus, or streptococcus. There are still lacking some elements of proof that the Bordet-Gengou bacillus is the cause of pertussis. Although it has been found in the great majority of cases examined by competent observers early in the disease, yet its absence in some typical cases cannot be explained. The results of complement fixation tests have not been uniform, but these may be due to differences in the strains of the organism. Finally there is evidence that other forms of infection of the respiratory tract may produce a group of symptoms which are clinically indistinguishable from true pertussis; i. e., a contagious catarrh and a paroxysmal cough with a duration of four to eight weeks. This we have repeatedly seen associated with the presence of the B. influenzae.

Proximity to a patient seems all that is required to communicate the disease and even close proximity is not necessary. Czerny places the infective distance at about five feet from the patient. The disease seems to be spread chiefly by droplets diffused by coughing and sneezing.

Predisposition.—Fully one-half the cases of pertussis occur during the first two years of life. The following are the statistics of Szabo (Buda-Pesth), showing the ages at which the disease was met with in 4,591 cases, comprising the records of one clinic for thirty-four years:

Under one year	1,028 cases	Three to four years	904 cases
One to two years	1,008 "	Four to seven years	803 "
Two to three years	659 "	Over seven years	189 "

The susceptibility of young infants to pertussis is very great. To them unquestionably the disease may be carried by a third person. Many cases are on record in which pertussis has occurred during the first month, and one has come to our notice where a child twelve days old was attacked, whose mother was suffering from the disease. The disease is nearly twice as frequent in the winter and spring as in the summer and autumn. Epidemics of pertussis often occur at the same time with or follow those of measles.

The susceptibility to pertussis is very great, and is equalled only by that to measles. Biedert reports that of 401 children exposed during an epidemic in a certain village, 366, or ninety-one per cent, took the disease.

As a rule one attack protects the individual during his life. The great majority of the reported instances of second attacks are certainly to be explained by mistakes in diagnosis. These may be almost unavoidable; for it is at times almost impossible to distinguish true pertussis from the paroxysmal cough which occurs in some cases of influenza.

Infective Period.—Pertussis may be communicated from the very beginning of the catarrhal stage; it is more contagious at this period than later. There seems little doubt that it is contagious throughout the spasmodic stage, but the infectivity of the disease after the first few weeks is slight. The recurrence of the whoop with a fresh cold, after it has once ceased, cannot be considered a relapse nor regarded as contagious. Quarantine is generally required for two months. The usual source of the contagion is the patient, rarely the room or the clothing.

Incubation.—The very gradual onset of pertussis renders it impossible in the majority of cases to fix the exact date, and hence to establish the definite duration of the period of incubation. In cases where this could best be determined it has usually been from seven to fourteen days, or about the same as in measles. If, after an exposure, sixteen days pass without the development of a cough, the probabilities are very strong that the disease has not been contracted.

Lesions.—The only constant lesion of pertussis consists in a catarrhal inflammation of varying intensity, which affects the mucous membrane of the larynx, trachea, and bronchi, and sometimes that of the nose and pharynx. Mallory claims that the presence of the bacilli between the ciliae of the epithelial cells of the trachea and bronchi is the specific lesion. Others have found a similar condition in influenza. If the child dies during a paroxysm, either with or without convulsions, the brain is found intensely congested and may be the seat of punctate hemorrhages, or even larger extravasations. The lungs always show emphysema if the attack has been severe or protracted. The other pulmonary lesions

are due to complications, the most frequent of which is bronchopneumonia. Catarrhal enteritis and colitis are not infrequent.

Symptoms.—The symptoms of pertussis are usually divided into three stages—the catarrhal, the spasmodic, and the stage of decline.

The catarrhal stage continues on the average for about ten days, although cases show considerable variation on this point. Some children whoop almost from the very beginning of the disease, while others may cough for three or four weeks before a typical whoop is noticed. The symptoms in the beginning are indistinguishable from those of an ordinary attack of subacute tracheobronchitis, and unless there has been an exposure to pertussis no suspicion is excited. After five or six days, however, the cough, instead of abating as in an ordinary cold, gradually increases in severity and occurs in paroxysms. At first these are mild, and there are only two or three a day, but they gradually increase in frequency and severity until the typical whoop is heard which marks the beginning of the spasmodic stage. During the first stage there may be symptoms of a mild grade of catarrhal inflammation of the nose, pharynx and larynx, and often there is a slight elevation of temperature.

The Spasmodic Stage.—In a typical paroxysm of average severity the child, who can usually foretell it, will often run for support to the lap of the mother or the nurse, or seize a chair with both hands. There now occurs a series of explosive coughs, from ten to fifteen in number, coming in such rapid succession that the child can not get his breath between them; the face becomes a deep-red or purple color, sometimes almost black; the veins of the face and scalp stand out prominently; the eyes are suffused, and seem almost to start from their sockets; there follows a long-drawn inspiration through the narrowed glottis, producing the crowing sound known as the whoop; and then another succession of rapid coughs follows and another whoop. In a single severe paroxysm, which lasts several minutes, the child may whoop half a dozen times; with the final paroxysm a mass of tenacious mucus is usually brought up. In a young child vomiting is almost certain to follow, if food has been recently taken. Epistaxis sometimes occurs with nearly every severe paroxysm, but in most cases the bleeding is slight. After a severe attack the child is at times so exhausted as to be hardly able to stand. There is profuse perspiration; his mind is confused, and he may be completely dazed. In infants the attack may result in a degree of asphyxia requiring artificial respiration. Those old enough to describe their sensations tell of a sense of impending suffocation, the suffering from which is almost indescribable.

The number of severe paroxysms or "kinks" in twenty-four hours varies, according to the severity of the case, from half a dozen to forty

or fifty. There are always many more of a milder form. Paroxysms are often excited by eating or drinking anything cold, by a draught of air, or by imitation; they are usually more frequent during the night than the day, and in a close room than in the open air.

In less severe cases no paroxysms of the grade above described may occur, and no typical whoop may be heard throughout the attack; but the paroxysmal nature of the cough which continues until the plug of mucus is expelled, the watery eyes, and the vomiting which follows a paroxysm, stamp the disease as pertussis. In young infants the whoop is frequently not marked. The child sometimes coughs until he is asphyxiated, and yet no whoop occurs. The paroxysms are also modified by intercurrent disease, especially by attacks of pneumonia or severe bronchitis. At such times they usually become less frequent and less typical, and may be absent for several days, returning as the complication subsides.

The seat of the irritation which produces the cough has been variously located by different observers. Some have thought it to be in the nose, others in the trachea, the bronchi, or the larynx. It is very probable that it may not always be in the same place and that the infectious catarrh, which is really the most important element in the disease, may vary in its intensity and location in different cases. The weight of evidence seems to be that in the great majority of cases the source of irritation is in the larynx or trachea. From laryngoscopic examinations made during the disease, Von Herff found the mucous membrane of the larynx to be swollen and congested, and occasionally the seat of small hemorrhages or superficial ulcers. He states that the frequency and severity of the paroxysms corresponded with the degree of laryngitis, and he found that a paroxysm could always be excited by irritating the mucous membrane between the arytenoid cartilages. During a paroxysm he observed that there was a collection of mucus on the posterior laryngeal wall, the removal of which had the effect of shortening the paroxysm.

Rossbach made laryngoscopic examinations, with negative results so far as the larynx was concerned, but he states that a plug of mucus could always be seen in the lower trachea for one or two minutes before the paroxysm occurred. There is little doubt that this collection of mucus is the exciting cause of the paroxysm, as it is a familiar clinical fact that the paroxysm continues until this is dislodged.

The average duration of the spasmodic stage is about one month. It increases in intensity for the first two weeks, remains stationary for about a week, and then gradually diminishes in severity. The course and duration of this stage are, however, subject to wide variations. In mild cases it may last only a week; in severe cases, especially in the winter

season, it may continue for three months, at times almost subsiding, but lighting up again with all its previous severity with every fresh catarrhal attack. After it has entirely ceased the whoop may return with an attack of bronchitis, and continue for a month or more. This is not to be regarded as a true relapse of pertussis. The habit of the paroxysmal cough once established, it tends to recur with every slight bronchitis, often for months afterward.

The Stage of Decline.—Gradually the severity of the paroxysms abates, the whoop ceases, and the cough resembles more and more that of ordinary bronchitis. This stage usually continues about three weeks, but may be prolonged indefinitely in the winter months.

Complications.—*Hemorrhages.*—The hemorrhages of pertussis are mechanical, and depend upon the intense venous congestion which accompanies the paroxysm. Epistaxis is the most frequent variety, and occurs in a considerable proportion of the severe cases, in a few with almost every severe paroxysm, but it is rarely severe enough to require local treatment. Hemorrhages from the mouth may have their origin either in the pharynx or the bronchi, the blood being brought up by the cough; such hemorrhages are usually small. Conjunctival hemorrhages are less frequent, and are usually slight, although we have seen the entire conjunctiva covered. In a case under our observation there was bleeding from both ears with every severe paroxysm for more than a week. This child had previously suffered from scarlatinal otitis, with perforation of the drum membrane. Small extravasations into the cellular tissue beneath the eyes are occasionally seen, giving an appearance somewhat like an ordinary "black eye." Intracranial hemorrhages are not frequent, but many examples have been recorded, and they may be severe enough to produce death. They are usually meningeal, very rarely cerebral; according to their extent and location they may produce hemiplegia, monoplegia, aphasia, facial paralysis, or disturbances of sight, hearing, or sensation; in addition, there may be convulsions or rigidity, but rarely complete coma. The extravasations are sometimes small and the symptoms which they produce may disappear at the end of a few weeks. More extensive hemorrhages cause serious results. In almost every instance these hemorrhages have occurred as a direct result of the severe paroxysms. Purpura hemorrhagica is occasionally seen as a sequel of pertussis.

Respiratory System.—The most serious complications of pertussis are connected with the lungs. By far the largest proportion of deaths is due to pulmonary complications, usually bronchopneumonia. This is more frequent in winter and spring than in the summer months, and is especially to be dreaded during infancy. In later childhood lobar pneu-

monia is occasionally seen. Pneumonia rarely begins before the second week of the disease, and most frequently develops at the height or toward the close of the spasmodic stage. The physical signs present no peculiarities; the cough changes somewhat in character during the pneumonia, and the whoop may not be heard. The prognosis of the pneumonia is bad, because of the debilitated condition of the children at the time of its occurrence. A great danger is from the supervention of convulsions, this being a frequent mode of termination. As there is always considerable emphysema, the rapidity of breathing is frequently out of proportion to the temperature, which often is only moderately elevated. If the child escapes the dangers of the acute stage, death may still occur from exhaustion, owing to the protracted course which the disease frequently runs.

Bronchitis of the large tubes is present in almost all the severe cases, and is not of itself serious. Bronchitis of the small tubes has the same dangers and the same complications as bronchopneumonia.

Vesicular emphysema is invariably present in every case of pertussis which comes to autopsy. A certain amount of it certainly occurs in every severe case. It is produced by the forcible cough of the paroxysm. In very severe cases interstitial emphysema is also found. Rupture of the air-blebs which form on the surface of the lung may lead to emphysema of the cellular tissue of the mediastinum, and the air may find its way along the great vessels into the neck, and finally into the subcutaneous cellular tissue of the entire body. Cases of general subcutaneous emphysema have been reported by Croker and by Hodge, both of which ended fatally, one in three and one in eight days from the beginning of the emphysema. In the great majority of the cases vesicular emphysema is not permanent.

Digestive System.—During the summer, infants with pertussis are almost certain to suffer from diarrhea; it may be only an occasional symptom, or the attack may be severe and prolonged, resulting in the development of ileocolitis. The intestinal complications may be almost as serious in summer as are those of the respiratory tract in winter. Vomiting is even more frequent than diarrhea, and while it may be distressing at any age, it is especially so in infancy. So frequently does the taking of food excite vomiting, that the nutrition of these patients often becomes a matter of the greatest difficulty, and in fact the most serious problem in the management of a case. Malnutrition and even marasmus may follow, or the general resistance of the child may become so reduced by lack of food that he falls a ready prey to pneumonia.

Nervous System.—There may be convulsions, coma, paralysis, aphasia, disturbances of sight or hearing, and in rare cases even the

mental condition may be affected. The most serious of these complications are convulsions. They are much more frequent in infancy than later, and particularly in those who are rachitic, in whom they are often fatal. Convulsions are of course more common in severe attacks, but they may occur suddenly when there has previously been no cause for anxiety. They are especially to be dreaded if pneumonia is present. The attack of convulsions may be the culmination of the extreme degree of nervous irritability which accompanies the paroxysm, it may be due to asphyxia, or to an intracranial lesion; if the latter, there is usually meningeal hemorrhage. This is to be suspected if there are continued convulsions for several hours, with general rigidity or hemiplegia.

Disturbances of sight are not infrequent in severe cases; usually these are transient, but there may be blindness lasting two or three days or even weeks. The transient symptoms depend most likely upon circulatory changes that occur in the brain during the paroxysm, while those which last for two or three weeks are probably due to meningeal hemorrhage. Disturbances of hearing are rare. The different forms of paralysis occurring with pertussis may likewise be transient or permanent. They are to be explained in the same way as the disturbances of the special senses. The most common form is hemiplegia.

Albuminuria is not infrequent, being found in sixty-eight of eighty-six examinations by Knight. The quantity of albumin is rarely large, and it may be accompanied by a few hyaline casts. Both are probably the result of circulatory disturbances in the kidney. Other complications of pertussis are hernia, prolapsus ani, and ulcer of the frenum linguae.

Diagnosis.—The only constant features of pertussis are the course of the disease and its communicability. In many cases the typical whoop is never heard. There are no symptoms by which a positive diagnosis can be made in the catarrhal stage; but a cough not accompanied by fever or physical signs, which steadily increases in severity for two weeks, in spite of treatment, and which occurs chiefly at night, is always suspicious. When, in addition, the cough begins to come in paroxysms, accompanied by suffusion of the face and occasionally by vomiting, there can be little doubt even though no whoop is heard. If the disease is prevalent the diagnosis is practically certain. Mild cases which do not go even as far as the symptoms mentioned are most puzzling. But if there is a history of exposure, if the cough continues from four to six weeks, little influenced by treatment, and if other typical cases follow, the disease must be pertussis. Without evidence of communicability, however, one may be in doubt even after the disease is over. In certain cases of influenza there may be a paroxysmal cough which by its symptoms

and course can not be distinguished from pertussis, but which may be recognized by an examination of the blood and sputum (vide Influenza).

In early infancy any cough may have more or less of a spasmodic character, and a fairly typical whoop is often heard in the course of an ordinary bronchitis. We have several times seen abortive or very short attacks in one member of a family of children, the others having the disease in a typical form. Occurring by themselves such cases can not be recognized.

Irritation of the pneumogastric or recurrent laryngeal nerve from tuberculous tracheal or bronchial lymph nodes, or from a foreign body in the air passages, may give rise to a spasmodic cough, which in certain cases may be indistinguishable from pertussis. The prolonged duration of the symptoms is sometimes the only diagnostic point; but the paroxysms are usually not so severe as in true pertussis, and the course is generally less typical.

The blood examination is of much assistance in diagnosis. The leucocytosis accompanying pertussis far exceeds that of any other afebrile disease of the respiratory tract. It appears in the early part of the convulsive stage, and disappears slowly with improvement. The total count is usually between 15,000 and 30,000, although it may reach 50,000. There is a great increase in the lymphocytes at the expense of the polymorphonuclear neutrophiles. The lymphocytes may form 60 to 80 per cent of the total leucocytes. The leucocytosis is little influenced by complications, and even during bronchopneumonia the lymphocytes may continue to be in excess.

Prognosis.—The most important factor in the prognosis of the disease is the age of the patient. After the fourth year it is indeed rare that either a fatal result or serious complications are seen; but during infancy, and particularly during the first year, there are few diseases more to be dreaded. This is especially true on account of the connection of whooping-cough with the three most fatal conditions of infancy —bronchopneumonia, diarrheal diseases, and convulsions. Fully two-thirds of the deaths from whooping-cough occur during the first year of life. The prognosis is very much worse in infants under three months than in those who are older and consequently have more resistance. It is better in the summer than in the winter, because bronchopneumonia is then less frequent. It is particularly bad in delicate infants, in those who are rachitic, in those who are prone to attacks of bronchitis, in those who have suffered previously from pneumonia, and in those with a strong tendency to tuberculosis.

The exact mortality of whooping-cough it is difficult to state in figures. During the first year of life it is probably not far from twenty-five

per cent, although it diminishes rapidly after this time. In foundling asylums and hospitals for infants it is to be ranked among the most fatal diseases, and in some epidemics the mortality in such institutions is as high as fifty per cent.

Fully two-thirds of the deaths during whooping-cough are from bronchopneumonia; the next most frequent cause is diarrheal diseases. Convulsions may be the mode of death in either of the above conditions, or may occur apart from them. During the first year, death often results from marasmus, the child having been reduced by the prolonged disease. Occasionally death is due to asphyxia following a severe paroxysm, to intracranial hemorrhage, or to general emphysema.

As a predisposing cause of generalized tuberculosis, pertussis is second only to measles. In both diseases tuberculosis develops in much the same way and from practically the same causes.

Prophylaxis.—Pertussis is a contagious disease, and a child suffering from it should be isolated from other children whenever this is possible. Children with pertussis should never be allowed to attend school, and needless exposure should always be avoided.

Young infants, delicate children, and those with a predisposition to tuberculosis, should be most carefully protected against exposure, since it is in them chiefly that the disease is likely to be serious. As it is from the patient that the disease is nearly always contracted, there does not exist the same necessity for the careful disinfection of apartments as after other contagious diseases. In institutions, however, this should always be practiced, and in private houses if the room is subsequently to be occupied by an infant. The prophylactic use of vaccines is referred to under Treatment.

It is as undesirable as it is impossible to confine a child with pertussis to a single room during the attack; all those persons for whom exposure would be dangerous should therefore be sent away from the house. Quarantine should continue for at least six weeks, or until the spasmodic stage is over.

Treatment.—We have as yet no specific remedy for pertussis. The important thing in most cases is the hygiene or general management of the case; fully half of the cases seen in practice require nothing more. Much harm is done by indiscriminate drug giving.

General Measures.—Fresh air is important throughout the attack. It is almost invariable that the paroxysms are fewer while patients are out of doors, and more frequent when they are in close rooms. Older children with pertussis may go out even in winter except on stormy, raw, or windy days. With infants and delicate children, however, the outdoor treatment in cold weather so enthusiastically advocated by some writers should be used with the greatest caution. It should not be permitted

if the patient has even the slightest amount of bronchitis. Our experience is that during the winter in a climate like that of New York or New England, the class of patients just referred to are better off indoors, taking their airing in their rooms. In warm weather or in a mild climate all children should be kept in the open air as much as possible.

A change of climate is desirable when the cough is unduly prolonged, also for delicate children in winter. A warm place at the seashore is one which is most likely to be beneficial. The improvement following a sea voyage is often very marked, surpassing even a residence at the seashore.

The rooms occupied by children suffering from pertussis should be frequently changed, thoroughly aired and cleaned. A change of rooms, clothing, bedding, etc., sometimes exerts a marked influence on the course of very prolonged attacks, the inference being that continued re-infection takes place. Such a change should be made twice a week, and it is of special importance in hospitals, where many children quarantined in a single ward seem to cough interminably.

Careful feeding and attention to the bowels are matters of the greatest importance; with infants particularly, chronic indigestion and abdominal distention have a very marked effect in increasing the frequency of the paroxysms. The abdominal support furnished by a snugly fitting band, adds materially to the comfort of the patient in a severe attack. Feeding is difficult since vomiting occurs so easily. In most cases it is necessary to repeat the meal in a short time, if the first one has been vomited. Children over two years old should in all such cases be kept largely upon a fluid diet; the meals should be smaller and more frequent than in health. For infants, milk should be modified according to the child's digestive symptoms. Any medication which causes disturbance of the stomach should be omitted.

Local applications to the rhinopharynx or to the larynx by means of a spray or swab have been advocated by many. We have never seen the beneficial results claimed, and believe them to be exaggerated. The application of cocain to the larynx should under no circumstances be employed in young children.

Inhalations are of much more value. They are useful to modify the catarrh by allaying irritation, facilitating the expulsion of the mucus, and possibly as antiseptics. Those most employed are creosote and cresolene. In our experience creosote is the best. These substances may be used upon cotton in a respirator, or vaporized over an alcohol lamp. The possibility of absorption should not be forgotten, and the urine should be watched. When the paroxysms are frequent and of great severity, chloroform may be used to ward off convulsions or prevent dangerous asphyxia. In such conditions O'Dwyer used intubation with striking benefit. The

tube entirely overcomes the glottic spasm which is the chief cause of suffering and danger.

Internal Medication.—Of the innumerable drugs which have been recommended for this disease, there are two which possess undoubted advantages over all others, viz., belladonna and antipyrin. In giving belladonna it is important to begin with a small dose and cautiously increase both its frequency and size. To an infant two years old, one-fourth of a minim of the fluid extract may be given every four hours as an initial dose, gradually increasing to every two hours; if atropin is used, gr. 1-800 may be given in the same way. Although belladonna usually has a decided influence in reducing both the frequency and the severity of the paroxysms, it causes many unpleasant symptoms, and its effects must be closely watched.

Antipyrin has been in our experience more generally useful than any other single drug. It may be given with safety, even to young infants, in considerably larger doses than are ordinarily employed. For a child six months old the initial dose may be one grain every three hours; later this may be given every two hours. For a child two years old the initial dose may be two grains repeated every four to six hours, gradually increasing up to two grains every two hours. Should pneumonia develop, the antipyrin should be discontinued. A combination of the bromid of sodium with antipyrin is often better than the latter given alone.

Nearly all drugs which allay nervous irritability have a certain amount of effect in controlling the paroxysms of pertussis; codein, chloral, and trional are useful where the night attacks are so severe as to prevent sleep. We do not believe that any form of internal medication or local treatment shortens pertussis; but, inasmuch as the disease is self-limited, great benefit to the patient results from the reduction of the number and the diminution of the severity of the paroxysms.

Vaccines have been much employed in the treatment of pertussis during recent years with exceedingly variable results. Vaccines made from stock cultures of the Bordet-Gengou bacillus have been most widely used. Several facts militate against success by this treatment: first, our uncertainty regarding the bacterial cause. While the Bordet-Gengou bacillus has been altogether most frequently found, a paroxysmal cough which clinically is indistinguishable from pertussis may be associated with the different forms of so-called hemoglobinophilic bacteria. In the second place there are apparently several distinct strains of the Bordet bacillus. The evidence as to curative value of vaccines is as yet inconclusive. There is somewhat more evidence that they are useful as a means of prophylaxis; but this point is by no means established. However, inasmuch as they are harmless the use of vaccines is advisable as a preventive measure in the case of young infants exposed. The question

of therapeutic dosage is still unsettled; from 25 to 100 millions, according to the age of the child, repeated every two to four days is at present to be advised. For prophylaxis full doses are also needed; they should be repeated for three or four doses at intervals of five or six days.

In establishing the value of any method of treatment, it should be remembered that the number of cases in which the duration of the disease is short is quite large, and also that almost any method of treatment if employed after the attack has reached its height will be thought beneficial, as the natural tendency is then to improve. The value of any particular line of treatment is to be judged in a given case only by its effect in reducing the number and severity of the paroxysms. This ought to be evident in the case of drugs or vaccines within a few days, and can only be determined by keeping a careful record of the number of severe paroxysms day and night.

In a mild case, when the number of paroxysms does not exceed eight or ten during the day, when there is no vomiting and the general health is not affected, it is not usually advisable to continue the administration of any drug throughout the disease. A single dose of antipyrin or codein at night may be all that is necessary. All cases in infants must be watched with great care and the parents warned of the possible dangers which may supervene suddenly, even in the course of mild attacks. For severe cases antipyrin should be given to diminish the frequency and the severity of the paroxysms, and inhalations of creosote used if much catarrh is present. All the fresh air possible should be allowed, but without exercise. For older children the same plan of treatment may be followed, or quinin or belladonna may be substituted for the antipyrin.

As these drugs are given solely for the purpose of diminishing the frequency and severity of the paroxysms, their continuous use should be deferred until the symptoms are sufficiently severe to greatly disturb the child, the benefit at this period being more striking than if they are begun early and used continuously.

CHAPTER VII

MUMPS

(*Epidemic Parotitis*)

Mumps is a contagious disease characterized by swelling of the parotid, and sometimes of the other salivary glands, with constitutional symptoms which are usually mild. Both severe complications and a

fatal termination are extremely infrequent. The disease is not a very common one, and general epidemics are not common.

Pathology and Lesions.—The contagious character, regular incubation period and typical course, stamp the disease as a general one due to a specific organism, but this has not been definitely determined. Unquestionably the virus is present in the saliva of affected persons and in all probability the poison is eliminated by Steno's duct. By inoculating the saliva from patients with mumps into the parotid gland of cats, Wollstein has reproduced a similar disease in these animals with typical symptoms and transferred this again to other animals with the production of the same symptoms. It has long been a popular tradition that domestic cats were occasionally the subjects of mumps.

The precise nature of the changes in the gland is still a matter of dispute, as opportunities for pathological examination are very rare. From existing evidence it would appear that the gland substance is first involved, and afterward the surrounding connective tissue. The gland is the seat of an intense hyperemia and edema; the walls of the salivary ducts are swollen, and the ducts are obstructed. While the primary disease does not tend to excite suppuration, pyogenic germs may occasionally gain entrance and an abscess form; but this is to be regarded as a rare accidental infection.

In the great proportion of cases the parotids alone are affected, although the same changes are occasionally found in the other salivary glands. There are no other essential lesions of the disease, those which are found depending upon complications.

Etiology.—Mumps is spread by contagion, close contact being usually required to communicate the disease, although it is known to have been carried by a third person and even by clothing. The susceptibility of children to the poison of mumps is much less than is the case with the other contagious diseases, so that only a small number of those who are exposed take the disease. The greatest predisposition is between the fourth and fourteenth years. Infants are rarely affected, although a case in a child three weeks old is vouched for by so good an observer as Demme.

Mumps is contagious from the beginning of the symptoms. Two cases have come under our notice in which the disease was communicated before any swelling was seen. It is impossible to fix with certainty the duration of the infective period. The disease is undoubtedly communicable for a few days after the swelling has subsided; and for safety a case should be isolated for three weeks from the beginning of symptoms, or one week after the swelling has disappeared.

Incubation.—In forty-eight collected cases in which the incubation was definitely determined, it varied between three and twenty-five

days. It was less than fourteen days in only four cases, and in twenty-six of the forty-eight cases it was between seventeen and twenty days. In three cases of our own in which it could be definitely fixed, the incubation was nineteen days in one case and twenty days in two cases. The average period of incubation, then, may be stated to be from seventeen to twenty days.

Symptoms.—In the milder cases the local symptoms are the first to attract attention; in those which are more severe there are frequently prodromal symptoms of from twelve to forty-eight hours' duration—anorexia, headache, vomiting, pains in the back and limbs, and fever. Soltmann has reported a case ushered in by convulsions. The initial temperature in a mild attack is 100° to 101° F.; in a severe one, from 102° to 104° F.

Of the local symptoms, the pain usually precedes the swelling; it is increased by movement of the jaws, by pressure, and sometimes by the presence of acid substances in the mouth. It is usually referred to the posterior part of the jaw just below the ear. The swelling may begin simultaneously in both parotids, but more frequently one side is involved a day or two in advance of the other. It usually reaches its maximum on the third day, remains stationary for two or three days, and then subsides gradually. The degree of swelling varies with the severity of the attack. When it is marked, the patient may be so changed in appearance as scarcely to be recognizable. The swelling fills the lateral region of the neck between the jaw and the sternomastoid muscle and extends forward upon the face to the zygomatic arch, so that the center of the tumor is usually the lobe of the ear. The other salivary glands may swell simultaneously with the parotids, or several days later, even after the parotid tumor has disappeared. Occasionally swelling of the submaxillary or the sublingual glands occurs before that of the parotid, and in rare instances these may be the only glands affected.

As a rule, the parotid of each side is involved. Of 282 cases both sides were affected in 215. When one side alone is involved, it is the left a little more frequently than the right. The interval between the swelling of the two sides may be a week, or even five or six weeks, but usually it is only two or three days.

The salivary secretion is usually very much diminished, and the dry mouth causes great discomfort. Exceptionally, distressing salivation occurs, the secretion amounting to six or eight ounces daily.

Although as a rule the patient is not seriously ill, mumps may in rare cases produce most alarming and even dangerous symptoms. The temperature may for several days reach 104° F. or more, deglutition may be extremely difficult, pressure on the jugular veins may lead to venous hyperemia of the brain, causing headache and sometimes delirium; there

is sometimes great prostration and the symptoms of the typhoid condition. These severe attacks are nearly always in patients over twelve years old.

The constitutional symptoms of mumps usually last from three to five days; the swelling continues on an average about a week. If the case has been a severe one, slight swelling may continue for two weeks or even longer. Relapses, in which the opposite side from the one first affected is involved, are quite frequent, occurring in about ten per cent of the cases.

The blood findings in mumps are quite characteristic. The total leucocytes vary considerably; they may be normal or there may be a leucopenia throughout the disease. There is a constant reduction in the polymorphonuclears and an actual and relative increase in the lymphocytes.

Complications and Sequelae.—In childhood the complications are few and usually unimportant; but in adolescence they are occasionally serious. Orchitis is exceedingly rare in childhood; of 230 cases observed by Rilliet and Barthez, this was seen in but ten, and only three of these cases were in children under fifteen years, and no case in one under twelve years old. When orchitis occurs it is generally toward the end of the second or the beginning of the third week; it is usually marked by an accession of fever, sometimes by a chill; if severe, nervous symptoms may be present. The body of the testicle and not the epididymis is generally affected. The acute symptoms continue for three or four days, and the entire duration of the attack is about a week; although the testicle is often enlarged for some time afterward, and atrophy of the organ may follow. When orchitis is double, sterility may be the consequence.

In females, congestion and swelling of the breasts, ovaries, or labia majora may occur; and, although these complications are all very rare, most of them have been observed even in young children. The interrelation between the parotids and the sexual glands has not yet received a satisfactory explanation.

Nephritis has in a few instances followed mumps, sometimes coming on as late as four or five weeks after the attack. Single cases have been reported by Croner, Isham, Henoch, and others. Nervous sequelae are more frequent, but even these are rare. We have seen multiple neuritis in a boy of twelve which developed two weeks after a severe attack of mumps. The paralysis was general, lasted for six weeks, and was followed by complete recovery. Jaffrey has reported a similar case. Facial paralysis three weeks after mumps has been reported by Hillier, apparently due to an extension of inflammation from the gland to the seventh nerve. Meningitis may occur as a complication of mumps. We have

seen one such case accompanied by high fever, delirium, opisthotonus, and a turbid cerebrospinal fluid containing a great many polymorphonuclear cells. It was, however, sterile. The child recovered after five days' illness.

Pearce has collected an interesting series of forty cases of deafness following mumps, in which there was no sign of otitis, the symptoms coming on suddenly with vertigo, a staggering gait, and often with vomiting. In most of the cases the deafness was unilateral and the loss of hearing was permanent. The cause assigned was disease of the auditory nerve, the seat of the trouble being in the labyrinth. Toynbee has reported an instance of hemorrhage into the labyrinth. Otitis media is rarely seen.

Suppuration of the parotid gland occurs in about one per cent of the cases, and is probably due to accidental infection. Gangrene and sloughing of the parotid were observed twice by Demme in 117 cases; both of these proved fatal. Pneumonia, meningitis, endocarditis, and pericarditis have been observed as complications of mumps, although all are extremely rare.

Prognosis.—In the great proportion of cases mumps is a mild disease, and terminates in complete recovery in a few days. In young children complications are infrequent, and those which occur are rarely severe.

Diagnosis.—Mumps is most likely to be confounded with acute swelling of the cervical lymph nodes. In a parotid swelling, the lobe of the ear is near the center of the tumor, which extends backward to the sternomastoid muscle and forward upon the face as far as the zygomatic arch, embracing the angle and ramus of the jaw.

A swollen lymph node is usually entirely below the ear and behind the jaw, not extending upon the face. The tumor is generally smaller and more circumscribed if only a single node is involved, and it comes on much more slowly than does mumps. When only the submaxillary or sublingual glands are affected, the diagnosis from swollen lymph nodes is sometimes impossible except by the course of the disease. Mumps is characterized by the rapidity with which the swelling occurs, and by its relatively short duration.

Treatment.—The disease is self-limited and the individual symptoms rarely distressing, so that in most cases very little treatment is required. If constitutional symptoms are present the patient should be kept in bed, and if there are none he should be confined to the house. The gland should be protected by flannel or absorbent cotton, and if the pain is severe heat should be applied. The diet should be liquid, on account of the pain produced by mastication. The mouth should be kept clean by the use of some antiseptic mouth-wash. The general symptoms and com-

plications are to be treated according to the indications presented. Cases of mumps occurring in schools or institutions should be quarantined for three weeks, and in private practice where there are susceptible persons. Fumigation and disinfection after an attack are unnecessary.

CHAPTER VIII

DIPHTHERIA

Diphtheria is an acute, specific, communicable disease due to the bacillus of Klebs and Loeffler. It is usually characterized by the formation of a false membrane upon certain mucous membranes, especially those of the tonsils, pharynx, nose, or larynx. Like other pathogenic organisms, however, this germ acts with varying intensity, and may cause inflammation of all degrees of severity, from a mild catarrhal angina to the most serious membranous inflammation; but to all alike the term diphtheria should be applied. In its mild form it may be almost without constitutional symptoms; but in its severe form it is attended by great general prostration, cardiac depression, and anemia; it is frequently complicated by pneumonia and nephritis, and it may be followed by localized or general paralysis; it then constitutes one of the diseases most to be dreaded in childhood.

Etiology.—*The Bacillus Diphtheriae.*—This was first described by Klebs in 1883, and during the following year it was isolated by Loeffler and shown to be pathogenic. It is a Gram-positive bacillus and varies considerably in size and shape even in the same culture. In a specimen it occurs singly or in pairs, sometimes in chains of three or four; the bacilli may lie parallel, but frequently two form an acute or an obtuse angle. They are straight or slightly curved, and sometimes branching; they may be swollen or club-shaped at their ends.

Distribution and Mode of Communication.—In most large cities diphtheria prevails endemically, with periods in which outbreaks of considerable severity are observed. In the country it prevails chiefly as an epidemic. The disease is often introduced into remote districts in some inexplicable manner, and before its nature is recognized a large number of persons may be exposed, and an epidemic results.

Diphtheria does not arise *de novo*. Every case has its origin in a previous case either directly or remotely. The bacilli may enter the body through the inspired air; they may be taken into the mouth with toys or other articles upon which they have lodged, or by kissing, and

sometimes by accidental inoculation. As a rule, the bacilli first gain a foothold upon the mucous membrane of the tonsils, nose, or larynx.

Direct infection is the cause in the great majority of the cases. There is no proof that the bacilli are contained in the breath of a person suffering from the disease. They are present in great numbers in the saliva and mucus from the mouth and nose, often being distributed by sneezing, coughing, or even by talking. They are contained in pieces of membrane which are discharged; they are not present in the feces. In rare instances they have been found in the urine but in such small numbers as to make it very improbable that this is an important source of infection. The most contagious cases are those of pharyngeal diphtheria on account of the amount of discharge which accompanies them. The least contagious are those in which the membrane is limited to the larynx and lower air passages.

Direct infection may occur from persons convalescent from diphtheria, whose throats still contain virulent bacilli, or from persons suffering from a mild form of the disease, which is not recognized as diphtheria. In the latter way it is often spread in schools. It has been repeatedly shown that a person may harbor virulent bacilli in his nose or throat, and may even communicate the disease to others, without himself suffering from diphtheria at any time. Such persons are known as "carriers" and are responsible for spreading the disease to many persons.

The length of time during which a patient with diphtheria may convey the disease to others is somewhat uncertain. Transmission is possible so long as virulent bacilli remain in the throat; these are frequently found two weeks after the membrane has disappeared and the patient is regarded as entirely well, and in a few cases they are found for many months after recovery.

Indirect infection is uncommon. It may occur from dishes, feeding-bottles, or drinking-cups, from swabs and brushes used for local applications to the throat; from spoons and tongue-depressors, and from surgical instruments with which tracheotomy or intubation has been done. It is undoubtedly very unusual for infection to occur from the bed or clothing of a patient, from carpets, toys, books, etc. Diphtheria may be carried by a third person but rarely, except by one who has been in close contact with the patient—either the physician or nurse—and has not taken sufficient precautions. The frequency of diphtheria in physicians' families bears witness to the danger of infection in this manner.

Bacilli may retain their virulence for an indefinite period. Both Park and Loeffler have found cultures in blood-serum to be virulent after seven months; Roux and Yersin, bacilli in dried membrane to be virulent after twenty weeks, and Abel, upon a child's toy after five months.

Domestic animals may in rare instances be carriers of infection, and,

in the case of pigeons at least, they may themselves suffer from the disease. Diphtheria has been repeatedly spread by milk, but very rarely through the contamination of a water supply.

Predisposing Causes.—Local conditions in the throat influence largely the occurrence of diphtheria. An important predisposing cause is the existence of a chronic catarrhal inflammation of the mucous membranes of the nose and throat, frequently found in children suffering from adenoid growths of the pharynx or from enlarged tonsils. These adenoid growths, the tonsillar crypts, and the cavities of carious teeth may harbor the bacilli for a considerable time both before and after an attack. The condition of the mucous membranes of the nose and pharynx in other acute infectious diseases furnishes a marked predisposition to diphtheria. This is most striking in the case of measles and scarlet fever. While diphtheria is seen throughout the year, it is more frequent during the cold than the warm months.

Immunity.—The most important factor which determines if a person who has been exposed is to contract the disease is the presence or absence of immunity. Schick has shown by means of his test (described later) that many persons who have never had diphtheria or received antitoxin, already have antitoxin, or a substance similar to it, in their blood. Those who possess this natural antitoxin are immune to the disease, and even though they may harbor virulent diphtheria bacilli in the throat or nose, they never show any clinical evidences of the disease. This natural antitoxin is possessed by most newly-born infants, only about 7 per cent being without it. Infants gradually lose their immunity; at the end of the first year about 40 per cent, and by the second or third year fully 60 per cent, have lost it altogether and are consequently susceptible to the disease. After four years the incidence of natural antitoxin slowly increases so that at the age of ten or twelve years, only about 25 per cent of children are without protection. These figures, obtained by combining those of Schick and Park, are in accordance with clinical experience. Very few newly-born infants acquire diphtheria, but the number of susceptible children steadily increases with age until about the third year, when it declines. Children from two to six years of age make up the majority of patients in diphtheria hospitals. Those persons who after the first year possess an immunity probably always retain it; while those who at ten years of age do not possess an immunity probably will never acquire it. There is no difference in the sexes in this respect.

The immunity conferred by one attack of diphtheria is not of long duration, amounting probably to a few weeks or months only; the passive immunity conferred by antitoxin is still shorter, lasting but a few days or weeks. Even in patients, therefore, to whom antitoxin has been given, a second attack may occur after a brief interval.

The incubation of diphtheria is short. In most of the cases in which it could be definitely traced it has been between two and five days. The virulence of the bacillus varies much in different cases and in different seasons, and while it is frequently true that persons infected from a mild type of the disease have a mild attack, and those infected from a malignant case a severe attack, there is no certainty that such will be the sequence. Park states that, out of many hundreds tested in the laboratory of the New York Health Department, by far the most virulent bacillus was obtained from the throat of a boy who had what was clinically a very mild form of tonsillar diphtheria.

Lesions.—The essential lesions of diphtheria consist not in the production of a membrane, but, as long ago pointed out by Oertel, in certain acute degenerative changes in the cells of the body caused by the diphtheria toxins. These changes are seen particularly in the epithelial cells of the affected mucous membranes, the heart muscle, the kidney, the liver, the central and peripheral nervous system, the spleen, and the lymph nodes. There are other lesions which are the result of the action of other organisms, especially the streptococcus pyogenes and the pneumococcus, either alone, together, or in conjunction with the diphtheria bacillus. The most important lesions due to these organisms are bronchopneumonia and nephritis; but there may be found in the blood, and in many of the organs of the body, the evidences of the invasion of these bacteria, i. e., a streptococcus septicemia, less frequently a general pneumococcus infection.

Distribution of the Diphtheria Bacillus in the Body.—Unlike many other pathogenic organisms, the diphtheria bacillus is not in most cases widely distributed throughout the body. It is found in great numbers on the surface of the affected mucous membranes and in the false membrane itself, particularly in its superficial portion, but it does not invade deeply the subjacent structures.

The frequency with which the diphtheria bacillus and other organisms are found in the blood and viscera in severe cases is shown in a series of 209 autopsies studied by Councilman, Mallory, and Pearce, of Boston, in 1901. The following table shows the percentage of cases in which the different bacteria were found by culture:

	Heart's blood.	Liver.	Spleen.	Kidneys.
Diphtheria bacillus	6 per cent.	20 per cent.	12 per cent.	19 per cent.
Streptococcus	20 "	30 "	27 "	28 "
Staphylococcus aureus	2.5 "	4 "	3 "	8 "
Pneumococcus	1.5 "	2.5 "	1.5 "	5 "

In this series, 153 were cases of pure diphtheria; 56 were complicated by measles or scarlet fever or both. The streptococcus was much oftener

found in the viscera in the complicated cases; otherwise there was little difference in the two groups of cases.

The Diphtheria Toxins.—The wide-spread effects seen in diphtheria are due to the action of certain substances called *toxins* which the diphtheria bacillus produces during its growth on mucous membranes. They are very diffusible, readily entering the lymphatic circulation and the blood, and through these channels may affect the entire body. In susceptible animals there may be produced by the injection of these toxins all the characteristic lesions of diphtheria except the membrane, as well as the essential symptoms of the disease, even including paralysis. For the production of the membrane living bacilli are required.

Catarrhal Diphtheria.—The routine practice of making cultures from diseased throats has established the fact that catarrhal inflammation may often be the only result of diphtheritic infection. Although to the naked eye there were only the ordinary changes of a simple inflammation, Oertel found the characteristic degenerative changes in the epithelial cells, varying in degree with the severity of the process.

The Diphtheritic Membrane.—The membrane in diphtheria is most frequently seen upon the mucous membrane of the tonsils, soft palate, uvula, pharynx, nose, larynx, trachea, and bronchi: less frequently upon the mouth, lips, esophagus, conjunctivae, middle ear, stomach, and genital organs. It may also affect fresh wounds, notably a tracheotomy wound, or any abraded cutaneous surface. The gross appearance of the membrane varies greatly. It is most frequently yellowish-white or gray, but it may be pearly-white, green, and sometimes almost black. It is composed of fibrin, cells, granular matter, and bacteria. Its consistency varies with the relative proportions of the different elements. When made up chiefly of fibrin it is firm and retains its form, often being discharged as a complete cast of the nose, larynx, or trachea. When the amount of fibrin is small the membrane is soft, friable, and sometimes granular. It is more closely adherent upon the mucous membranes covered with squamous epithelium, as in the pharynx and upper air passages, than upon those covered with columnar and ciliated epithelium, as in the lower air passages.

The microscopical examination shows the fibrin to be sometimes granular, but usually in the form of a network, inclosing in its meshes small round cells and epithelial cells in various stages of degeneration. On the surface and in the superficial layer there is usually found quite a variety of bacteria including diphtheria bacilli. Beneath this is a cellular layer containing little or no fibrin, in which also the diphtheria bacilli are usually found. In the deepest parts of the false membrane and in the mucous membrane itself the bacilli are few in number or absent.

Changes which are similar in all the affected mucous membranes, are found in the epithelial cells which undergo marked degeneration with fragmentation of their nuclei; the mucosa is infiltrated with leucocytes. The infiltration with small round cells is variable in degree in the different mucous membranes; in some it extends deeply into the submucous and even the muscular layers, while in others it is very superficial. Marked evidences of degeneration are seen also in the cells infiltrating the deeper layers. In places the epithelium is detached, in others the line between the false membrane and the granular mucous membrane is scarcely distinguishable.

The Seat and the Distribution of the Membrane.—This varies somewhat with the age of the patient, the season, and the peculiarity of the epidemic.

Our own records show that the larynx is involved in about twenty-five per cent of the cases in children under three years. In general the statement may be made that the younger the child the greater the liability of the disease to attack the larynx. The larynx and lower air passages are rather more frequently attacked in winter than in summer.

The tonsils are the most frequent and usually the earliest seat of the diphtheritic membrane; it may form here a tough, leathery patch, partially or completely covering and very adherent to them; or the disease may affect only the tonsillar crypts, so that the gross lesion may resemble that of ordinary follicular tonsillitis. There is in most cases only moderate swelling, but it may be so great that the tonsils are in contact. The surrounding cellular tissue is infiltrated with inflammatory products.

The membrane covering the pharynx and uvula is also usually very adherent. The uvula is swollen and edematous. Membrane may be seen only upon the fauces and uvula, or the posterior and lateral pharyngeal walls may be covered down to the level of the cricoid cartilage, but generally not below this point. If the posterior pharyngeal wall is covered, the membrane is apt to extend into the rhinopharynx, and even the posterior nares.

The nose may be involved secondarily to the rhinopharynx, or the infection may be through the anterior nares; if the latter, it is not infrequently the only part involved. The membrane in the pure nasal cases is usually thick and tough and often separates *en masse*.

The observations of Councilman, Mallory, and Pearce have shown that it is very common for the accessory sinuses of the nose, especially the antrum of Highmore, to be involved in fatal cases. It seems highly probable that infection of these parts explains the remarkable persistence of diphtheria bacilli in the nose which is occasionally seen.

The epiglottis is swollen to three or four times its normal thickness and the aryteno-epiglottic folds are edematous. The anterior surface

of the epiglottis is rarely covered by membrane; but its lateral borders and posterior surface, and the aryteno-epiglottic folds are involved in most of the severe pharyngeal cases.

The lesions of the larynx, trachea, and bronchi are similar to the above, although much more superficial. The interior of the larynx may be completely covered, the membrane coating the true and false vocal cords and lining the ventricles of the larynx. The membrane in the larynx is not usually very adherent, and it frequently separates and is coughed up in large pieces or even as a cast. That covering the epiglottis and the aryteno-epiglottic folds is very adherent, like that in the pharynx.

In a considerable number of cases the membrane stops abruptly at the lower border of the larynx. In the trachea it is generally loosely attached, and often it is found at autopsy entirely separated from the mucous membrane. It is almost invariably associated with membrane in the larynx. As a rule, the bronchi of both sides are affected, and to the same degree.

The extent of the membrane varies greatly in different cases. It may stop at the bifurcation of the trachea or at the bifurcation of the primary bronchi; but if it goes beyond this point it is likely to extend to the minutest subdivisions. Exceptionally a very tough fibrinous membrane forms in the trachea and bronchi, of sufficient thickness and consistency to be expelled as a cast, reproducing almost the entire bronchial tree.

The buccal cavity is very seldom covered by the membrane; but in the worst cases of pharyngeal disease it may line the cheeks, cover the lips, gums, and more or less of the hard palate, but rarely the tongue. It usually occurs in patches rather than as a continuous membrane. In one case we saw the membrane on the lower lip, extending on to the face, though the buccal cavity was free. It is not common for the diphtheritic membrane to spread down the digestive tract. In 127 autopsies studied by Councilman, Mallory, and Pearce, in which the extent of the membrane was carefully noted, it was found twelve times in the esophagus, five times in the stomach, and once in the duodenum. The accompanying changes consist in infiltration, hemorrhage, and cell degeneration. In the intestines there is often found a hyperplasia of the lymphoid elements—solitary follicles and Peyer's patches—with changes similar to those in the lymph nodes elsewhere in the body, but nothing else that is characteristic.

The writers just referred to found otitis, usually double, in sixty per cent of 144 autopsies; although in less than one-third of the number was the complication recognized during life. Mastoid disease is infrequent. Otitis is usually the result of direct extension from the pharynx.

It may be due to the diphtheria bacillus, to the streptococcus, or to both combined. Conjunctival diphtheria is rare and probably due to accidental infection rather than to extension through the lachrymal duct. Before the advent of antitoxin, it almost invariably resulted in destruction of the eye; but many cases successfully treated have been reported. Diphtheria may attack any mucocutaneous surface, especially the anus, prepuce, or female genitals; any abraded cutaneous surface, or recent wound, most frequently the tracheotomy wound of the neck. The diphtheria bacilli have in rare instances been found in pure culture in superficial abscesses.

Visceral Lesions.—The visceral lesions of diphtheria are due partly to the action of the diphtheria toxins and partly to the invasion of the body with other organisms, especially the streptococcus. It is to experimental diphtheria that we owe our most accurate knowledge of the former changes, for in human diphtheria the large proportion of all the fatal cases show infection with other organisms.

The visceral lesions of diphtheria consist in wide-spread areas of cell degeneration similar to those which have already been described as occurring in the epithelial cells of the affected mucous membranes, together with hemorrhages due to changes in the blood-vessels and possibly in the blood itself.

The lymph nodes of the cervical region are the most constantly and the most seriously affected. Similar but less marked changes are seen in the tracheobronchial and the mesenteric groups, and in the lymph nodules of the mucous membrane of the stomach and intestine. There are degenerative changes in the cells of the nodes most affected, with marked infiltration with leucocytes and frequently small hemorrhages. The cellular tissue in the neighborhood of the cervical nodes is often extensively infiltrated with cells. The process in the lymph nodes usually terminates in resolution, rarely in suppuration.

The spleen is swollen, sometimes very much so, and deeply congested. Hemorrhages are often seen beneath the capsule; the spleen pulp is soft, the follicles are large, and cell degeneration is quite constantly observed similar to that which takes place in the lymph nodes.

There are frequently small hemorrhages beneath the capsule of the liver, and sometimes these are seen throughout the organ. There are found scattered through the liver, areas of necrotic hepatic cells; some of these areas are infiltrated with leucocytes.

The kidneys are involved in almost all fatal cases except when death occurs early from laryngeal stenosis, also in nearly every severe case which terminates in recovery. Acute degeneration of the epithelium of the tubes and the tufts is seen in less severe cases and those of shorter duration, and is the direct result of the action of the toxins. In the

more severe and protracted cases there is acute diffuse nephritis of variable type and intensity.

In children dying suddenly in the early stage of the disease, cardiac thrombi are occasionally found. They may form rapidly only a short time before death, or slowly during several days when the circulation is very feeble. Portions of these thrombi may be carried into the pulmonary or systemic circulation, causing embolism in any of the arteries of the extremities, the lungs, or other viscera. Even in the early fatal cases the heart muscle may be seriously affected; in the later ones this is almost constant. The changes consist in a toxic myocarditis, the left ventricle being most involved. (See Myocarditis.)

Degeneration of the arteries, especially of the endothelial layer, is occasionally seen, and there may be infiltration of the adventitia.

Lesions of the brain are rare; both hemorrhage and embolism may be met with. In the spinal cord and membranes multiple hemorrhages occasionally occur. The chief lesion, however, consists in degenerative changes which are found to some degree in nearly all the more severe cases which have been examined. These affect the ganglion cells of the anterior horns, the anterior and posterior nerve-roots, and sometimes the pyramidal tracts and columns of Goll. Some writers are of the opinion that the cord lesions are primary and the degeneration of the spinal nerves secondary. However, the general opinion prevails that certainly the less severe cases of diphtheritic paralysis are due to peripheral rather than to central lesions. Degenerative changes have been found also in the pneumogastric, spinal accessory, hypoglossal, motor-oculi, and in the cardiac nerves. These nerve degenerations produced by the diphtheria toxin constitute one of the most striking lesions of diphtheria. (See Multiple Neuritis.)

In infants and young children bronchopneumonia is found at autopsy in fully three-fourths of the cases. It is well-nigh constant in cases of diphtheritic bronchitis of the finer tubes, and is usually present where the membrane has extended to the bifurcation of the trachea. The largest factor in the production of pneumonia is the aspiration of diphtheria bacilli and streptococci from the upper air passages.

With laryngeal stenosis, some emphysema is invariably present, and usually it is of the vesicular variety. Rupture of some of the larger blebs may lead to the escape of air into the cellular tissue of the mediastinum or of the neck, which may result in the production of a general emphysema of the subcutaneous cellular tissue.

Blood.—There is found in all severe cases of diphtheria a reduction in the number of red cells to the extent of 500,000 to 2,000,000. There is a nearly proportionate reduction in the hemoglobin, this amounting to from 10 to 30 per cent. While the hemoglobin falls coincidently with

the number of red cells, it is regained much more slowly. Leucocytosis is generally present, and usually proportionate to the severity of the attack, but is occasionally wanting in the most severe as well as in some of the very mildest cases. The increase in the leucocytes is in the polymorphonuclear forms. Engel has noted the frequent presence of myelocytes, especially in fatal cases, the proportion of these in some instances reaching sixteen per cent of the white cells.

Symptoms.—The clinical picture of diphtheria is one which presents wide variations, depending upon the principal location of the disease, its severity, and its complications. For practical purposes the following seems the simplest grouping that can be made:

1. The mild cases, in which there is either no membrane, or the amount of membrane is small and limited to the tonsils or to the nose, with few or none of the constitutional symptoms which follow absorption of the diphtheria poison. These cases partake essentially of the character of a local disease.

2. The severe cases in which there are marked evidences of constitutional poisoning from the diphtheria toxin. This form is usually accompanied by an extensive formation of membrane in the pharynx and sometimes in the nose.

3. The laryngeal cases in which the larynx may be primarily or alone affected or in which it is involved secondarily to the severe pharyngeal form.

4. The malignant cases. In these cases the symptoms of inflammation are especially prominent, not only in the pharynx but sometimes in the lymph nodes and cellular tissue of the neck, which may be followed by suppuration or sloughing. This form is frequently complicated by bronchopneumonia even without laryngeal disease, and sometimes by severe nephritis.

Cases without Membrane.—During an epidemic of diphtheria in a family or an institution, cases are frequently seen which present the clinical evidences of only a catarrhal inflammation of the nose or pharynx, and yet cultures show the presence of the diphtheria bacillus. Such cases may be examples of simple catarrhal inflammation with the accidental presence of the diphtheria bacillus; or the inflammation may be caused by infection with the diphtheria bacillus, but not of sufficient intensity to lead to the production of a membrane. The latter is the view of pathologists, and the one to which clinicians must, it seems, inevitably come.

Catarrhal diphtheria may be either pharyngeal or nasal. In the pharyngeal cases there are present the usual appearances belonging to a catarrhal inflammation of moderate severity, often accompanied by swelling and tenderness of the cervical lymph glands.

The nasal cases, in our experience, have been most frequent in infants or very young children. Constitutional symptoms may be wanting or so slight as to be overlooked. The only striking thing is a persistent nasal discharge which may be serous and frothy, purulent or bloody. It is usually copious, often excoriating the upper lip and sometimes continuing for three or four weeks before any other symptoms are observed. We have several times known it to be mistaken for a syphilitic coryza. Such cases can be recognized with certainty only by cultures. Clinical evidence of their true character is sometimes afforded by the appearance of visible membrane in the nose or pharynx, by the development of croup, or by the fact that they cause diphtheria in other children. The bacilli are non-virulent in quite a large proportion of these cases, but in others they are of extreme virulence.

Catarrhal diphtheria is not in itself serious, but it may be followed, particularly in young children, by laryngeal diphtheria, or pharyngeal diphtheria may develop in its usual form.

Cases with a Small Amount of Membrane.—Tonsillar Diphtheria.—The exudation is usually limited to the tonsils and may partake of the character of either follicular or croupous tonsillitis; sometimes there is a slight extension to the faucial pillars or to the pharynx. These cases are quite common, and are more frequent in older children and adults than in infants and young children.

The onset is accompanied by a little soreness of the throat; the initial temperature is from 101° to 103° F.; but the symptoms are often not severe enough to keep the patient in bed. If seen early, the throat shows slight redness, followed by a gray film, and later by a gray or white deposit upon the tonsils. This may start as a small patch which enlarges, or as small, isolated spots which coalesce or remain separate. The membrane is quite adherent; and can not easily be removed with a swab; usually it is sharply defined. In many cases the patch is not larger than the finger nail. The inflammatory changes in the pharynx are slight; a faint red areola is present at the border of the patch. The lymph glands behind the jaw may be slightly swollen. There is no nasal discharge and very little increase in the saliva or mucus from the pharynx. Some constitutional symptoms are present, but they are not severe. The temperature commonly continues above the normal while the membrane lasts, its usual range being from 100° to 102° F. The membrane remains from three to seven days—a shorter time if antitoxin is used. It is very often a matter of surprise that so small an exudate is so persistent. The urine is generally normal. The parents are loath to believe that strict quarantine is necessary in so mild an illness; and when the membrane is only upon the tonsils, even after the disease has run its course, the physician may be led to doubt the diagnosis of diphtheria.

In many cases one with experience can usually make an accurate diagnosis from the clinical symptoms alone; but there are many others in which the diagnosis from ordinary tonsillitis is impossible, except by cultures. When diphtheria bacilli are found in these mild cases the question often arises whether they may not be the non-virulent form. Park tested forty such cases, and found the bacilli to be virulent in thirty-five and non-virulent in five. In twenty of the forty cases the clinical diagnosis was follicular tonsillitis.

Severe Cases.—The clinical picture of diphtheria is so modified by the use of antitoxin that those who see it given regularly and early can have but little conception of the horrors of this disease when not thus influenced. The onset in severe cases may be gradual, even insidious. There is then a slight indisposition for a day or two, and perhaps some soreness of the throat; the temperature may be but little elevated, sometimes less than 100° F. The symptoms may steadily increase in intensity for four or five days, until the maximum is reached. At other times the disease begins abruptly with vomiting, headache, chilly sensations, and a temperature of 103° or 104° F. Occasionally, the first thing to attract attention is the swelling of the cervical lymph nodes, which may be so great that mumps is suspected. The abrupt onset is more often seen in young children than in those who are older.

The membrane upon the tonsils resembles that of the mild form previously described, but, instead of remaining limited to them, it gradually spreads to the fauces, the lateral wall of the pharynx, the uvula, the rhinopharynx, and the posterior nares. In some cases it may cover all the parts mentioned in twenty-four hours from its first appearance; in others this may require several days. When the nose is first affected there is an abundant discharge of serum and mucus, occasionally tinged with blood, which may continue some days before any membrane is visible.

When a severe case is fully developed there is a very abundant discharge of mucus from the mouth and nose. The tonsils, the entire faucial ring, and the pharynx are covered with membrane which is at first gray and gradually becomes darker, often being of a dirty olive-green color. There is obstruction to nasal respiration from the swelling of the palate, the tonsils, and the tissues of the rhinopharynx; the mouth is half open, the breathing noisy, the tongue dry, and the lips are fissured and bleed readily. Occasionally large nasal hemorrhages occur which may necessitate plugging the nares. Both nostrils are generally blocked by the swelling and the false membrane; the discharge excoriates the upper lip, and frequently has a fetid odor. During the second week there may be regurgitation of fluids through the nose, owing to paralysis of the palate. The lymph glands at the angle of the jaw swell rapidly;

in severe cases they are very prominent, and there may also be extensive infiltration of the cellular tissue about them.

The constitutional symptoms usually increase steadily with the extension of the membrane. In the most severe cases the system is overwhelmed with the poison, and all the evidences of intense toxemia are present by the third day of the disease. This is shown by great muscular weakness and prostration, by a feeble, rapid pulse, and a mental state of complete apathy or stupor, sometimes alternating with great restlessness. The pulse becomes rapid, weak, and compressible, sometimes irregular; the heart sounds are faint and there is a great and steadily increasing anemia. The course of the temperature is irregular, and may bear no constant relation to the severity of the other symptoms. Its usual range is from 101° to 103° F., but in some of the worst cases it may never go above 101° F. It fluctuates irregularly with the development of complications, and sometimes without apparent cause. By the second or third day the urine regularly shows the presence of albumin, and by the end of the first week the quantity is often large. Granular and hyaline casts, and occasionally blood in small quantities, are also found. The amount of urine secreted is not noticeably diminished, and dropsy is rare. Nervous symptoms are seen in all the very severe cases. There may be dulness and apathy, but more frequently, owing to the discomfort arising from local symptoms, there is extreme restlessness and excitement, sometimes followed by delirium.

At any time during the first week, but not often after that time, symptoms may arise indicating that the disease has extended to the larynx. The first signs of laryngeal invasion usually appear from the second to the fifth day of the disease. These are at first hoarseness, a croupy cough, and slight dyspnea. In the severe cases these symptoms steadily increase until all the signs of laryngeal stenosis are present.

The local process in the pharynx seems to be a self-limited one, even when no antitoxin is used. It usually reaches its height by the fifth or sixth day, and after that the appearances do not change materially for two or three days. From the seventh to the tenth day, in favorable cases, the diphtheritic membrane begins to loosen and separate from its attachment. It hangs loosely from the palate or uvula, and can often be pulled away in large masses. The detachment is frequently rapid, and in two or three days from the time when the first improvement is seen, the tonsils and pharynx may be almost free from membrane. The mucous surface left behind is of a bright-red color and bleeds easily. The separation of the membrane in the nose and rhinopharynx takes place more slowly. From the former it may disintegrate gradually or come away *en masse*. With the disappearance of the membrane the local symptoms abate rapidly—the discharge ceases, the swelling of the lymph

glands subsides, deglutition becomes easy and natural, and nasal breathing is re-established. When antitoxin is given the local process passes through similar stages, but much more rapidly.

Simultaneously with these changes in the throat the constitutional symptoms improve, but much more slowly. Convalescence is often protracted. The anemia and muscular weakness, and most of all the feeble heart action may persist for weeks. Symptoms due to myocarditis may appear in the second or third week or even later. (See Myocarditis.)

Instead of the usual course just described, the diphtheritic membrane may persist for two or three weeks. In rare cases relapses occur, the membrane forming again after it has entirely or partially disappeared.

The early course of the disease in the fatal cases often does not differ from that of the severe cases which end in recovery, except in the malignant form, which kills in twenty-four or forty-eight hours, and which is rare. In very young children death is most frequently due to bronchopneumonia, usually accompanying diphtheria of the larynx and bronchi. It may also be due to progressive asthenia, the result of diphtheritic toxemia, or to heart failure.

Laryngeal Diphtheria.—In cases of primary laryngeal diphtheria there are wanting most of the characteristic clinical features which distinguish diphtheria of the pharynx. There are two reasons for this: one is the relatively rapid course of the disease, often producing death from local causes before the constitutional symptoms resulting from the absorption of the toxin have developed; the second reason is, that absorption of the poison by the laryngeal mucous membrane is very feeble as compared with that which takes place from the pharynx. Hence it follows that glandular enlargements, albuminuria and asthenic symptoms are generally wanting; also, that in the cases which come to autopsy early, the parenchymatous degenerations of the heart, kidney, and other organs are seldom found, but instead only such lesions as are connected with the laryngeal disease. The feeble contagion is due to the fact that the course is much shorter, and that the discharge from the nose and mouth is slight, or absent altogether.

In its onset, diphtheria of the larynx is indistinguishable from catarrhal inflammation. It is usually somewhat less abrupt, and apparently not quite so severe for the first twelve hours or even for a longer time. There are present the same hoarse cough and voice, with slight stridor, gradually increasing. The constitutional symptoms are usually not quite so marked, the temperature ranging from 99° to 101° F. The pulse is accelerated, but not weak or intermittent. It is the progress of the disease which indicates its character, usually during the first twenty-four hours. A child beginning in the morning with such symptoms as have been described, may by evening show a decided change for the

worse, or the symptoms may increase with great rapidity during the night. At first the voice is hoarse; later it is entirely lost. Dyspnea in the beginning is scarcely noticeable, but steadily increases hour by hour. Sometimes, from the first sign of hoarseness to such extreme dyspnea as to necessitate intubation may be but a few hours. During the second twenty-four hours all the symptoms are usually well developed. The respiration is often somewhat accelerated, but it may be slower than normal. The face is pale and anxious. The alae nasi dilate with each inspiration. The loud, "sawing," stridulous breathing is present, indicating obstruction both to inspiration and expiration. As the dyspnea increases, all the accessory muscles of respiration are brought into action. There is now with every inspiration deep recession of the suprasternal fossa, the supraclavicular regions, and the epigastrium. The child tosses uneasily from side to side in his crib, at times struggling violently to get more air into the lungs. The pulse grows rapid and weaker. There is slight blueness of the finger nails and the lips; the face is usually pale; but later this too may be cyanotic. The skin is covered with clammy perspiration. On auscultating the chest, very rude respiratory sounds are heard, but no vesicular murmur. As the symptoms increase in severity the temperature usually rises gradually, in some very severe cases at the rate of a degree an hour, until shortly before death it reaches 104° or even 106° F. Late in the disease the intellect becomes dull, the violent struggles for air cease, and the child passes into a condition of semi-stupor which gradually deepens until death occurs, which may be preceded by convulsions.

Such is the usual course of the disease when unrelieved by treatment. Its progress is most rapid in infants, in whom death usually takes place in from thirty-six to forty-eight hours from the first symptoms. In older children the course is rather slower, and the attack may last from two days to a week, death occurring more frequently from bronchial croup or pneumonia. They are indicated by continued high temperature, rapid respiration, cyanosis, and increased prostration.

The course of the disease is not always so regular. Occasionally for a week or more the symptoms are precisely like those of catarrhal laryngitis of moderate severity—hoarseness, laryngeal cough, little or no fever, and slight or occasional dyspnea. Then there may be the sudden development of very severe symptoms, and death in a few hours. Great improvement may follow the dislodgement of the membrane by vomiting or coughing, although in most cases it forms again.

The issue of every case of diphtheritic laryngitis is doubtful. The prognosis is worse in infants and very young children than in those over three years of age. Before the days of antitoxin the mortality of cases not operated upon was from eighty to ninety per cent. Even with mod-

ern methods of treatment the outlook in infants under a year is bad; fully forty per cent die.

It may be difficult in a given case to decide whether the dyspnea is due to laryngeal inflammation, and whether this inflammation is catarrhal or diphtheritic. The dyspnea of retropharyngeal abscess, of foreign bodies in the larynx or trachea, or of bronchopneumonia, may be mistaken for that due to laryngitis. But in none of these conditions should there be any doubt if a careful examination is made and a history obtained. Retropharyngeal abscess may be recognized by digital examination of the pharynx; bronchopneumonia by the signs in the lungs, the difference in the character of the dyspnea, and especially by the absence of the noisy stridor; in the case of foreign bodies, whether they enter through the mouth or consist of ulcerating caesous glands which have ruptured into the trachea, the dyspnea comes suddenly, and is not accompanied by fever. The main points by which catarrhal laryngitis is distinguished from the diphtheritic form have been considered under the former disease. In brief, diphtheritic inflammation may be assumed if there is severe, constant, and increasing dyspnea with aphonia.

Malignant Diphtheria.—The symptoms are usually severe from the outset. The exudation in these cases may be of a yellow, dirty-gray, or olive color, sometimes being almost black from the presence of blood. The membrane is usually extensive, covering the entire pharynx, often extending to the nose and the middle ear, and occasionally spreading to the buccal cavity. There is great swelling of the tonsils and uvula, and it is often impossible to obtain a view of the pharynx. Sometimes the inflammation is of a necrotic character, and there may be extensive sloughing of the tonsils, the uvula, or the soft palate. The nasal discharge is generally abundant, and often offensive. There is marked swelling of the cervical lymph glands, and frequently extensive infiltration of the cellular tissue of the neck, so that the head is thrown back to relieve the pressure upon the larynx and trachea. The swelling sometimes forms a distinct collar, reaching from ear to ear and filling out the whole space beneath the jaw. The pressure upon the jugular veins leads to congestion and swelling of the face and congestion of the brain.

The temperature is usually high; it follows no regular course, but generally fluctuates widely from 102° to 106° F. In some cases, however, it may never be above 101° F. In the form characterized by very high temperature there is sometimes found a general streptococcus or pneumococcus infection, usually the former. The pulse is weak, rapid, and compressible. The peripheral circulation is poor, the extremities are often cold, there is extreme muscular prostration, and both vomiting and diarrhea are frequent. There may be excitement, restlessness, and active delirium, or dulness, apathy, and stupor. Nephritis is very frequent and

is often severe; the urine contains a large amount of albumin and casts of all varieties, but rarely blood. In a large proportion of the children under three years old bronchopneumonia develops. Severe symptoms continue for from two days to a week; the patient may die from the sudden invasion of the larynx, or there may be suppression of urine and uremic convulsions; but more frequently the cause of death is circulatory failure or bronchopneumonia. Death usually occurs while the local disease is at its height. Occasionally it comes later from myocarditis after the signs of local improvement have begun. Evidences of myocarditis are present post.mortem in nearly every case.

Those who manage to escape the dangers of the acute period have still others to encounter. Among the latter may be mentioned, extensive sloughing in the throat or of the cellular tissue of the neck, which may be followed by severe or even fatal hemorrhage, diffuse suppuration of the same region, late nephritis, pneumonia, or pleurisy, and finally paralysis of the heart or respiration.

Complications and Sequelae.—Most of the complications of diphtheria have already been mentioned either under the head of Lesions or Symptoms. It only remains to consider their clinical association.

Otitis occurs particularly in the rhinopharyngeal cases, and is sometimes due to the diphtheria bacillus alone, but more often to mixed infection. The type of inflammation is often a severe one, and it may be accompanied by necrotic changes in the drum membrane which resemble those of scarlet fever.

Bronchopneumonia is the most frequent complication in young children. It occurs especially in laryngeal cases, and in those of a severe type whether the larynx is involved or not. Other pulmonary complications are infrequent. Emphysema is a complication of laryngeal diphtheria; it is nearly always vesicular, rarely interstitial. It may become general, extending into the cellular tissue of the neck and afterward that of the entire body.

Pericarditis, endocarditis, and meningitis are all rare and are seen chiefly in septic cases of the most severe type. Myocarditis is much more frequent, and is present to a greater or less degree in nearly all severe cases. It usually causes no distinctive symptoms but can be detected by physical examination. Heart block has been described in the course of and following diphtheria, but is rarely permanent. It is to be referred to a lesion of the bundle of His.

Thrombosis and embolism are among the less frequent complications. If cerebral, they may cause hemiplegia, aphasia, and sometimes convulsions; if peripheral, they usually affect one of the lower extremities, where they may cause sudden pain, numbness, and coldness of the limb, followed by partial paralysis, edema, and sometimes even by gan-

grene. Thrombosis of the pulmonary artery or of the heart may be a cause of sudden death.

Hemorrhages are usually nasal, and while in most cases they are not serious, they may necessitate plugging of the posterior nares. Bleeding from any other mucous membrane may occur, but it is rare except from the mouth. Subcutaneous hemorrhages are infrequent, and are evidence of a very high degree of diphtheritic toxemia. They usually occur as small petechial spots, but are sometimes extensive. They may be seen upon almost any part of the body, most frequently upon the abdomen and lower extremities; but the most extensive extravasation we have ever seen was in the neck, reaching from the clavicle almost to the ear and covering nearly one lateral half of the neck.

Albumin is present in the urine of almost every case of moderate severity, usually depending upon acute degeneration of the kidneys. Acute nephritis is most frequently seen in severe cases. It then usually develops at the height of the local disease, but may come during convalescence. Chronic nephritis very infrequently follows diphtheria.

Diarrhea is of frequent occurrence. There may be no intestinal lesion or ileocolitis may be present, which, however, seldom goes on to ulceration. It is extremely rare that the membranous form of ileocolitis is seen, and then it is associated with the presence of other organisms than the diphtheria bacillus.

Diphtheria is usually followed by a severe and often persistent anemia which may continue for weeks. Pneumonia, nephritis, and cardiac disease may first show themselves during convalescence, and so be ranked as sequelae. The most important sequel of diphtheria, however, is post-diphtheritic paralysis, already discussed in the chapter on Multiple Neuritis.

Pneumogastric Paralysis.—Some cases of diphtheria, especially those which receive no antitoxin or when the antitoxin is administered late or in too small amount, present a group of symptoms which have been referred to degeneration of the pneumogastric nerves. The evidence, however, is by no means conclusive that this is the true explanation of the clinical picture, which is a familiar one.

These symptoms may come on at any time in the course of the disease, but seldom earlier than the end of the second week. By this time the throat has usually cleared off entirely, and the patient is considered convalescent. The symptoms relate to the stomach, the heart, and the respiration. Usually the first thing to attract notice is that the patient refuses food and vomits occasionally, afterward persistently, without apparent cause. If the pulse is carefully observed it is found to be much slower than previously, being only 80 or 90 when it was formerly 120 or more. It is also weaker, compressible, and often somewhat irregular.

The face is pale or slightly cyanotic, and moderate dyspnea may be noticed. There are frequent attacks of severe abdominal pain which comes in paroxysms, and is usually referred to the epigastrium. These symptoms in most cases gradually increase in severity for two or three days, but sometimes develop with such intensity that death occurs within twelve or twenty-four hours. The later symptoms are a continuance of the abdominal pain and vomiting; there is a feeling of great precordial oppression and distress accompanied by dyspnea; the respiration is shallow and often rapid; the face is either pale or cyanotic; the extremities, cold; the pulse, slow, irregular, and intermittent, becoming rapid on the slightest exertion. The heart sounds are weak, the muscular quality is absent, and the rhythm much disturbed. There may be no murmurs. There is great restlessness, but the mind is entirely clear. Death usually results from heart failure, which may come quite suddenly, often from so slight exertion as turning over in bed or attempting to take food.

Not all the cases are so severe. In the milder forms there is some palpitation, an irregular pulse, slight dyspnea, and occasional syncopal attacks, but of no great severity. Such symptoms may come and go for several days and then disappear; but more frequently they prove to be the beginning of the more serious form of the complication. The time of occurrence of these symptoms varies considerably. It may be as late as the third or fourth week. The late cases are generally associated with some other form of postdiphtheritic paralysis.

Sudden heart failure may be seen late in diphtheria quite apart from the symptoms just described. It may occur with few or no premonitory symptoms; as when a child falls dead after walking across a room, or suddenly sitting up in bed, or from some other muscular effort, or possibly as a consequence of passion or excitement. We knew of one little girl who was considered well enough to go coasting and who died suddenly after the effort.

The explanation of heart failure during or after diphtheria is therefore not always the same. When it occurs at the height of the disease it is sometimes due to cardiac thrombosis, probably always associated with changes in the muscular walls. When it occurs late and follows some sudden muscular effort or excitement without premonitory symptoms of any sort, it is probably the result of changes in the muscular walls—a true myocarditis.

Diagnosis.—The diagnosis of diphtheria rests upon two kinds of evidence—clinical and bacteriological. In mild cases and in the early stage only bacteriological evidence can be relied upon. However, the clinical manifestations of the disease are important and should not be ignored. It is in most cases possible to say from clinical symptoms that a case is one of diphtheria; but it is never possible to say from symptoms

alone that a case is not one of diphtheria. Cultures, therefore, should, if possible, be made in every case. They are necessary in the mild cases in order that a correct diagnosis may be made and proper quarantine regulations enforced.

The mere presence of diphtheria bacilli in the throat does not prove that a person has diphtheria any more than the presence of the pneumococcus in his saliva proves that he has pneumonia; but when diphtheria bacilli are associated with clinical evidences of inflammation of the throat or nose the diagnosis may be regarded as established. Again, the case may be one of diphtheria and the bacilli not found at the first examination, although found subsequently. In using antitoxin one must, in perhaps the majority of cases, be guided by clinical symptoms alone, not waiting for the result of the bacteriological examination. It is therefore important that both methods of diagnosis should be employed.

1. The Clinical Diagnosis.—Not much importance can be attached to the mode of onset; for diphtheria may begin in many different ways. The presence of a nasal discharge, especially if abundant, ichorous and tinged with blood, the early development of the symptoms of croup, and the rapid enlargement of the cervical lymph nodes, all point strongly to diphtheria. Later symptoms which are especially diagnostic are marked anemia, progressive asthenia, very feeble pulse which is sometimes slow, sometimes rapid, sudden attacks of syncope, nasal regurgitation from paralysis of the soft palate, contagion, and, finally, the development of paralysis of the muscles of the throat, eye, or extremities, with paralysis of the heart or respiration.

The membrane of diphtheria generally appears first upon the tonsils, usually as a gray film which gradually becomes more dense and white, and often has the look of being plastered on. The color of older membrane is gray, greenish-yellow, brown, sometimes black. Beginning as a small patch, it soon covers the tonsils. It frequently affects one tonsil twenty-four or thirty-six hours before the other, and occasionally it is confined to one side. In exceptional cases it begins in the crypts of the tonsil and appears as isolated dots, which may coalesce to form a continuous patch like that already described, or it may remain isolated like the exudate of an ordinary follicular tonsillitis. More important is the fact that the membrane spreads from the original seat, and also the manner of its spreading. If it extends beyond the tonsils to the walls of the pharynx, the faucial pillars, and the uvula, it is almost surely diphtheria. The same is true of doubtful patches on the tonsils or fauces followed by symptoms of croup. The rapidity of the spreading varies much in the different cases, but the gradual extension, as shown by observations made at intervals of six or eight hours, usually settles the diagnosis in the primary cases. However, if the throat symptoms complicate

measles or scarlet fever the above rules do not apply. Most of the membranous inflammations of the throat seen in these diseases are not due to diphtheria. This is particularly true of those which occur at the height of the primary disease. Those which develop at a later period are often due to diphtheria.

Primary membranous inflammation of the larynx may always be safely regarded as diphtheria; but if there is no visible membrane, the diagnosis is rendered positive only by a bacteriological examination. This may be true of many nasal cases where the only symptoms are a discharge of the character previously described. Such cases may continue for weeks with no symptoms other than the discharge, especially in infants.

It is seldom difficult to distinguish diphtheria from other diseases; but the exudation upon the pharynx or tonsils may be confounded with thrush or ulceromembranous angina. The appearance of the tonsils on the second or third day after tonsillotomy has been performed, may easily be mistaken for diphtheria by one who is unfamiliar with the appearance of the postoperative wound.

Diphtheria of the mouth may be mistaken for herpetic or ulcerative stomatitis; but, as a rule, it is seen only in the worst cases of pharyngeal diphtheria. Diphtheria of the mouth alone is so rare that it may be ignored.

It is sometimes difficult to distinguish cases of scarlet fever in which the throat symptoms are severe and appear early, from cases of primary diphtheria. In many of these cases the eruption appears late, and is not characteristic. Much importance is to be attached, as pointing toward scarlet fever, to a prevailing epidemic, a history of exposure, a sudden onset with severe symptoms, vomiting, prostration, very high temperature, and to a very active inflammation in the pharynx. In all cases with a sudden onset, in which from the throat symptoms one is inclined to make a diagnosis of diphtheria, the possibility of scarlet fever should not be forgotten, and one should never omit to examine the patient thoroughly for an eruption.

2. The Bacteriological Diagnosis.—*The Technic.*—In many cases an immediate diagnosis may be reached by the examination of a cover-glass smear from the throat. This method, although often valuable, is not adapted for general use, as bacilli directly from the throat are much less typical than those from cultures, and the chances of contamination are much increased. Furthermore, the mouth often contains other bacilli which somewhat resemble the diphtheria bacillus.

In taking a culture from the throat nothing but the membrane should be touched and this should be rubbed firmly with a swab, which is then rubbed over the surface of the culture-medium. In laryngeal cases the

culture should be taken from the posterior wall of the pharynx, and in nasal cases from the nostril.

The Reliance to be Placed upon Bacteriological Diagnosis.—The diphtheria bacillus will almost invariably be found, if there is visible membrane in the pharynx, if no antiseptics have been applied shortly before using the swab, and if the culture has been made with sufficient care to avoid contamination.

The diphtheria bacillus sometimes disappears early; hence cultures made while the membrane is loosening may be negative. If the membrane has disappeared, or if none has been present, it is not infrequently necessary to obtain material from the tonsillar crypts in order to discover bacilli. It is therefore important in all cases to consider the duration of the disease before drawing a conclusion from a negative culture. In cases of laryngeal disease without pharyngeal exudation, an early culture is negative in nearly half the cases; although a little later bacilli may be coughed up and found in the pharynx in abundance. A single negative culture should never be taken as conclusive.

For diagnostic purposes, all bacilli present in suspicious throats, having the morphological and cultural characteristics of diphtheria bacilli, are to be regarded as virulent.

Non-virulent Bacilli Resembling the Diphtheria Bacillus.—There may be found in throats a form which corresponds in every other characteristic with the diphtheria bacillus, but which lacks virulence, as shown by animal tests. Also, another form, which, though in many particulars resembling the diphtheria bacillus, differs from it in being shorter, plumper, and more uniform in size, and in producing an alkali in broth cultures; to this the term *pseudo-diphtheria bacillus* has been given. It is more frequently seen than the form just described and like it is non-virulent. Both these forms are rare in throats where a suspicion of diphtheria exists.

The Presence of Virulent Bacilli in the Throats of Healthy Persons.—That virulent bacilli may be harbored for an indefinite period in the throat or nose of a healthy person is proved by many observations. The New York Health Department made observations upon forty-eight children in fourteen families in which one or more cases of diphtheria had occurred, and where no attempt at isolation had been made. In one-half these cases bacilli were found, and animal tests showed them to be virulent in every one of six cases tested, although four of the children did not develop diphtheria. Of the entire number, forty per cent subsequently developed diphtheria. Our own experience in two institutions where diphtheria has been endemic, fully confirms the observation that bacilli of all degrees of virulence are very frequently found in the noses or throats of exposed children, although a large proportion of them

never develop the disease. Outside of institutions and infected tenement houses, however, such a condition is much less common. Moss and Guthrie took cultures from 1,217 public school children in Baltimore. In 44 children diphtheria bacilli were found, but in only eight were they virulent.

Prognosis.—Many possibilities exist, and even the mildest case must be regarded as serious and carefully watched, since one can never know when unfavorable symptoms may develop.

The factors to be considered in the prognosis of any given case are: the age and previous condition of the patient; the extent of the membrane and the rapidity with which it is spreading; the degree of diphtheritic toxemia as shown by the condition of the pulse and the nervous symptoms; whether or not the membrane has invaded the larynx; and the presence or absence of complications, especially nephritis and bronchopneumonia; but of more importance than any or all these things is whether antitoxin is used and when it is administered.

The following figures are from the Report of the Health Department of Chicago of cases treated for a series of years.

		Died.	Mortality.
Injected 1st day	355	1	0.27 per cent.
" 2d day	1,018	17	1.67 " "
" 3d day	1,509	57	3.77 " "
" 4th day	720	82	11.39 " "
" later	469	119	25.37 " "
Totals	4,071	276	6.77 " "

In all these cases the diagnosis of diphtheria was confirmed by cultures.

Diphtheria mortality is highest during the first two years of life, from its strong tendency to invade the larynx and lower air passages, and from the frequency with which bronchopneumonia occurs as a complication. Those whose experience with this disease does not antedate the introduction of antitoxin can scarcely appreciate the results previously obtained. Of eighty-five consecutive cases under twenty-six months of age observed in the New York Infant Asylum, in a period extending over two years, the mortality was sixty-eight per cent; in over two-thirds of the fatal cases the disease involved the larynx. In diphtheria hospitals, where most of the mild cases included in the above statistics would probably not have been admitted, the mortality in children under two years formerly varied from sixty to eighty per cent; in private practice it ranged for this age from thirty to sixty per cent.

It can not be too often emphasized that the danger from diphtheria is not over when the throat has cleared. The most frequent causes of death after this time are bronchopneumonia and cardiac paralysis.

Prophylaxis.—In no infectious disease, smallpox alone excepted, can so much be accomplished in the way of prevention as in diphtheria.

Public funerals of children dying from diphtheria should invariably be prohibited. Schools should be closed whenever the disease is epidemic. Children from families where diphtheria exists should not be allowed to attend school, nor mingle in any way with other children, for the reasons that they may, while healthy, be the carriers of the disease; and, what is even more important, that they may be themselves suffering from diphtheria in an early stage or in a mild form.

In every large city, hospitals for diphtheria patients should be established, not only for the poor, but with private rooms for cases developing in hotels or other places where isolation is impossible. Every city should be provided with a steam disinfecting plant, where carpets, blankets, bedding, etc., can be sent from the sick-room for disinfection.

Quarantine.—Not only every undoubted case of diphtheria, but every suspected case, should be immediately isolated. Quarantine for the latter should continue until the diagnosis is settled either by a bacteriological examination or by the course of the disease. Positive and suspected cases should not be isolated together. The quarantine in every instance must be complete. If possible, cultures should be taken from the throats of all exposed children. Those containing diphtheria bacilli should be quarantined like cases of diphtheria, for they may be equally dangerous; they should use gargles and sprays, and the nose and throat should be closely watched.

Bacteriology has furnished some very definite data from which the necessary duration of the period of quarantine may be determined. In this the physician is to be guided by the time that the bacilli remain in the throat, for the patient is to be considered as dangerous while they persist. This point was investigated by the New York Health Department in 605 cases: In 304 of these the bacilli had disappeared by the third day after the membrane was gone; and in 301 they persisted for a longer time—in 176, for seven days; in 64, for twelve days; in 36, for fifteen days; in 12, for twenty-one days; in 4, for twenty-eight days; in 4, for thirty-five days; and in 2, for sixty-three days. In many of the cases in which the bacilli persist for an unusual time they are found deep in the crypts of the tonsils. Others are cases of nasal diphtheria; in some of these doubtless the antrum has been invaded. While it is unquestionably true that in a certain number of cases these persistent bacilli are non-virulent, the opposite has been frequently shown. Of 15 cases in which the virulence was tested, virulent bacilli were found in 9 at periods varying from eight to twenty-five days after the membrane was gone.

Treatment of Suspected Cases.—During an epidemic of diphtheria,

especially in an institution, every child with sore throat or nasal discharge should be looked upon with suspicion, and isolated pending the result of a bacteriological examination, even though no membrane is present. If there are patches on the tonsils or any other visible membrane, the case should be treated as true diphtheria, in order that no time may be lost. If the bacteriological examination shows the disease not to be true diphtheria, the patient may be released from quarantine in two or three days, provided the throat symptoms disappear. It is, of course, important that the conditions laid down with reference to bacteriological diagnosis shall have been fulfilled. Should symptoms continue, however, a second culture should be taken.

Immunization of Persons Exposed.—When a case of diphtheria occurs in a family or an institution, every child and all adults should have their immunity determined by the Schick test. This is based upon the irritating action of unneutralized diphtheria toxin upon tissues, when injected intracutaneously even in the minute amount. The test therefore determines the presence or absence of natural antitoxin, and indicates whether or not persons are susceptible to the disease.[1]

The New York Health Department supplies an outfit for making this test.

Those persons with an immunity do not require antitoxin. Children who give a positive Schick reaction should be immunized. Adults who are not immune should be carefully observed. If they are to come in close contact with diphtheria patients they also should receive an immunizing dose of antitoxin. When it is impossible to apply the Schick test, children under five years of age should be immunized with antitoxin at once. With older children immunization may be postponed, provided only that they can be observed at least twice a day. If this can not be thoroughly done, all children under ten years of age should receive a prophylactic injection of antitoxin. Those older may be treated as adults are treated by close observation, but without antitoxin unless sore throat or other suspicious symptoms arise.

[1] The method of applying the Schick test is as follows: With a fine hypodermic needle and using a carefully graduated syringe 1/50 of a minimum lethal dose for the guinea-pig, of diphtheria toxin is injected intracutaneously in .1 or .2 c. c. of salt solution.

If natural antitoxin is present no reaction occurs beyond that due to the small puncture. If no antitoxin is present a circumscribed area of redness, ½ cm. in diameter, appears in twenty-four to forty-eight hours. This persists for six to ten days and gradually disappears, leaving a brownish pigmented spot that scales superficially, and that may be appreciable for months. There are no constitutional symptoms and no pain. The test is sharp and accurate. Occasionally a pseudo-reaction may be seen. This appears earlier and disappears in 48 hours. The area is less sharply circumscribed and more indurated.

The dose for immunization is from 500 to 1,000 units, the former being that required for an infant, and the latter for older children. There is no doubt that for a limited time—from two to three weeks—almost complete protection is conferred.

Diphtheria so often complicates scarlet fever and measles, particularly in institutions and in hospitals for contagious diseases, that special consideration should be given to such patients. The Schick test should be made on all, and those patients with no natural immunity should be given antitoxin. If the test can not be made, the only safe rule is to immunize every child admitted to a scarlet fever or measles hospital, and in institution epidemics of either of these diseases to immunize every child attacked.

A nurse who is not immune to diphtheria should not work in infectious hospitals nor, ordinarily, care for diphtheria patients in private practice. If it is necessary for her to take care of a diphtheria patient she should receive 1,000 units of antitoxin. These general rules do not apply to physicians who are in less close contact with patients. They should take the same precautions as in scarlet fever.

The injection of a mixture of toxin and antitoxin in which the toxin is not completely neutralized is often used with animals to cause a production of antitoxin. Theobald Smith suggested such a mixture for the immunization of children and von Behring put it to the practical test. Recent observations by Park and Zingher have shown that this method not only increases greatly the amount of antitoxin present in the blood of immune persons, but causes the production of antitoxin in a large proportion of those who are susceptible to the disease. A combination of the mixture with a vaccine of killed diphtheria bacilli seems to be advantageous. The effect is not evident at once, but after several weeks an immunity can be demonstrated which has been proven to last for many months; how much longer it is as yet impossible to say. Forty out of fifty susceptible persons in Park and Zingher's series developed an antitoxic immunity. It is evident that a means is thus offered of producing immunity in susceptible persons, which may be of great service, not only for the individual, but one which can be employed to prevent outbreaks of diphtheria in institutions in which children remain for a length of time. The method is not, however, applicable for use during epidemics.

Treatment.—*General Measures.*—The directions to be carried out in the sick-room have been outlined in the introductory pages on Infectious Diseases. It is important in every case of diphtheria that there should be plenty of fresh air in the room throughout the attack. Hospital patients should never have less than 1,000 cubic feet of air space, and if possible 1,200 should be allowed. Even in mild attacks the patient

should be kept in bed throughout the entire illness, and in severe attacks this should be continued for some time during convalescence.

Nursing infants may be fed on breast-milk obtained by a breast-pump, but should not be put to the mother's breast. Those who are not nursed and older children should be fed very much as in other cases of severe illness. Milk is the main reliance; it should usually be diluted. The greatest difficulty in feeding is seen in the latter part of the disease, when the patients are septic and have a strong aversion to food, when vomiting is easily excited and when swallowing is difficult on account of the swelling and pain. It is then that gavage is most valuable. In older children the tube may be passed through the nose.

Stimulants.—In most cases they are not needed until the third or fourth day, and in many they may not be required at all. The indications for stimulants are marked prostration, a feeble pulse, and a weak first sound of the heart. Of alcohol, half an ounce of whisky or brandy in twenty-four hours is enough for a child four years old. This should be diluted with at least eight parts of water. In very severe cases two or three times as much may be given; but more than this, except for a short period, is seldom wise. More reliance is to be placed upon the other circulatory stimulants, especially caffein, camphor, and digitalis, which are given for the same indications as in other acute diseases. In cases of threatened cardiac paralysis occurring late in the disease or during convalescence, morphin should be used hypodermically. Full doses must be given and repeated every two to four hours, so that the child may be kept under its influence.

Except for stimulation or the control of special symptoms such as diarrhea, all internal medication should be omitted; for there is yet wanting proof that drugs influence the course or the result of the disease.

Local Treatment.—Since the introduction of antitoxin local treatment has become a matter of secondary importance; and under conditions when it can be carried out only with great difficulty and the use of force it is often wise not to attempt it regularly.

The purpose of local treatment, it is now generally agreed, should be cleanliness, and not the destruction of bacilli. Cleanliness of the nose, mouth, and pharynx is important, inasmuch as one of the chief dangers of the disease is the aspiration of bacteria contained in the abundant secretions of these parts, into the larynx and bronchi. Our aim should therefore be to keep the parts as clean as possible without too severely taxing the strength of the child.

For cleansing the nose and pharynx only syringing can be depended upon. Nasal syringing is indicated when there is much nasal discharge, whether membrane is visible in the anterior nares or not. In septic

cases with a profuse fetid discharge it may be necessary to syringe the nose, no matter how strongly the child resists. Whether it shall be done, will depend upon the condition of the patient's strength and his pulse. The purpose in syringing is not so much to clear the nose, from which absorption is slow and imperfect, as to flush the rhinopharynx, from which absorption is always very active. Only bland solutions should be employed, such as a saline solution, one per cent, or a boric-acid solution, one- to four-per-cent strength. For some cases, a piston syringe may be used; but for most a fountain syringe possesses manifest advantages, and it is more convenient for hospital purposes. Irrigation of the pharynx is best done with the fountain syringe, and is of especial value where there is much swelling or abundant discharge. All solutions should be used as warm as can be borne, and in sufficient quantity to irrigate the parts thoroughly, a few such irrigations being much better than a great many partial ones. By a skilful nurse syringing can in most cases be done with comparatively little disturbance to the child.

Slight nasal hemorrhages may necessitate less frequent syringing, and a free hemorrhage may require it to be discontinued. Astringent solutions of alum and epinephrin are often beneficial in such cases, but they must be used carefully. In children who are old enough gargles should be used. A solution of boric acid, or Dobell's or Seiler's solution much diluted, may be employed.

In cases with a moderate nasal discharge it is usually sufficient to syringe three or four times a day; but in severe septic cases, with very abundant discharge, syringing should be repeated as often as every two hours during the day and every four hours at night.

External applications have no effect upon the disease, but are often useful to relieve pain and tension in the swollen lymph-glands. Poultices should not be employed. As a continuous application, only cold is to be advised, generally by means of an ice-bag well protected to prevent wetting the clothing.

The treatment of cardiac and other forms of post-diphtheritic paralysis has been considered in the chapter on Multiple Neuritis.

The Serum Treatment.—Antitoxin is produced by the cells of the body under the stimulus of the diphtheria toxin. It is intimately combined with the globulin of the blood, and is itself possibly a globulin. It directly neutralizes the toxin produced by the diphtheria bacillus, and also has some effect upon the bacilli themselves, the nature of which is not understood. It induces a condition in the blood which inhibits the growth of the bacilli, and thus arrests the membranous inflammation which they excite.

Properly prepared, it will keep without deterioration for from three

to six months; but after one year it loses somewhat its antitoxic properties. It should be kept in a cool, dark place, and after a bottle has been opened it should be used within a few days. Antitoxin is now prepared in a dry form, which is to be preferred only when it must be kept for a very long time.

The strength of the serum is measured in antitoxin units, the unit being an arbitrary one, viz., the amount of antitoxin which will protect a guinea-pig weighing 250 to 300 grams against one hundred times the fatal dose of diphtheria toxin. Behring's serum first used contained but one unit in each c. c. At present there can be obtained sera containing 1,000 antitoxin units or more in each c. c. This concentration is of immense advantage and has to a large degree done away with the unpleasant symptoms.

Method of Administration and Dosage.—The skin should be thoroughly cleansed with alcohol; the needle should invariably be boiled and the whole syringe either boiled or rinsed with alcohol. The seat of injection is not a matter of great importance; our own preference is for the cellular tissue of the abdomen or axilla or the muscles of the buttock. Absorption from the cellular tissue is slower than from the muscles. For very rapid effect, however, intravenous injections should be employed. After the injection is made the puncture should be covered by adhesive plaster.

The union of the toxin with the cells takes place rapidly. To prevent this the maximum required dose should be given early in a single injection, rather than in divided doses. While the deleterious effect of the toxin bound to the cells can not be neutralized except to a slight extent, the blood can be supplied with sufficient antitoxin to neutralize new toxin as fast as it is produced. Convinced now of the essential harmlessness of the serum, the tendency everywhere has been to use larger and larger doses. For a child over two years old an initial dose for a severe attack, including all laryngeal cases, should not be less than 7,000 or 8,000 units administered intramuscularly or preferably intravenously. Children under two years should receive from 5,000 to 6,000 units. Cases of exceptional severity, in older children, should receive from 10,000 to 15,000 units intravenously. Mild cases should receive from 3,000 to 5,000 units, a repetition of the dose in any patient being usually unnecessary.

In cases receiving antitoxin late, even though the symptoms may not seem particularly severe, the dose should be increased in proportion to the length of the illness, and given intravenously. Only serum from a trustworthy manufacturer should ever be used. The most concentrated serum which can be obtained should be selected.

All experience shows that the results are greatly modified by the

time of its administration. The serum can not undo the serious damage already done to the cells of the body, and this at the time of injection may be so great that death will result. In very mild cases, with older children, one may wait for the result of a bacteriological examination, but never in a severe case and never in a young child. In the group of severe cases should be placed every one which at the first visit shows a pharyngeal exudate covering more than the tonsils, also all cases with symptoms of laryngeal invasion, and all with an exudate on the pharynx and a profuse nasal discharge. If in a doubtful case twelve hours' observation shows that the membrane has spread from its original seat, no further delay is admissible. In human diphtheria marked benefit usually follows injections made as late as the third day; but after this time the value of the serum diminishes very rapidly, and although striking examples of benefit are sometimes seen after later injections, they can not be depended upon. In very severe or in malignant cases so much harm may be done during the first twenty-four hours of the attack that the subsequent use of antitoxin is without avail.

The effect upon the diphtheritic membrane is usually noticeable within twenty-four and often in twelve hours; it first stops spreading, and soon begins to soften and loosen. The swelling of the mucous membrane subsides and the local disease abates, very much as when the disease runs its usual course. The striking thing after the use of antitoxin is the rapidity with which these changes take place, and the abrupt transition from an advancing to a retrograde process. The subsidence of the inflammatory conditions in the larynx and trachea is quite as marked as in the pharynx. The symptoms of stenosis, even when severe, often diminish in a few hours, making operation unnecessary in a very large number of cases when previously it seemed inevitable. The membrane loosens rapidly in the larynx and trachea, sometimes necessitating the frequent removal of the intubation tube, when operation has been performed. Improvement is also shown by the cessation of the nasal discharge, the re-establishment of nasal respiration, and the diminution in the swelling of the glands of the neck.

The effect upon the constitutional symptoms is not less striking. In favorable cases there is seen, often in twelve hours, a fall in temperature and improvement in the pulse and in the nervous symptoms.

The Limitations of Antitoxin.—It is important that these should always be kept in mind. The serum must be given early, for if given late it can not undo the mischief already done by the diphtheria toxin. Cases of great severity have often passed the period when recovery was possible, before the antitoxin is given. This period may in some cases be four days, in others it may be less than twenty-four hours. The tissues most susceptible to the diphtheria toxin are probably those of the nervous

system, the heart, and the kidneys; and the consequences of its action may be seen in the production of nephritis, in heart failure at the height of the disease, or in later paralysis of the heart, respiration, or the voluntary muscles, in spite of the fact that antitoxin is given at a period early enough to avert death from local disease in the larynx or bronchi. Against the phlegmonous inflammation of the throat or the cellular tissue of the neck, bronchopneumonia, and nephritis, antitoxin is powerless; and just in proportion to the severity of these inflammations are negative results seen.

Eruptions and Other Unpleasant Effects.—Some transient, local edema usually follows the injection and a slight rise of temperature is very frequently observed. In a few hours there may be seen a general erythema; this, however, is rare and usually of short duration. The most important eruptions are seen between the eighth and fourteenth days. They follow from five to ten per cent of the injections made, and appear to be quite independent of the amount of serum used. The exact cause is not known. The most common eruption is urticaria. This is often intense, very annoying, and may nearly cover the body. It may be accompanied by a slight rise of temperature; it usually lasts for two or three days; but is rarely severe for more than twenty-four hours. Various forms of erythema are occasionally met with. In several instances we have seen hemorrhagic eruptions, generally in the neighborhood of the large joints, and always in children suffering from extreme malnutrition. In a few cases a moderate swelling of some of the joints has been observed, and a transient albuminuria. One occasionally meets with patients who seem unusually susceptible to serum injections, and in whom even small immunizing doses cause headache, muscular pains, and general malaise, so that they feel quite wretched for several days. All of the above symptoms except the urticaria are rare, and should not for an instant deter one from using antitoxin when indicated. They are much less common with the refined and concentrated antitoxin in use at the present time.

Real and Alleged Dangers from Antitoxin Injections.—In a few instances sudden death has followed antitoxin injections, but the evidence that antitoxin was the cause of death has not always been conclusive. In some of these patients the autopsy has revealed a status lymphaticus not before suspected. In this condition the shock of so slight a thing as a needle puncture may produce death. There are other cases which do not admit of this explanation. Almost all have occurred in patients during adolescence or adult life. The symptoms usually come on within a few seconds or minutes after the injection and occur quite independently of the dose given. Several have followed small immunizing doses given to apparently healthy persons, but the majority have been suf-

ferers from hay fever or asthma, usually from that form excited by contact with horses. In some recorded cases the patients had received antitoxin before; in the great majority, however, the sensitiveness to the protein of horse serum had been acquired in some other way. The most striking symptoms are a rapidly developing dyspnea with cyanosis and great prostration. In the most severe cases death may follow in a few minutes from respiratory failure; in those less severe, a gradual recovery takes place with no permanent after effects.

Such experiences are, fortunately, exceedingly rare. No fatalities or even severe respiratory symptoms due to the administration of antitoxin have been observed since its introduction in the Willard Parker Hospital in New York where many thousands of injections of antitoxin are given each year. Certainly in children with diphtheria one should not hesitate one moment in regard to its use. If the patient gives a history of asthma, and inquiry should always be made regarding this, special precautions should be employed in giving antitoxin. As concentrated a preparation as possible should be used and injected subcutaneously a drop or two at a time, at intervals of ten or fifteen minutes. If there is no reaction after the first few drops the rest may be injected at once. If there is any reaction it will not be severe and after a time a drop or two more may be given. Thus the whole dose may be given, though it may require much time. With a clear history of asthma, injections for immunization may well be omitted and the child kept under close observation. If symptoms develop after the injection of serum, atropin should be given in full doses; epinephrin and morphin are also useful. In some instances artificial respiration has apparently been beneficial.

Results with Antitoxin Treatment.—Since 1895 the serum has been tested on such an extensive scale as the prevalence of diphtheria all over the world has made possible, with results so uniformly good that it seems quite unnecessary any longer to cite statistics in proof of the value of this remedy.

The beneficial effects of antitoxin may be summed up in the following statements: (1) The percentage mortality from diphtheria in hospitals both in Europe and in America has been reduced to a little more than one-third the previous figures; (2) the proportion of cases now requiring operation for laryngeal stenosis has been reduced to about one-half; (3) the mortality after tracheotomy has been reduced to one-half, and that after intubation to about one-third the former figures; (4) but even more convincing is the effect of the serum treatment upon the actual diphtheria mortality of cities and countries where it has been used.

Convalescence.—After a severe attack of diphtheria convalescence is always slow on account of the anemia and the depressing effects of the

disease. Patients should invariably be kept in bed for at least a week after the throat has cleared, and much longer if any tendency to cardiac weakness is seen. The pulse should be carefully watched, and irregularity, intermission, dicrotism, or a weak first sound of the heart, should make one apprehensive. An abnormally slow pulse is generally more serious than one which is rapid. In such circumstances the patient should be kept recumbent and absolutely quiet, since fatal syncope may be the result of a violation of these rules. The extreme degree of anemia frequently requires that iron be given for a considerable time during convalescence.

Great difficulty is occasionally experienced in getting rid of the bacilli in the throat. The tonsillar crypts, the adenoid tissue of the rhinopharynx, and the nasal sinuses are the places where the bacilli are most likely to remain. Inasmuch as it is now generally made a condition of release from quarantine that the throat shall have been shown by cultures to be free from bacilli, this becomes a matter of much importance. Nasal syringing with a very weak solution (1-10,000) of bichlorid to which ten per cent solution of glycerin has been added is sometimes efficacious. The fluid should be warm and the syringing gently done twice daily. The same solution may be used as a gargle. For children under four years old a simple salt solution, or a dilute Dobell's solution, should be substituted and the gargle omitted. In some obstinate cases the best procedure is to omit all local treatment and get the patient into the open air of the country. When bacilli are very persistent, as they often are for weeks, their virulence should be tested. In the great majority of such cases they are found to be non-virulent and further quarantine is unnecessary. When virulent bacilli long persist, the question of the removal of the tonsils should be considered. It is sometimes successful when all other means of getting rid of the bacilli have failed.

Laryngeal Diphtheria.—Emetics, inhalations of steam, and solvents for the membrane, although they all sometimes give relief, are not to be relied upon.

Opinions will always differ as to the time when operative interference is called for. One should never wait for general cyanosis, for often this does not occur until just before death. It is better to operate too early than too late. If, in spite of other measures, the dyspnea increases steadily, operation should not be deferred longer. Intubation has almost universally superseded tracheotomy as a primary operation for the relief of membranous laryngitis. Tracheotomy is still needed at times for the cases, few in number, in which intubation fails to give relief on account of the position of the membrane or for some other complication.

Intubation

Intubation is the introduction of a tube through the mouth into the larynx for the relief of laryngeal dyspnea. For the operation, as now performed, the world is indebted to the late Dr. Joseph O'Dwyer, of New York.

A set of O'Dwyer's instruments consists of seven tubes, an introductor, an extractor, a mouth-gag, and a gauge. The tubes are made of hard rubber and lined with gold-plated metal. So carefully did O'Dwyer perfect his instruments that nothing of importance has been added by others. It is interesting to note that nearly all the modifications which have been suggested since his first publication had already been tried by him and discarded. No one thing is more essential to success with intubation than properly constructed instruments. The operation is not difficult if one has had practice on the cadaver. Without this it should not be attempted. The tube is selected according to the age of the patient, this being indicated on the gauge. A very large child will often require a tube of larger size than his age would call for.

Introduction of the Tube.—Either one of two positions may be employed, the choice depending upon the preference of the operator. In one the child is seated upon the lap of a nurse while his head is steadied by a second assistant standing behind. In the other position the child lies upon his back upon a table, his head being steadied by an assistant. In both positions the arms should be pinioned to the sides by a sheet. In the recumbent position the child can be held more firmly; it has also the advantage of dispensing with one assistant, and in an emergency with both of them. The tube is attached to the introductor, and the gag is inserted at the left angle of the mouth and opened as widely as possible. The attempts at introduction must be made quickly, for during them respiration is practically arrested. Several short attempts are always better than a single prolonged one. Very little force is ordinarily required in introducing the tube, that used in passing a catheter being a good general guide. In cases of subglottic stenosis, however, quite a little force may be necessary.

The index finger of the left hand is used as a guide in introduction. This is passed well back into the pharynx, then brought forward until a hard nodule—the upper border of the cricoid cartilage—is encountered. This is the best of all landmarks, since the soft parts are often distorted by swelling. Directly in front of the cricoid cartilage may be felt the epiglottis and the opening of the larynx, which are readily recognized after the touch has become somewhat educated. The epiglottis is drawn forward and the tube is passed along the palmar surface of the left index finger, by which it is guided into the larynx; it is then pushed off the

introductor by a thumb-piece attached to its handle. When it is certain that the tube is in position, and the patient breathes properly, the loop of silk attached to the head of the tube is cut off and pulled through, the removal of the tube being prevented by placing the left forefinger upon its head. The silk is not usually left attached unless there is evidence of loose membrane below the tube. It may be desirable to leave the silk attached in case no one is within reach who is able to remove the tube should it become obstructed. The child's arms and hands should then be secured to prevent him from seizing it himself. When not removed, the silk is fastened to the cheek by a piece of adhesive plaster. The tube is known to be in place, first, by the hissing breathing sounds, somewhat similar to what is heard when the trachea is opened; secondly, by a severe paroxysm of coughing, which is usually excited by a tube in the larynx; thirdly, by the relief of the dyspnea. If this relief is not very apparent the physician may still be in doubt as to whether the tube is in the larynx or the esophagus. If in the former, it can not be pushed down by the finger without depressing the larynx with it; and by introducing the finger into the pharynx, the posterior wall of the larynx can be felt between the finger and the tube. The most common mistake made is to pass the tube into the esophagus. This sometimes happens because the position of the child's head is improper—too far forward or too far backward—but more often because the operator has not been quite sure of his landmarks. If this has occurred, there is no relief to the dyspnea, no hissing sound, and the tube can be pushed down indefinitely. When this condition is recognized, the tube is withdrawn by the loop of silk and after a few moments a second attempt made.

False passages in the larynx are most frequently made by employing too much force or because the operator has worked at the angle of the mouth instead of keeping in the median line. The tube usually goes into one of the ventricles of the larynx and may be pushed quite through the larynx into the cellular tissue. This is not very likely to happen, however, unless undue force has been used. The production of a false passage is recognized by the fact that, although the tip of the tube can be felt to enter the larynx, the tube does not descend, but projects above the epiglottis.

False membrane which has become loosened is sometimes crowded down by the tube and obstructs the larynx just below it. This is one of the most serious accidents that may occur, but fortunately it is not a frequent one. It is more likely to happen when the disease has existed for several days than in recent cases. The tube may be in place in the larynx as shown by all the signs above mentioned, except relief of the dyspnea. In such a case the immediate withdrawal of the tube is necessary, it being often followed by the discharge of masses of loose mem-

brane. This is aided by the administration of half a teaspoonful of pure whisky or brandy to excite a strong cough. Artificial respiration may be required, and if there is no relief by any of these means tracheotomy is indicated. Asphyxia is sometimes produced by prolonged and injudicious attempts at intubation.

After-treatment.—So far as the tube itself is concerned no treatment is required. The original disease is to be treated as before. The operation has removed only one danger from the patient, viz., that of asphyxia from mechanical obstruction of the larynx. A good expulsive cough should occur after the tube is in place. This is necessary to clear the tube of mucus, as the pharynx and larynx are generally filled with it as a result of the manipulation.

The child should not be allowed to lie upon his face, nor should he be held over the nurse's shoulder face downward, for in either position a slight cough is enough to expel the tube. Nursing infants may sometimes continue at the breast after the operation; ordinarily they have but little difficulty in swallowing. Older children often experience considerable trouble in taking liquids. This may be overcome by the device suggested by Casselberry, of having the patient's head lower than his body while he drinks. When fluids cause excessive coughing, or at other times when they can be taken only with the greatest difficulty, they may be given through a nasal tube or one passed through the mouth. Semi-solid articles, such as condensed milk, wine jelly, cornstarch, ice cream, or scrambled eggs, may be well taken when fluids are not. Feeding is always easier after the first day or two, and patients who wear a tube for chronic disease soon experience no trouble whatever, showing that the difficulty depends more upon the inability to co-ordinate the movements of the muscles of deglutition when the tube is in place than upon mechanical causes, for the head of the tube is effectually covered by the epiglottis.

When the tube is removed by extubation or coughed up, the dyspnea does not usually return for two or three hours, but may come back at once. It may happen that the tube is coughed up and not seen by the nurse, or it may be coughed up and swallowed by the child. When called because of dyspnea after operation, the physician should make a digital examination of the pharynx to discover if the tube is still in place. Swallowing the tube generally causes no harm to the child, for tubes have repeatedly passed through the intestines. Should the tube be coughed out at any time its introduction should be delayed until dyspnea returns.

It sometimes happens that the tube is coughed out soon after its introduction because too small a size has been used. At other times this occurs repeatedly even with tubes of the proper size. Such cases

are probably due to paralysis of the laryngeal muscles. As patients in such circumstances are unable to breathe for even a few minutes without the tube it is usually necessary with repeated self extubation to perform tracheotomy.

The entrance of food into the bronchi through the tube is a danger that does not exist, and bronchopneumonia following intubation does not depend upon this cause.

Deep ulceration at the head of the tube rarely occurs, provided properly made tubes are employed, but superficial ulceration is almost invariably produced at the base of the epiglottis and in the trachea at the lower end of the tube. Deep ulcers extending to the tracheal rings may occur in ill-conditioned children, usually in connection with other complications serious enough to cause death.

Spontaneous descent of the tube into the larynx is almost impossible, and it can not be crowded down without using considerable force and severely lacerating the larynx.

Sudden blocking of the lower end of the tube by membrane loosened from the trachea or bronchi occasionally occurs. The usual result of this is the immediate expulsion of the tube by coughing, the discharge of the loose membrane following. This condition is one of the safety valves of the operation. One of the strong points in favor of intubation is that the forcible cough which the patient is able to make on account of the narrow opening of the tube, often enables him to expel large accumulations of mucus, and even membrane, more readily than through a much larger tracheal opening.

The period for which the tube is required varies much in different cases. It has been materially shortened by the use of antitoxin. The average time of wearing the tube is about five days, and in many it can be dispensed with in two or three days. An attempt should be made to have the child go without the tube whenever the temperature reaches normal. If complications are present that still cause fever extubation should not be deferred beyond the fifth or sixth day. The majority of cases do not require re-intubation. If this is necessary, extubation should be done again in three or four days and repeated thereafter at this interval until the tube is no longer necessary. If, after several weeks the tube cannot be dispensed with the treatment described later for retained intubation tubes should be adopted.

Removal of the Tube—Extubation.—This is rather more difficult than its introduction. The general arrangement of the patient and assistants is the same as for introduction. The left index finger is placed upon the head of the tube, which is steadied externally by the thumb of the same hand. The beak of the extractor is introduced within the opening of the tube, its jaws are then separated by pressure upon the lever

at the handle, and the instrument withdrawn, very slight force being required.

The tube is first removed tentatively, the physician waiting to see if dyspnea returns. It is well to give a full dose of morphin an hour before the removal of the tube, since this operation is almost invariably followed by a marked degree of laryngeal spasm which lasts for ten or fifteen minutes. To avoid the production of vomiting and the entrance of food into the larynx, food should not be given for three hours previously. If dyspnea does not return in the course of three or four hours, the probabilities are that the tube will no longer be required. It is exceptional that the patient has great difficulty in dispensing with the tube, as so often happens after tracheotomy.

The only objection of much force urged against intubation is that asphyxia may be produced by crowding down loose membrane into the larynx. This is an infrequent accident; should it happen, and the asphyxia not be relieved by removing the tube and inserting another, tracheotomy may be performed.

There is always some degree of hoarseness following intubation, but in the majority of cases it disappears within a week, occasionally it continues as long as three or four weeks, but it is very rarely if ever permanent. The duration of the aphonia seems to have little relation to the length of time the tube is worn, unless this is many weeks.

Experience has clearly proved that intubation relieves the dyspnea due to laryngeal stenosis promptly, efficiently, and certainly; it does this without many of the dangers and objectionable features of tracheotomy, while at the same time it does not deprive the patient of any essential advantage which tracheotomy affords.

Retained Intubation Tubes—Prolonged Intubation.—Difficulty is experienced in dispensing with the intubation tube much less frequently than with the cannula after tracheotomy; yet when this condition occurs it is the cause of much concern and even danger. Trouble of this sort is seen in about five per cent of the cases of intubation. In the majority of these the patient is able to do without the tube in a few weeks, and such cases require very close attention, but no special treatment other than the substitution at times of a special O'Dwyer tube with an extra large "retaining swell." But occasionally there are met with cases in which every effort to dispense with the tube proves futile. Although the children breathe well with the tube in place, still if it is removed or expelled by coughing, in a short time, varying from a few minutes to several days, the dyspnea returns with such severity that the tube must be replaced to prevent asphyxia. Inasmuch as these patients sometimes expel the tube several times a day, surgeons have often resorted to tracheotomy to avert the danger of suffocation, which might easily occur if no

one were at hand who could replace the tube. This operation, however, gives only temporary relief. Many of these children, after wearing tubes of one sort or another for years, ultimately die from some accident connected with the tube or from pneumonia.

The causes and the exact pathological condition underlying this difficulty are subjects regarding which there has been much difference of opinion. The cause of the returning dyspnea is probably subglottic swelling and edema which occur in tissues which are the seat of chronic inflammation, as soon as the pressure of the tube is removed. In a few cases a cicatricial condition, the result of previous ulceration, has been found; but it is doubtful if granulations, so frequent a cause of retained cannula after tracheotomy, play an important part. The chronic inflammation of the mucous and submucous tissues of the subglottic region of the larynx which produces the symptoms, is aggravated by a faulty tube or a clumsy operation, but it may occur under the most favorable conditions.

For the relief of this condition, O'Dwyer advised in recent cases the application of astringents by means of an intubation tube coated with gelatine with which some astringent was combined. For those patients who cough out the tube frequently, tracheotomy is at times a necessity to prevent sudden death. But this does not affect the original condition, for the same difficulty exists in doing without the tracheal cannula. The operations of laryngotomy, curetting, etc., have been such signal failures as to discourage one from repeating them.

The most successful method of treatment thus far proposed is that of Rogers, which consists in increasing intra-laryngeal pressure by the insertion of larger and larger intubation tubes. This is not to be adopted until long after all acute symptoms have subsided. The first tube used is as large a one as can be introduced without force; after a few weeks, the next larger size, and after a longer interval, possibly a still larger one. When the very large tube has been worn for several weeks one is usually able to dispense with all tubes.

True cicatricial stenosis may best be relieved by opening the trachea and dilating from below, and afterward inserting an intubation tube. When there is complete destruction of the cricoid cartilage, as sometimes occurs, tracheotomy is the only remedy, but this is only palliative, as the tube must be worn permanently.

CHAPTER IX

TYPHOID FEVER

Typhoid fever is an acute infectious disease due to a specific organism—Eberth's bacillus. It may affect the fetus *in utero*, or the newly-born child, and it is seen in infancy and throughout childhood.

Paratyphoid.—This is a disease in all respects similar to typhoid fever and one that cannot be differentiated from it except by bacteriological examination. It may be due to organisms known as paratyphoid "A" and paratyphoid "B." This disease is much less common than true typhoid, but small epidemics from time to time appear. These are usually due to paratyphoid "B" which, in this country at least, is much more common than paratyphoid "A." There are no clear distinguishing features between them. Widal reactions in these infections and in true typhoid somewhat overlap one another; but they may, in certain instances, be fairly distinct so that from the Widal alone the diagnosis can be suspected. Not many autopsies have been reported after infection with these organisms; but in general the lesions do not differ markedly from those of true typhoid.

Fetal Typhoid.—When a pregnant woman develops typhoid fever, infection of the child *in utero* is a frequent but not an invariable occurrence. The fetal form of the disease is a general blood-infection, since the intestines are not functionally active. The most common result is death of the fetus and consequent abortion; but the child may be born alive still suffering from the infection. On account of the infant's feeble resistance death usually occurs.

Infantile Typhoid.—Modern methods of diagnosis, particularly blood cultures, have answered the question, long discussed, as to the frequency of infantile typhoid. It is a relatively rare disease. In over 14,000 admissions to the Babies' Hospital, New York, covering a period of thirteen years, but eleven cases of typhoid were observed under two years of age and but five cases of one year or under, the youngest case observed being in a child eight months old. In Philadelphia, where there has been much more typhoid generally than in New York, Griffith reports under his personal observation or in the Children's Hospital forty-five cases under two years and nine under one year; his youngest cases were aged three, five, and nine months respectively. Typhoid has been seen by Murchison at six months and by Ogle at four and a half months, the diagnosis being, in both instances, confirmed by autopsy. It is during epidemics that most of the infantile cases are seen, but even in epidemics it is surprising that so few infants are attacked.

Typhoid in childhood is by no means rare, but it is not until after the fifth year that it can be said to occur frequently. The following figures, embracing groups of cases reported by eight writers, represent the relative frequency with which the disease is seen at the different ages: Of 970 cases, eight per cent occurred under five years, forty-two per cent between five and ten years, and fifty per cent between ten and fifteen years.

Typhoid fever is almost invariably contracted by drinking water or milk which contains the germs of the disease. The infrequency of typhoid even in infants who are artificially fed is explained, in part at least, by the fact that most of the water and a large part of the cow's milk taken have been previously boiled, or heated in some manner.

Lesions.—In a general way these resemble those of adults except in severity. In a considerable number of the cases the pathological process in the intestines does not go on to ulceration; and when ulcers form they are seldom large or deep, and perforation is very rare. Montmollin gives the following facts concerning twenty-three autopsies, most of them, however, being in children over eight years old: ulcers were present in seventeen cases; they were situated in the lower ileum in sixteen, and in ten they were only there; in the ascending colon in nine, and only there in one case; perforation occurred in three cases, in every instance in the lower ileum. Autopsies made upon infants may show even less severe intestinal lesions than those mentioned. In fact, some cases in which the clinical diagnosis was beyond question, have shown only moderate redness and swelling of Peyer's patches, the solitary follicles and the mesenteric lymph nodes—lesions which are exceedingly frequent in cases of simple diarrhea. In a doubtful case such post mortem findings do not establish the diagnosis of typhoid. Indeed, they prove nothing unless cultures from the intestinal contents, the mesenteric glands, or other organs, show the typhoid bacillus. Enlargement of the spleen is practically constant. The degenerative changes in the heart, the kidneys, and the liver are much less frequent and generally less severe than in adults.

Symptoms.—The peculiar features of typhoid in early life are seen only in children under ten years old; for after this time the disease does not differ essentially from the adult type. In brief, the typhoid of early childhood may be described as a fever characterized more often by nervous symptoms than by intestinal symptoms.

Onset.—A sudden onset with well-marked symptoms—fever, prostration, vomiting, etc.—is not uncommon; in fact, it is more frequently seen than the insidious beginning, with lassitude, headache, coated tongue, anorexia, and gradual rise in temperature. In cases developing abruptly it often appears as if an acute indigestion had been the means of precipitating the attack. The most frequent initial symptoms are

vomiting, diarrhea, prostration, headache, anorexia, and fever. Chills are rare; occasionally there is abdominal pain or tenderness. Epistaxis occurs as an early symptom much less frequently than in adults.

Condition of the Bowels.—There is no constant relation between the severity of the intestinal lesions and the condition of the bowels. Taking large groups of cases together, diarrhea is present in only about half the total number. It is rarely profuse, from two to four discharges a day being the average. The appearance of the stools is seldom characteristic; they are usually thin and fluid, often containing mucus. Constipation may be present at the beginning only, or throughout the attack. Tympanites is generally moderate, and is often entirely absent; it usually accompanies constipation. Marked iliac tenderness and gurgling are infrequent.

Spleen.—By the end of the first week this is usually found to be enlarged to a sufficient degree to be recognized by palpation. In most cases it extends but an inch or an inch and a half below the ribs, but at times it may be three inches or more; persistent enlargement may indicate that the disease is not at an end even though the temperature has reached the normal, and a relapse should be expected.

Eruption.—It is the experience of nearly all who have seen much of typhoid in children that the eruption is less constant, usually less abundant, and less characteristic than in adults, but appears rather earlier. We have, however, seen it so abundant as to suggest measles. The typical eruption consists of small, scattered, rose-colored spots, which appear chiefly or solely upon the abdomen at the beginning of the second week. They come in successive crops, each one of which generally lasts three days, the whole duration of the eruption being about ten days.

Prostration, Emaciation, etc.—As a rule the prostration is quite sufficient to keep a child in bed after the first few days. The general weakness after this time is in direct proportion to the height of the temperature. Loss of flesh is steady and usually marked; and in a prolonged attack there may be emaciation.

Temperature.—In the cases with a gradual onset, the typical temperature curve is one which rises steadily for from two to seven days, fluctuates within the limits of one to three degrees during the second week, and steadily declines during the third week, reaching the normal on the average at the end of the third week. In cases with an abrupt onset, the temperature rises at once to from 102.5° to 105° F., but subsequently may run the same course as in the first group.

The following are the most important variations from the temperature curve of adults: the initial rise is much more frequently rapid; during the second week the remittent character is less marked; the average duration is shorter. In young children the proportion of cases

in which the fever lasts only from eight to fourteen days is quite large (Fig. 171). After the age of ten years the type of the fever is much like that seen in adults. The maximum temperature in the mild cases is 103° or 104° F.; in the severe ones it often reaches 105° or 106° F., but rarely goes above this point. The range is usually higher than in adult cases of the same severity. At the beginning of convalescence a subnormal temperature is very frequent, and by many writers is considered to be the rule. A secondary rise is most frequently due to errors in diet, but may occur from the development of complications. A sudden fall often indicates either perforation or intestinal hemorrhage.

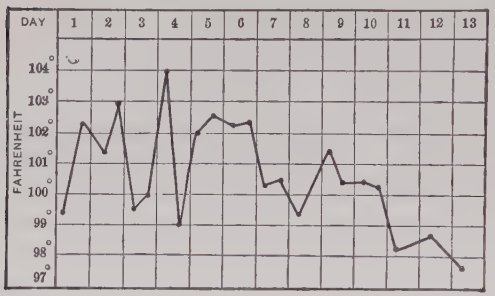


FIG. 171.—TYPHOID FEVER OF SHORT DURATION IN A CHILD THIRTEEN MONTHS OLD. Spleen enlarged; eruption typical; no diarrhea and only moderate abdominal distention. There were two other cases in the family, all being due to the same cause—infected milk. (After Northrup.)

Relapses occur in approximately 10 per cent of the cases. They follow about the same course as in adults (Fig. 172).

Nervous Symptoms.—In many cases these are more prominent in severe cases than the intestinal symptoms, and are directly proportionate to the height of the temperature. The extreme nervous symptoms belong-

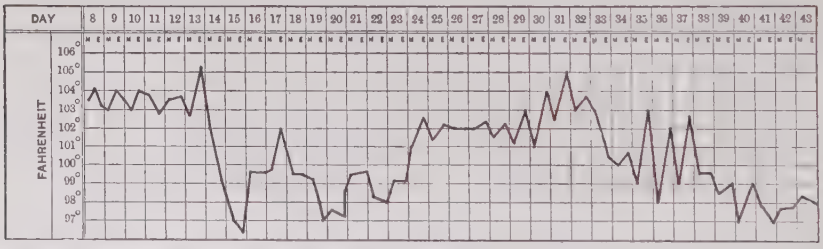


FIG. 172.—TYPHOID FEVER WITH RELAPSE. Child two and a half years old; early temperature high and symptoms typical; natural fall on fourteenth day; rise on seventeenth day apparently due to otitis; relapse on twenty-fourth day, with fresh eruption and return of splenic swelling which had disappeared. Temperature was subnormal at the end both of primary and secondary fever.

ing to the typhoid state in adults are rare in childhood, except in patients over ten years old. Headache and mild delirium at night are very frequent, the former being seen in the majority of cases. Young children are usually dull, apathetic, and often in a state of semi-stupor. Occasionally the disease may closely simulate meningitis. The nervous

symptoms are usually most severe in the second, or early in the third week, and subside as the temperature declines, but may continue for several days thereafter. Exaggerated reflexes and ankle clonus are not infrequent and may persist well on into convalescence in severe cases.

Pulse.—This is increased in frequency, but not to the degree that is seen in most diseases of childhood with a similar elevation of temperature. The force and rhythm of the pulse are usually good, irregularity and dicrotism being rare in children as compared with adults.

Urine.—A small amount of albumin is found in the urine of most of the severe cases at the height of the disease, and is due to acute renal degeneration; but a marked degree of nephritis is infrequent. In from one-fourth to one-third of the cases typhoid bacilli are found in the urine, generally in pure culture. They usually appear in the latter part of the disease, the second or third week, and may continue for months or even years. They are sometimes accompanied by evidence of cystitis or nephritis. Their number is in some cases so large as to render the urine turbid; in others they give no indication of their presence. Ehrlich's diazo reaction is usually present at the height of the fever.

Blood.—The characteristic blood picture in typhoid is a low leucocyte count, generally under 10,000, accompanied usually by a slightly increased proportion of lymphocytes. Blood cultures, with great uniformity, show the bacilli even in the first week of the disease. These usually have disappeared from the blood by the third week.

Intestinal Hemorrhage.—Of 946 collected cases, mainly from hospital reports, intestinal hemorrhage occurred in thirty, or about three per cent; the majority of these were in children over ten years old. Of twenty-four collected cases of hemorrhage in children, ten terminated fatally. The youngest case of this nature which has come under our own notice was in a child of four and a half years.

Intestinal Perforation.—This is even more rare than hemorrhage. In 1,028 collected cases, this accident occurred but twelve times, or in 1.1 per cent. Perforation is indicated by a sudden fall in the temperature, with collapse; usually there is vomiting and the rapid development of tympanites with leucocytosis.

Complications and Sequelae.—The complications of typhoid in early life are infrequent and usually mild. Bronchitis is present in most of the severe cases. Pneumonia has been noted in nine per cent of the cases reported by various authors. Both serous and purulent effusions into the chest are occasionally seen, and sometimes abscess of the lung.

Complications referable to the nervous system are not very frequent, but are of much interest. Meningitis is extremely rare. Morse has collected twenty-one cases of aphasia, in two of which it was clearly due to embolism; in the remainder, however, it apparently was not dependent

upon any organic lesion. In two-thirds of the cases it came on during convalescence, and in nearly all complete recovery occurred after an average duration of three weeks. Aphasia usually followed a severe type of the disease, and in most of the cases was not accompanied by any other paralysis or by mental disturbance. Insanity is a rare sequel of typhoid in children, the usual type being acute mania. Recovery is usually complete. Chorea is seen rather oftener than after the other infectious diseases.

Otitis is not an infrequent complication, occurring much oftener than in adults. It is principally seen in young children and during the cold season. Among the less frequent complications may be mentioned: parotitis, which is usually suppurative and is seen in septic cases; abscess of the liver, examples of which have been reported by Bokai, Asch, and others; gangrenous inflammation of the mouth or genitals; pericarditis, endocarditis, and peritonitis, suppurative inflammations of joints, multiple abscesses and furunculosis. Tuberculosis of the lungs or bones not infrequently follows typhoid.

Diagnosis.—The diagnostic symptoms of typhoid are, the Widal blood reaction, the discovery of the bacilli in the blood, urine or feces, the eruption, the course of the temperature, the enlargement of the spleen and the abdominal symptoms—diarrhea, tympanites, hemorrhage, and perforation.

The Widal reaction is present at some period in from ninety-five to ninety-eight per cent of the cases, and thus becomes the most valuable single symptom for diagnosis. It is seldom obtained before the seventh day and frequently not before the tenth or twelfth; it may not be present until convalescence or a relapse. Repeated tests should always be made if the first reaction is negative or doubtful. The reaction is therefore of much less value for an early than for an exact diagnosis. A positive reaction may be present if the patient has previously had typhoid, something much less likely to be the case with children than with adults; in rare instances it has been obtained in other diseases or in health when no history of previous typhoid existed. Both these conditions, however, are very exceptional, and a positive reaction may as a rule be taken to establish the diagnosis.

Typhoid bacilli may be demonstrated in the stools by culture in a large proportion of the cases. They are found in the urine, usually in the latter part of the disease, in about one-third the cases. Their discovery in either of these discharges is conclusive evidence of previous or existing typhoid. An examination of both urine and feces should, if possible, be made in all doubtful cases.

The course of the temperature is an important aid to diagnosis, but alone is not to be depended upon. The characteristic feature is a fever

which continues for two, three, or four weeks, and subsides gradually. The variations from the adult type have already been mentioned, also the frequency of the eruption, the enlargement of the spleen, and the abdominal symptoms. We are not warranted in making the diagnosis of typhoid, if repeated tests fail to show the Widal reaction or if the eruption and splenic enlargement are absent, and no bacilli can be demonstrated in the blood or discharges, no matter what the course of the temperature may be.

One should hesitate to make the diagnosis of typhoid in a child under two years old, unless typhoid is prevalent in the community. The great majority of sporadic cases reported as occurring in infancy are probably not typhoid. After the fifth year the disease is more frequent, and its symptoms in general resemble those seen in adults, except in severity.

A differential diagnosis is to be made from malarial fever, ileocolitis, meningitis, tuberculosis, and from other ill-defined continuous fevers of unknown origin. From malarial fever the diagnosis is to be made by the temperature curve, the organisms in the blood, and the effect of quinin. In most of the cases of malaria the temperature will be found to touch the normal at some time in the twenty-four hours. The administration of full doses of quinin is a diagnostic test of much practical importance; an irregular or remittent fever which yields promptly to quinin is most certainly not typhoid.

Ileocolitis and typhoid fever are not often confounded. The former is chiefly seen in the first three years of life, a time when typhoid is rare. The intestinal symptoms of ileocolitis are marked even though the temperature is not high, and they are altogether more severe than is usual in typhoid; while enlargement of the spleen, tympanites, and the eruption are not present.

The cerebral symptoms of typhoid may be difficult to distinguish from meningitis, unless one has watched their development. Irregular respiration, a slow, irregular pulse, localized paralysis and complete coma are seldom, if ever, seen in typhoid, and a retracted abdomen very rarely, while the enlarged spleen and the peculiar eruption are not seen in meningitis.

General tuberculosis very often resembles typhoid so closely that a differential diagnosis is almost impossible from symptoms alone until local signs of tuberculosis have appeared, usually in the lungs. The cutaneous test is in most cases a valuable aid.

Prognosis.—Of 2,623 cases in children, collected from the reports of twelve different writers, the mortality was 5.4 per cent. These are, however, almost all taken from hospital reports, where as a rule the mildest cases are not brought for treatment. The mortality of the disease in

children over three years old probably does not exceed three or four per cent. Death seldom occurs from the disease itself, but usually from some accident or complication, the most frequent being pneumonia and intestinal hemorrhage or perforation. Griffith's collection of cases occurring in infancy indicates a much higher mortality for this period. The death-rate for the first year reached nearly fifty per cent.

Treatment.—The usually low mortality of this disease shows how successful all methods of treatment are likely to be considered. In the great majority of cases very little active treatment is required. Every patient with typhoid should be put to bed and kept there during the febrile period, and a few days beyond it, no matter how mild the attack may be. The diet should consist of sterilized milk, broths, cereal gruels, milk toast, soft eggs, custard, and plain ice-cream. These articles should be given liberally every four or five hours, but not pushed beyond the desire of the patient. Milk may be diluted, and kumyss or buttermilk may be substituted for it if the stomach is irritable. Plenty of water should be given. Solid food should not be allowed until the temperature is normal.

Both the urine and feces should be immediately and thoroughly disinfected by a solution of carbolic 1:20. If the movements are in a chamber or a bed-pan they should be covered with this solution for at least six hours before they are thrown into the water-closet. If napkins or diapers are used, they should be soaked in some effective antiseptic solution for twelve hours and then thoroughly boiled. Sheets stained by discharges should be treated in the same way, and all bed-linen should be boiled for an hour, apart from the washing of the family. The efficiency of hexamethylenamin (urotropin) in removing typhoid bacilli from the urine seems now to be well established. It should be given at the close of the attack in doses of three to five grains, three times a day, and continued for a week or ten days.

Diarrhea calls for treatment only when the movements exceed four or five in twenty-four hours. If no more than this number are present, they should not be interfered with. Opium and bismuth are undoubtedly the best means for controlling excessive diarrhea, but care should be taken that they are not pushed to the degree of inducing constipation.

Constipation early in the disease may be relieved by castor oil, but all active purgation should be avoided. Later in the disease irrigation of the colon with tepid water is better than anything else. On the whole, constipation is more troublesome to overcome than diarrhea.

Tympanites does not often require treatment; it may be relieved by turpentine stupes, by a glycerin suppository, or a small glycerin injection (one teaspoonful of glycerin to four ounces of water), or, better still, by

the use of the rectal tube. If the distention is continuous and extreme it may be necessary to stop all food for several hours until it is relieved.

Whenever the temperature remains above 104° F., antipyretic measures are indicated. In mild cases cold or tepid sponging is generally sufficient. In those which do not yield to such measures, baths may be employed. Not all children bear baths well, and considerable discretion should be used in employing them. One should be guided quite as much by the effect upon the pulse and the nervous system as by the temperature. The best method is usually the graduated bath; the child is placed in the tub with the water at a temperature of 95° or 100° F.; this is gradually lowered to 95°, 90°, or even 85° F., but seldom lower. The body should be actively rubbed while the child is in the bath, to prevent shock and cardiac depression. The pack may be substituted for the bath when circumstances make the latter impracticable. The bath or pack should be repeated in an average case in from three to six hours.

The milder nervous symptoms—headache, restlessness, sleeplessness, etc.—may be relieved by an occasional dose of phenacetin, either alone or in combination with the bromids, or by cool or tepid sponging; the more severe ones usually occur with high temperature, and are best controlled by the bath.

Stimulants in most of the cases are not called for. They are to be given according to the indications afforded by the pulse, the first sound of the heart, and the child's general condition. They are seldom needed earlier than the end of the second week. Intestinal hemorrhage calls for absolute quiet, morphin hypodermically, and an ice-coil to the abdomen, nothing being given by mouth except stimulants and possibly opium. Intestinal perforation is successfully treated only by early laparotomy.

CHAPTER X

TUBERCULOSIS

Tuberculosis is an infectious, communicable disease due to the bacillus tuberculosis of Koch. It may be local or general, and may involve any organ and almost any structure in the body.

Etiology.—*Age and Frequency.*—No age is exempt from tuberculosis. It was formerly believed that the disease was rare in infancy, but recent observations have shown the opposite to be the case.

Statistics taken chiefly from three New York institutions where only infants and young children are received give the following figures for 382 cases of tuberculosis, the diagnosis being confirmed by autopsy in

nearly every instance: In the first year there were 160 cases, and of these 67 were under six months, 10 of which were under three months of age. The frequency of tuberculosis appears to increase steadily as age advances, as shown by the following table, in which results found by Veeder and Johnston in St. Louis are compared with those of Hamburger and Monti and von Pirquet in Vienna. The cutaneous or intracutaneous test was applied in all instances. Cases of clinical tuberculosis were excluded.

Age (years).	Veeder and Johnston, St. Louis.		Hamburger and Monti Vienna.		von Pirquet, Vienna.	
	No. of Cases.	Percentage of Tuberculosis.	No. of Cases.	Percentage of Tuberculosis.	No. of Cases.	Percentage of Tuberculosis.
Under 1	202	1.5	23	0	388	0
1 to 2	109	5.5	46	9	89	0
2 to 4	163	19	131	27	162	13
4 to 6	172	23	113	51		
6 to 8	152	29	76	61	343	37
8 to 10	126	30	61	72		
10 to 12	107	34	48	94	147	70
12 to 14	94	38	34	94		
	1,125	21	532	51	1,129	22.5[1]

[1] The total incidence of tuberculosis is small on account of the large number of infants tested.

From the facts at hand it would seem that the percentage of children with tuberculosis is much greater in Europe than in this country. The following table gives figures for three institutions in New York, as compared with data taken from Vienna and Munich. The difference in the ages of the children makes comparison difficult.

Frequency of Tuberculosis as Shown by Autopsies

Institution.	Age of Patients.	No. of Autopsies	No. Showing Tuberculosis.	Percentage Showing Tuberculosis.
N. Y. Infant Asylum	Nearly all under 2½ years	726	56	8.0 per cent.
Babies' Hosp., 1st series	" " " 3 "	1,000	168	16.8 "
Babies' Hosp., 2d series	" " " 3 "	1,320	178	13.5 "
N. Y. Foundling Hosp.	" " " 3 "	1,000	136	13.6 "
Müller—Munich	Children of all ages	500	200	40.0 "
Hamburger—Vienna	All ages up to 14 years	848	335	40.0 "
" "	Including only children of 2 years and under.	497	120	24.4 "

These percentages are not to be taken to represent the occurrence of tuberculosis in the community generally, but only its frequency in

the class which furnishes hospital and institution inmates. Nor are these figures to be interpreted as showing the percentage of active tuberculosis. In the cases showing tuberculosis at autopsy nearly one-third of the number died from other diseases, tuberculosis being latent and its existence being discovered only post mortem. Likewise in nearly one-fifth of the cases giving positive skin reactions there were no evidences of active tuberculosis.

Predisposing Causes.—These include all the conditions which bring about a diminished resistance of the body to tuberculous infection. This susceptibility may be inherited, as when parents have suffered from tuberculosis or other constitutional disease—syphilis, alcoholism, etc. It may be due to the fact that children have been reared in crowded city tenements, in institutions, or under other unfavorable surroundings. A local predisposition may be afforded by any pathological condition of the organs or mucous membranes exposed to infection. Thus, adenoid growths of the pharynx or large tonsils favor the development of tuberculosis of those structures and secondarily of cervical adenitis; and frequent attacks of bronchitis may precede pulmonary tuberculosis. Certain infectious diseases, particularly measles, whooping-cough, and influenza, increase a child's susceptibility to tuberculosis, but they chiefly cause a latent tuberculosis to develop into an active process. General or pulmonary tuberculosis is therefore often seen as a sequel to the diseases mentioned, the latent focus for which has been tuberculous bronchial glands.

Modes of Infection.—Intra-uterine infection, although rare, has been established by the report of a number of complete and well-authenticated cases. Tuberculosis of the placenta is more frequent. In most of the cases of congenital tuberculosis the mother has been suffering from the disease in an advanced form, and the child is either still-born or dies soon after birth. Besides tuberculosis of the placenta, tubercle bacilli are found in the organs of the child, and, when life is prolonged, there are generalized lesions showing infection through the blood. Cheesy nodules have been observed in the umbilical cord. Intra-uterine infection is highly probable in many of the children born of tuberculous mothers, who develop the disease during the first few months of life, although they may show no evidence of it at birth. Among our own cases there was one only twenty days old and another six weeks old. The children were born prematurely of mothers suffering from advanced tuberculosis. Besides other lesions, the autopsy showed, in the case of one mother, tuberculosis of the endometrium.

Tuberculosis may be communicated by direct inoculation, as in the case of a bite from a person suffering from the disease, several instances of which are on record. The rite of circumcision performed by a rabbi

suffering from tuberculosis we have known to cause the disease. One of the most striking instances of direct infection is that reported by Reich. In a town of about 1,300 inhabitants, the obstetric practice was divided between two midwives. Within fourteen months no less than ten infants, who had been delivered by one of these women, died of tuberculous meningitis. In none of these families was there a history of tuberculosis. This midwife was found to be suffering from pulmonary tuberculosis, and died from that disease. It was her custom to remove the mucus from the mouth of the newly-born infants by direct mouth-to-mouth aspiration, and then to establish respiration by blowing into the nose. In the practice of the other midwife, who was healthy, no cases of tuberculosis occurred, although she treated the newly-born infants in the same fashion.

Altogether the most frequent means by which young children acquire tuberculosis is from association with persons suffering from pulmonary tuberculosis. Some of these are persons in the active stage of the disease; many are supposed to have been cured; in others the disease has not yet developed so as to be recognized. Bacilli may be directly conveyed by kissing. Dried sputum containing bacilli may become a part of the dust of the room; it may be inhaled or it may be introduced into the mouths of children by hands, toys, or other objects. The source of infection is usually one or other parent or some member of the household—a nurse, caretaker, servant, or a frequent visitor. A history of such exposure was definitely traced in forty-four per cent of 101 consecutive cases of tuberculosis in young children which were investigated at the Babies' Hospital. These figures do not represent the proportion of the cases in which the disease is so contracted. There is a very much larger number in which this connection can not be traced. Doubtless exposure antedates symptoms by a number of weeks at least, often by several months. In instances where it could be pretty accurately ascertained, the interval between exposure and development of symptoms was from four to twelve weeks.

Infection may take place from beds, rooms, sleeping cars, or any apartments previously occupied by tuberculous patients; from dishes or spoons, from glasses at public drinking places; also though very rarely from the meat of tuberculous cattle. Our own observations lead us to the conclusion that only a very small proportion of children contract tuberculosis in these indirect ways. Infection through milk is, however, of not infrequent occurrence. (See Chapter II, page 134, The Infant's Dietary.) It has been repeatedly shown that a considerable percentage of the milk offered for sale in cities contains tubercle bacilli. In almost all instances they are of the bovine type. However, they are usually present in small numbers and in most cases

doubtless pass through the digestive tract without inducing infection.[1]

Types of Bacilli.—Important information in regard to the source of infection is obtained from a study of the type of organism present in the different varieties of tuberculosis.

Park and Krumwiede give the following table of results of 513 cases of tuberculosis in children studied. About one-third of these were investigated by them personally; the remaining two-thirds were collected cases.

Lesions	Children Under 5 years		5 to 16 Years.	
	Human	Bovine	Human	Bovine
Pulmonary	35	1	14	0
Adenitis, axillary or inguinal	2	0	4	0
Adenitis, cervical	15	24	36	22
Abdominal	10	14	8	9
Generalized	74	7	5	1
Generalized, alimentary origin	17	15	3	4
Generalized and meningeal, alimentary origin	5	10	1	0
Generalized and meningeal	76	1	10	0
Meningeal	28	4	3	0
Bones and joints*	27	0	41	3
Skin	2	0	4	6
Genito-urinary	0	0	2	0
	291	76	131	45

* Frazer states that "of a series of cases of bone and joint tuberculosis studied in Edinburgh 62 per cent were bovine in their origin." Apparently the incidence of bovine infection varies considerably in different countries. The inference is that the milk supply of Scotland is more likely to be infected than that of other places.

These figures indicate that nearly all pulmonary and meningeal tuberculosis as well as tuberculosis of bones and joints is human in origin,

[1] In this connection the following incident is interesting as bearing upon the other side of the question: Near a large American city was a fancy stock farm of registered Jersey cows, which supplied milk for table use and infant feeding to a large number of families in the wealthiest part of the city, for a period of over ten years. At the end of that time the tuberculin test was used for the first time, and 45 per cent of these cows were found to be tuberculous, and were killed by order of the State Board of Health. The diagnosis was confirmed by autopsies upon the animals in every instance. An investigation was instituted among the children who had been fed upon this milk, but in only one case of many hundreds could it be learned that tuberculosis had developed, and in this instance it was by no means established that the milk had been the source of infection. It should be stated that this was before the days of sterilizing milk for infant feeding. Besides the families who took the milk in the manner mentioned, the employees at the farm were accustomed to drink the skimmed milk in large quantities daily as a beverage in the place of water. Many of them continued to do this for years, and yet not one of them developed tuberculosis.

but that on the other hand, tuberculosis affecting chiefly the abdomen or springing from the alimentary tract, and tuberculosis of the cervical glands is frequently bovine in origin.

Infection from the meat of tuberculous animals is a possibility, but hardly more. Bollinger's experiments in feeding animals with the expressed juice of such meat gave negative results.

Paths of Infection of the Tubercle Bacillus.—Tubercle bacilli may gain entrance to the body through the respiratory or the alimentary tract or the skin, the last, however, being so rare that it needs only to be mentioned. In infancy and early childhood infection is undoubtedly most frequent through the respiratory tract. The situation of the primary lesions strongly supports this view. The infection is the result of the inhalation of tubercle bacilli, probably in dried sputum, and is therefore nearly always an infection with the human type of the tubercle bacillus. Infection through the alimentary tract is by way of the tonsils or the intestines, and either the human or bovine type of organism may be introduced into the body in this way. If it is the human type, in all probability the patient himself is suffering from pulmonary tuberculosis and the tonsils or the intestines are infected from the sputum coughed up. There is also the possibility of human tubercle bacilli being taken into the mouth from contaminated articles or in milk. Bovine infection almost always results from drinking milk from tuberculous cows.

Animal experiments have shown conclusively that bacilli may pass through a mucous membrane without inducing either a macroscopical or microscopical form of tuberculous disease but that penetration is much easier if the mucous membrane is the seat of a catarrhal inflammation or if the epithelium has been injured. While it is possible that infection of the cervical, mediastinal and tracheobronchial glands may take place without a lesion of the mucous membrane which these lymph nodes drain, recent studies have shown that it is very uncommon. Thus, with tuberculosis of the cervical glands, pathological examination of the tonsils and inoculation experiments show that the tonsils are usually the seat of tuberculous disease. The same is true of the mesenteric glands. To superficial examination, the mucous membrane of the intestinal tract may appear normal; but careful examination of it has in our experience almost always resulted in the discovery of one or more tuberculous lesions. Such is the case also with the lungs, as shown by Parrot, Hervouët, Küss, H. Albrecht and Ghon. The tubercle bacilli which pass the upper respiratory tract may not be arrested until the smaller bronchi are reached. In one of these they set up a localized tuberculous process which may remain very small, but frequently reaches the size of a pea. This area undergoes the ordinary changes induced by the tubercle bacilli and eventually necrosis or perhaps calcification occurs. The tuberculous focus

is frequently surrounded by fairly firm fibrous tissue. From this original pulmonary focus, infection of the tracheobronchial glands takes place by way of the lymphatics. The focus may remain small and apparently innocuous. Further development of the tuberculosis may take place from the tracheobronchial glands, either in the form of a diffuse inflammation spreading into the parenchyma of the lung along the lymphatics, or from the softening and rupture of the gland either into a bronchus or into a vein. The original tuberculous lesion in the lung on account of its small size may be overlooked, but careful examination will usually disclose it. In a series of 169 autopsies at the Babies' Hospital upon children (mostly infants) with tuberculous bronchial glands, Bartlett and Wollstein found pulmonary lesions in 158 cases, or 93.5 per cent. Ghon found, in 184 autopsies upon children with tuberculous bronchial glands, a primary pulmonary focus in 170, or 92.4 per cent. It was his opinion that more careful examination would probably have revealed the focus in others. The changes in the tuberculous tracheobronchial glands are those of ordinary tuberculosis elsewhere—congestion, swelling, cell proliferation and caseation or the process may be arrested at any point and the products of inflammation become encapsulated by the proliferation of fibrous tissue in which condition they may remain latent in the body for an indefinite number of years, possibly for a lifetime. This occurs in many children and is consistent with every outward sign of health, but it is a smouldering ember which at any time may be fanned into flame under the stimulus of an inflammation excited by some other cause.

Lesions.—In the table (p. 1074) are given the lesions found in 255 autopsies, of which we have notes. These represent the lesions of infancy and early childhood, seventy per cent of these children being two years old or under. For comparison there are given statistics of 131 autopsies from the Pendlebury Hospital, Manchester, England. Few of the children in this series were under three years old. The greater frequency of abdominal tuberculosis, especially tuberculous peritonitis, will be noted. This difference obtains in nearly all the English statistics of the disease.

The Varieties of Tuberculosis seen at Different Ages.—During the first two years of life, tuberculosis most frequently involves the lungs and bronchial lymph nodes. It is the meningeal or pulmonary process which most often is the cause of death. Death from other forms of tuberculosis is rare at this time of life. Of 232 deaths from tuberculosis in the first three years of life, meningitis was the cause in 93, tuberculous peritonitis in only one, and hemorrhage from a tuberculous ulcer of the intestine in one.

After the second year, tuberculosis of the bones, cervical and mesen-

Frequency of the Different Visceral Lesions of Tuberculosis

Organs.	Personal Cases;[1] 255 autopsies (chiefly under three years).		Pendlebury Hospital Reports; 131 autopsies (chiefly over three years).	
Lungs	235	92.1 per cent	122	93.0 per cent
Pleura	93	36.5 "	100	76.0 "
Bronchial lymph nodes	208	81.5 "	91	70.0 "
Brain	85	33.3 "	60	46.0 "
Liver	178	69.8 "	86	65.0 "
Spleen	191	74.9 "	76	58.0 "
Kidneys	88	30.6 "	54	41.0 "
Stomach	7	2.7 "	1	0.8 "
Intestines	110	43.1 "	65	50.0 "
Mesenteric lymph nodes	118	46.2 "	77	59.0 "
Peritoneum	22	8.6 "	37	28.0 "
Pericardium	10	3.9 "	4	3.0 "
Endocardium	1	0.4 "	...	.....
Thymus	5	1.9 "	...	.....
Suprarenal capsules	4	1.5 "	2	1.6 per cent
Pancreas	4	1.5 "	...	.....

[1] In a second series of 178 autopsies at the Babies' Hospital the lungs were involved in 92.1 per cent.; the bronchial lymph nodes in 95.5 per cent.; the brain in 38.7 per cent., and the mesenteric lymph nodes in 63.5 per cent.

teric lymph nodes, peritoneum, and intestines becomes more frequent, and any of them may occur as the principal lesion, although at autopsy the lungs are usually involved to some degree.

Pulmonary Lesions.—As compared with that of adults, the pulmonary tuberculosis of young children is more widely diffused, and the predominance of cases in which the lesion is in the upper lobes is less marked, though it still exists. In those who have passed the sixth or seventh year, the pathological processes resemble those of adult life. Although localized tuberculous processes are frequently met with in patients dying from other diseases, those who die from tuberculosis usually show wide-spread lesions of the lungs.

1. Miliary Tuberculosis of the Lungs.—In nearly every case of pulmonary tuberculosis, miliary tubercles are found in some part of the lung, usually upon the surface and in the vicinity of some older process. Occasionally, they are distributed throughout nearly the whole of both lungs. In some places the lung, with the exception of these numerous gray granulations, appears quite normal; in others it is congested, and shows between the tubercles the lesions of simple bronchopneumonia in its various stages. There is also an acute bronchitis of the middle-sized and smaller bronchi. The microscope shows that the tubercles usually develop in the walls of the small bronchi or the blood-vessels. In their gross appearance, the lungs in these cases resemble those in ordinary acute bronchopneumonia, with the exception that everywhere upon the

surface and throughout the substance of the lung are seen the small gray granulations, and in most cases some small yellow tuberculous nodules. The pleura is usually normal except for the presence of the tubercles. This form of the disease represents the rapid dissemination of tubercle bacilli throughout the lungs, the miliary tubercles being the result of the inflammation excited by their presence.

2. Tuberculous Bronchopneumonia.—This is the most frequent and the most characteristic form of tuberculosis in infants and young children, and it is the one which at this age usually causes death. In this form of the disease there are produced in the lung caseous nodules, or larger caseous areas, some of which have usually undergone softening by the time the case comes to autopsy. The process generally runs a somewhat subacute course. With the lesions mentioned there are always associated those of simple bronchopneumonia.

The pleura is involved in almost every case. There may be simply dense connective tissue adhesions which bind the lung firmly to the chest wall, the diaphragm, and the pericardium, or the pleura may be greatly thickened and contain caseous deposits. Occasionally empyema is seen, but it is almost always sacculated and small.

Both lungs are usually involved, but one to a much greater degree than the other. There are found large areas of consolidation which sometimes involve an entire lobe, but more often smaller areas are seen in several lobes. These portions of the lung appear much firmer and harder than in ordinary pneumonia. The upper lobes are more often affected than the lower, and especially that part of the lobe which is near the root of the lung, on account of its frequent association with tuberculosis of the bronchial glands; the disease very often extends forward from this point to the middle lobe of the right, or the corresponding part of the left lung. On section the affected part of the lung usually shows many caseous nodules, varying in size from a pin's head to a walnut, which are of a pale-yellow color, and resemble caseous lymph nodes. They contain giant cells and are usually filled with bacilli, those which have softened containing yellow pus. There is nearly always seen in some part of the lung a large caseous area; and not infrequently there may be diffuse caseation of almost an entire lobe (Figs. 174, 175). Sometimes no spot of softening is seen even in these large areas, but in many cavities are present.

Softening and excavation represent the final stages of the process in tuberculous pneumonia. Softening usually begins in the center of a caseous part, often at several points at the same time. Areas of excavation large enough to deserve the name of cavities were present in about half of our autopsies upon tuberculous patients, two years old and under. They vary in size from a cherry to a hen's egg, and sometimes a much

larger one is seen (Fig. 174). They are usually rather deeply seated, and are partially or entirely filled with caseous masses or pus, but very seldom perforate the pleura, causing pneumothorax or pyopneumothorax. It is rare in a young child to find cavities surrounded by dense fibrous walls such as are seen in older children or in adults; for in infancy the

Fig. 173.—Tuberculous Pneumonia. A vertical section through the middle of the right lung of a child thirteen months old. The greater part of the upper lobe is uniformly caseous—a diffuse tuberculous pneumonia; near the center the commencement of a cavity is seen; below it has the appearance of a consolidation from simple pneumonia. The part of the lower lobe shown is normal.

Fig. 174.—Cavity from Breaking Down of Tuberculous Pneumonia. Another view of the same lung, the section being made very near the posterior border of the lung. The cavity occupies at this point nearly the whole of the upper lobe. At autopsy this cavity contained numerous loose caseous masses, the largest being the size of a marble. The lower lobe is normal. (For history, see Fig. 179.)

process of softening once begun usually advances steadily until the death of the patient.

The bronchial lymph nodes are in these cases invariably found to be tuberculous, and not only those at the root of the lung, but if a dissection is made, a chain of these tuberculous glands will be found to follow the larger bronchi for some distance into the lung (Fig. 175). Sometimes

one may be discovered which has softened and ulcerated through into a small bronchus.

Microscopical examination of these cheesy nodules shows that they most frequently begin as tuberculous deposits in the walls of the small bronchi, either in the mucous membrane, the fibrous coat, or the lymphatics; sometimes, however, they begin in the walls of a small vein or artery.

FIG. 175.—PULMONARY TUBERCULOSIS, EXTENSIVE CASEATION OF LEFT LUNG AND BRONCHIAL GLANDS. *History.*—Colored child, 2½ years old; signs over left lung were feeble breathing and flatness, suggesting empyema; twenty-three examinations of the sputum made for bacilli, all negative. For the last three and a half weeks, temperature showed a regular daily range from 100° to 104° F.

Autopsy.—Almost complete caseation of left lung; no spots of softening; throughout right lung were small tuberculous nodules and miliary tubercles. Bronchial glands very large and caseous, but none broken down; those affected were not only the group at the root of the lung but the chain following the main bronchus some distance into the lung itself,

Cell proliferation takes place, separating the coats of the bronchus or blood-vessel, and partly or entirely obstructing its lumen. Softening may take place and the contents be discharged into the bronchus or blood-vessel. About this focus other changes of an inflammatory character occur, as a result of which each cheesy nodule is surrounded by a zone of simple bronchopneumonia which tends, in a measure at least, to limit the tuberculous process. The larger caseous areas are formed by an

extension of this process to the zone of pneumonia which surrounds it; but in its further growth it is still preceded by a simple pneumonia. The rapidity with which the lesions advance differs much in the different cases; in infants the progress is apt to be continuous until the death of the patient; in older children it is usually slower, and interrupted by intervals of arrest and even of partial retrogression.

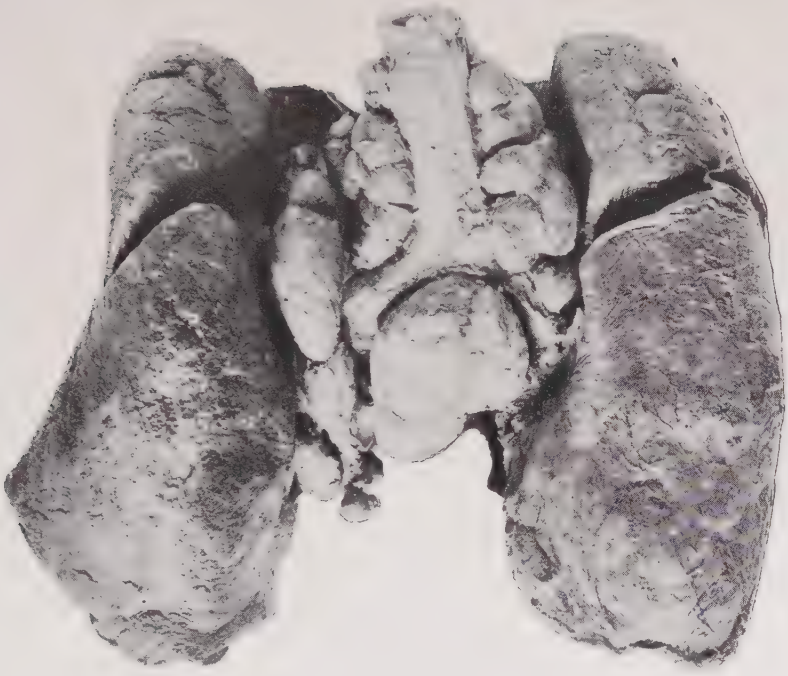
Not infrequently one sees in the post-mortem room one or two caseous, or less frequently calcareous, nodules encapsulated by firm, organized connective tissue when a most careful search fails to show any other tuberculous lesion in the lung. If, however, the nodules are widely scattered through the lung, such an arrest of the process is not to be expected.

3. Chronic Pulmonary Tuberculosis, Chronic Phthisis.—In children who have passed the seventh or eighth year the pathological process resembles that seen in adults; but in younger children, and especially in infants, nothing corresponding to it is met with.

At this period the nearest approach to this condition is seen in the cases of tuberculous bronchopneumonia, which run a slow, irregular, and somewhat chronic course. The essential features of the process in these patients is a chronic interstitial bronchopneumonia with tuberculous nodules which rarely undergo softening, but usually become encapsulated.

The gross lesions closely resemble those of simple chronic bronchopneumonia. There are the same generalized pleuritic adhesions and the shrunken cicatricial condition of the part of the lung most affected, with bronchiectasis, compensatory emphysema, etc. The tuberculous nodules are old and for the most part converted into dense fibrous tissue, in the center of which, however, some softened, caseous areas are often seen.

Bronchial Lymph Nodes (*bronchial glands*).—The prominence of the lesions of the lymph nodes is one of the most striking features of tuberculosis in infancy and early childhood. Those which are most frequently affected are connected with the bronchi. The lymph nodes, to which the term "bronchial glands" is generally applied, consist of three groups: the first of which surrounds the trachea; the second is situated at the bifurcation of the trachea and surrounds the primary bronchi; while the third follows the course of the bronchi into the lung, being found, according to anatomists, as far as the fourth division. The anatomical relations of the different groups should be borne in mind, since upon them the symptoms principally depend. The first group, or the peritracheal lymph nodes, are in relation with the superior vena cava, the pulmonary artery, the pneumogastric and recurrent laryngeal nerves; the second group, at the bifurcation of the trachea, with the esophagus, pneumogastric nerve, and aorta; the third group, with the bronchi and the branches of the bronchial and pulmonary arteries and veins.



TUBERCULOSIS OF THE TRACHEOBRONCHIAL LYMPH NODES

From a fairly nourished child, four months old, who was under observation for three weeks, with slight fever and a most severe, teasing, dry cough, which was almost constant, and upon which no treatment seemed to have the slightest effect. At first there were no signs of disease in the lungs; later there were a few coarse scattered râles.

There were small tuberculous deposits throughout both lungs, with quite a large area of cheesy pneumonia in the right middle lobe, and scattered miliary tubercles in other organs.

All the groups are usually involved at the same time, but in varying degrees, and in most cases those belonging to one lung to a greater extent than the other; in our own cases those of the right side have much more often been involved than those of the left. There may be simply two or three tumors as large as a hazelnut, or there may be a mass two or three inches in diameter, which is made up of ten to twenty of these nodes fused together by inflammatory products, completely surrounding the trachea and both the large bronchi. It is rare that the individual glands are more than an inch in diameter, and most of them are smaller than this. A well-marked but not unusual example of this condition is shown in Plate XIII. There is usually found a chain of these tuberculous glands following the course of the large bronchi for some distance into the lung; sometimes these are almost as large as the external group (Fig. 175); at other times they are not noticed unless a somewhat careful dissection is made. The process is not infrequently more advanced in these deeply seated glands than in those situated at the root of the lung; and lesions here are also more important, as it is very frequently from them that an extension of the process takes place.

The pathological changes through which these glands pass as a result of tuberculous infection are very similar to those already described with reference to the cervical glands. Suppuration is less frequent than in the region of the neck, while calcific degeneration is much more so. This applies especially to children over three years old. In infancy suppuration is not infrequent in the bronchial glands, while at this age calcification is relatively rare. Although the process has gone on to caseation, these inflammatory products with bacilli may become encapsulated, and may remain innocuous for an indefinite period. The bacilli may die or may exist here, living, for years. At any time the old process may be lighted up, and a more or less rapid dissemination of tubercle bacilli take place through the lungs or through the whole body. Latent tuberculosis more frequently exists in the bronchial lymph nodes than in any other structure in the body.

Secondary lesions may be produced by these lymph nodes. The pneumogastric and recurrent laryngeal nerves may be surrounded by one of these cheesy masses which may cause pressure or irritation. The esophagus, the trachea, or the bronchi may be compressed or opened by ulceration. The superior vena cava usually suffers only compression, but this or any of the other large vessels may be opened. Ulceration may also take place into one of the large or small bronchi or the trachea. If the gland has softened and broken down, and if the bronchus is a small one, the only result of this may be a rapid spreading of tuberculous infection throughout the lung. If sudden rupture occurs, a large caseous mass may escape into the trachea, or a large bronchus, with a result similar to that pro-

duced by any other foreign body. If suppuration occurs, the abscess may rupture into the surrounding cellular tissue, causing mediastinal or retro-esophageal abscess. This may open externally at the suprasternal notch, or in the first or second intercostal space, or may ulcerate into any of the large vessels, the esophagus, or the pericardium.

Pleura.—This is rarely normal in any case of tuberculosis. In acute general tuberculosis the only lesion may be a deposit of miliary tubercles upon the visceral pleura. In most of the other cases there are found fibrous adhesions over the part of the lung involved, binding it to the pericardium, the diaphragm, or the chest wall. The amount of thickening of the pleura varies a good deal, but is rarely great. Pleurisy with a serous effusion is not common in infants or young children; when it occurs it is apt to be sacculated. Hemorrhagic exudation is very rare at this age. Empyema is also rare, being seen in but five per cent of our cases, and then it has been small and sacculated. Pneumothorax and pyopneumothorax are very rare in children under three years of age.

Heart.—It is exceptional for the pericardium to be affected even in the most generalized forms of acute miliary tuberculosis. In such cases the usual lesion is a deposit of a few gray tubercles upon the visceral surface. In chronic cases other lesions analogous to those of the pleura may be seen, but very infrequently in childhood. Usually only localized adhesions are present, but we have seen complete obliteration of the pericardial sac from tuberculous inflammation in an infant of eleven months.

In several instances we have seen miliary tubercles and minute cheesy nodules upon the mural endocardium, most frequently in the conus arteriosus of the right ventricle. One case, an infant sixteen months old, had such lesions in both ventricles and in addition miliary tubercles upon the tricuspid valve.

Brain.—Tuberculosis of the brain is very common during infancy, being then associated in nearly all cases with general tuberculosis. Miliary tubercles are occasionally found in small numbers in cases which have presented no symptoms. The lesions of tuberculous meningitis have already been described. Cheesy nodules are rare in infancy, being noted in but 2.5 per cent of our own autopsies, which were mainly on children under three years old; while in the Pendlebury Hospital cases, including those between four and twelve years old, they were noted in 24.4 per cent. These nodules vary in size from a pea to a hen's egg; they are usually associated with tuberculous meningitis, but they may exist alone. When they are large they rank as cerebral tumors, being most frequently seen in the cerebellum.

Liver.—This is frequently involved in general tuberculosis, although it is doubtful if it is ever the seat of primary infection except in the congenital cases. Usually the only lesion is the presence of miliary tubercles

on its surface and in its substance, and in most cases these are not numerous. They are found in about two-thirds of the cases. In a smaller number there are tuberculous nodules of various sizes, especially about the biliary ducts. In nearly every protracted case the liver is markedly fatty. In very late cases of tuberculosis of the bones, it is frequently the seat of amyloid degeneration.

Spleen.—This is more frequently affected than the liver, but the lesions are similar. The size of the spleen is not much increased if only miliary tubercles are present; but with tuberculous nodules it may be greatly enlarged. Amyloid degeneration is found under the same conditions as in the liver.

Stomach.—Tuberculosis of the stomach is one of the rare lesions; both its contents and its acid reaction seem to protect it against direct infection from the mouth. Tuberculous ulcers were seen in five of our autopsies, which is a larger proportion than is usually noted.

Intestines.—That these are less seriously affected in infants than in older children is rather surprising when we consider how susceptible are the intestines of infants to other forms of infection. The explanation of this difference seems to be that in infancy intestinal infection is usually secondary to disease of the lungs, primary lesions being relatively rare. Infants die from the more rapid tuberculous processes in the lungs or brain before there has been time or opportunity for secondary intestinal lesions of importance to occur. The intestinal lesions and those of the mesenteric lymph nodes with which they are almost invariably associated, are described elsewhere.

Peritoneum.—In early infancy the peritoneum is not often involved even in general tuberculosis, and at this age it is very rare for it to be the seat of the principal tuberculous process. In older children it is more frequent. In most cases of general tuberculosis there are only deposits of miliary tubercles; less frequently there are tuberculous nodules with other inflammatory products. The lesions in these cases are described with Diseases of the Peritoneum.

Thymus Gland.—In several of our cases tuberculous nodules have been found in the thymus gland, the size varying from a small pea to a hazelnut. All the cases showed also widely disseminated tuberculous lesions.

Pancreas.—In a very few of our cases this organ also was the seat of small tuberculous nodules, all of them being cases of general tuberculosis.

Urogenital Organs.—Serious tuberculosis of any part of the urinary tract is very rare in children. Miliary tubercles have been found in the kidneys in about one-third of our autopsies on tuberculous patients. They are generally few in number. Large tuberculous nodules of the kidney are very rare before the fourteenth year. Tuberculous nodules are rarely

found in the suprarenal capsules. Tuberculosis of the testicle is very rare in children. We have seen but a single instance of it. This was in an eight months old child. We have records of two cases of tuberculosis of the prepuce and inguinal glands following ritual circumcision, in both cases followed by generalized infection.

Tuberculosis of the bones and of the external lymph nodes has already been described.

THE CLINICAL FORMS OF TUBERCULOSIS

I. General Tuberculosis.—Cases of tuberculosis present a wide variety in their symptomatology, depending upon the seat of infection, the rapidity with which the bacilli are disseminated through the body, or the numbers in which they enter. The general symptoms often precede the local ones, but are not recognized as those of tuberculosis. Often it is not suspected until the process is quite well advanced in some one organ.

In Infants.—The early symptoms in infancy are often only those of failing nutrition. The patients are pale, thin, do not gain in weight no matter how fed, and finally lose steadily without sufficient reason. There may be no cough or fever sufficient to attract attention, and the case may even go on to a fatal termination without anything else than simple marasmus having been suspected, tuberculosis being first recognized at the autopsy.

More frequently, however, there are developed toward the end of the illness both the symptoms and signs of pulmonary disease and fever. These are generally found together, as the process in the lungs is usually the cause of the rise of temperature. The febrile symptoms are often not seen until the last few weeks of life. The course of the temperature is irregular. It is never of the hectic type and rarely high. The usual range is between 100° and 102° F. The pulmonary symptoms are generally few and not very well marked. There is some cough, but it is rarely severe. The breathing is more rapid than would be explained by the temperature alone. Severe dyspnea and cyanosis are rare, and are seen only at the close of the disease. The physical signs are those of either localized or general bronchitis. Digestive symptoms are usually present late in the disease, but diarrhea is rarely due to a tuberculous lesion of the intestines.

The progress of the case after constitutional symptoms develop is usually steadily downward, and the child lives but a few weeks at most. Death generally occurs from progressive asthenia without the development of any new symptoms, or cerebral symptoms rapidly develop and the child is carried off in a few days by tuberculous meningitis. Some-

times there is a rapid spreading of the disease in the lungs, and death occurs with symptoms of acute pneumonia.

General tuberculosis in infants is to be differentiated from marasmus with bronchitis; rarely it may be confounded with hereditary syphilis.

In Older Children.—The development of active general tuberculosis in older children is usually preceded by a protracted period of indefinite symptoms. They are persistently anemic without evident reason; they lose weight; digestion is disturbed; the appetite is capricious; they sleep badly; they are irritable, fretful, and easily fatigued. These symptoms indicate only a gradual decline in general health, and may readily be explained by many other causes than tuberculosis. They should, however, excite a suspicion of tuberculosis in a child who by surroundings or inheritance is predisposed to that disease.

After these indefinite symptoms have lasted for a few weeks fever is added. Sometimes the prodromal symptoms are absent or unnoticed, and fever is the first evident symptom. From the beginning of fever some cases progress rapidly to a fatal termination in three or four weeks. In the majority, however, the disease runs a slower course. The fever often exists without evident cause and without any local manifestations of disease. The temperature is not often high, but it is continuous. The tympanites and the rose-colored spots are not present, but the general aspect of the patient is strikingly suggestive of typhoid fever. But the course of the temperature and the duration of the illness show that we have to deal with some other condition.

After the fever has lasted from one to three weeks there develop some signs of localized tuberculosis, generally in the lungs, or the fever may decline gradually, and although the patient improves he does not get well. He is still weak and does not gain in weight, and the thermometer shows the existence of a very slight amount of fever. Before long he may grow rapidly worse and the course of the temperature becomes irregular, with alternate exacerbations and remissions. Such an irregular and inexplicable fever sometimes puzzles the physician for several weeks before the characteristic features which stamp the process as tuberculous are present. Before very long wasting is added to the fever. This may not be rapid, but is progressive. The tuberculous cachexia is frequently unmistakable; but in most of the cases one must wait for the process to advance far enough in some one of the organs to give local signs or symptoms before he can be sure of tuberculosis. In four cases out of five this is in the lungs, and frequently repeated examinations of the sputum may reveal the bacilli. Less often it is in the peritoneum, the brain, or a general infection of the lymph glands throughout the body. If in the lungs, the process manifests itself as a bronchopneumonia whose tuberculous character may sometimes be suspected from its location—the

apex or the middle of the lung in front—but chiefly from the fact that the general symptoms, fever and wasting, have so long preceded the local signs. From this time, the course may be that of a typical tuberculous bronchopneumonia.

If the tuberculous process is localized in the brain, there may be vomiting, headache, drowsiness, irregular pulse, irregular respiration, and finally convulsions and coma—in short, the symptoms of tuberculous meningitis; if in the peritoneum, there are abdominal distention from gas or fluid, tenderness, pain, diarrhea, or constipation; if in the lymph glands, there is a general enlargement of those situated externally, sometimes with symptoms indicating similar changes in those at the root of the lung.

II. Pulmonary Tuberculosis.—Tuberculosis of the lungs in children may be seen in a variety of clinical forms which correspond with the different pathological conditions. The pathological conditions are often associated, yet the main clinical types are sufficiently distinct to give quite a definite picture. These types are: (1) miliary tuberculosis of the lungs; (2) bronchitis with small, scattered, tuberculous nodules; (3) tuberculous bronchopneumonia with areas of consolidation, often extensive, which may be followed by caseation and excavation, or by chronic fibrous induration.

Miliary Tuberculosis of the Lungs.—This is not a common form of pulmonary tuberculosis, but may be met with even in young infants. Both the general and pulmonary symptoms and the physical signs are rather obscure and indefinite, and often the diagnosis is not made. Occasionally the only symptoms are those of marasmus, neither fever nor physical signs in the chest being present (Fig. 176). As we have seen it in young children, it has seldom been attended by high temperature, 101° to 103° F. being the usual range. Throughout the greater part of the disease it is often lower than this, and toward the close perhaps rather higher. It is not a hectic type of fever, and it seldom touches the normal line.

The duration of the disease in these cases, after fairly definite symptoms begin, varies from ten days to a month. At first, and often for two or three weeks, the temperature is almost the only symptom. Cough is slight, inconstant, and seldom loose. There is no sputum. The respirations are only moderately accelerated, in many cases not enough to draw attention to the lungs as the seat of disease. There is no rapid wasting, the loss in weight being usually not more than would be expected with any other febrile disease. None of the other symptoms suggests tuberculosis. The usual problem in diagnosis is to discover the cause of the fever. Often the most careful examinations of the chest made daily reveal nothing more than a few scattered râles. These change

in position from time to time, and it frequently happens that for days no râles are heard. After the disease has progressed somewhat further, the liver and spleen are generally enlarged. Cerebral symptoms may develop, and the case terminate as tuberculous meningitis, but more often it is the pulmonary symptoms which are dominant. The respirations become more rapid; the cough is frequent, but rarely loose; there may be attacks of cyanosis. Still the only definite signs are the râles, now fine and moist, and diffused generally over the chest. The case usually

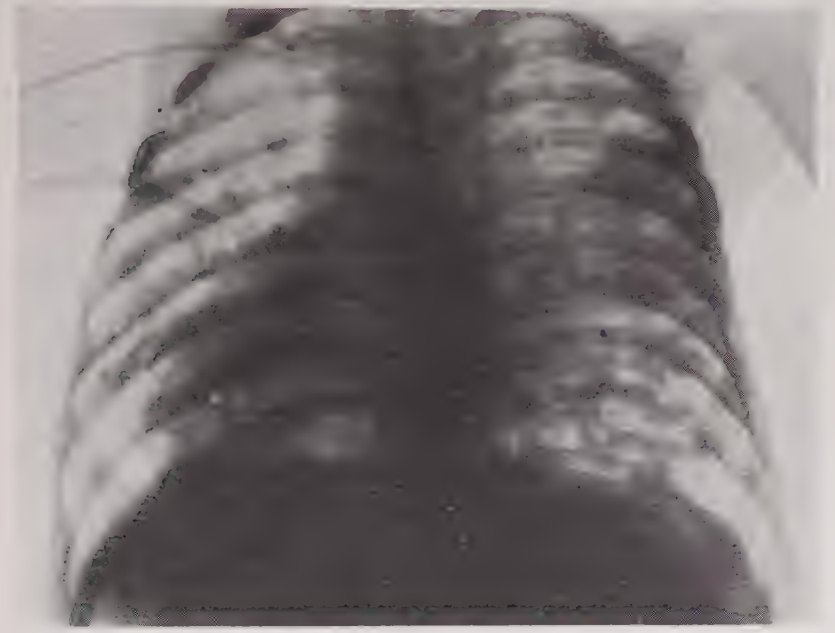


Fig. 176.—Miliary Tuberculosis of the Lungs. Infant fourteen months old; symptoms of marasmus; no elevation of temperature; tuberculides of the skin; positive von Pirquet reaction; no pulmonary signs or symptoms. The radiograph shows great numbers of small tuberculous deposits scattered through both lungs.

ends in death by exhaustion, but without rapid or marked wasting. One of the most striking things in the clinical picture is the disproportion between the severity of the general and pulmonary symptoms and the few physical signs in the chest.

Tuberculous Bronchitis.—This is not an infrequent condition even in infancy. In many, perhaps in most, cases it marks the earliest clinical stage of a tuberculous bronchopneumonia, but this is not always true. The condition seems, therefore, of sufficient importance to require separate consideration. Besides bronchitis, there are found at autopsy a few small tuberculous nodules and tuberculosis of the bronchial glands, although these may give neither signs nor symptoms during life. The

symptoms of this condition are few and not distinctive, and may differ in no respect from bronchitis due to other causes. Tuberculosis may not even be suspected until the lesion has so far developed as to be classed as tuberculous bronchopneumonia. Cough is present, but has nothing characteristic about it except its persistence. Fever may be absent for a long time, but comes as the disease advances. Then it is low and very irregular, the temperature generally varying from 99° to 101.5° F. There may be slow but progressive loss in weight, or the infant may gain regularly for a number of weeks in spite of the cough. This fact often leads to a mistake in diagnosis. The nutrition is influenced much more by the condition of the digestive organs than by the tuberculous process. Other symptoms generally regarded as belonging to early tuberculosis, such as pallor, anemia, perspiration, etc., are usually absent. The physical signs are few and not characteristic. Scattered râles, sometimes coarse and sometimes finer, but inconstant, are all the signs that are present for a long time, often several weeks.

Cases like these are recognized as tuberculous only by finding bacilli in the sputum or by the tuberculin test. It has been our custom to consider as probably tuberculous every infant who has been for any length of time in contact with a tuberculous parent or other member of a household. Regarding all such infants as suspicious has led us in hospital practice to search the sputum carefully for bacilli, with the result that we have found them, sometimes in great numbers, in infants whose only outward symptom was a moderate cough, and who were admitted to the hospital for some other reason. At other times the condition has been unexpectedly discovered by making routine tuberculin skin tests. A typical reaction having been obtained in a child not hitherto suspected, the diagnosis has been subsequently confirmed by finding bacilli in the sputum, although the only signs in the chest were a few râles and the only outward symptom a moderate cough. How many infants there are with such a form of tuberculosis and how long such a condition may continue without more definite signs developing, one can only conjecture; but the number of such cases is, we are convinced, not small. They form a very distinct but important group of tuberculous cases. The regularity with which bacilli are present in the sputum indicates what a factor they may be in spreading the disease. How many recover and in how many the disease goes on to the development of more serious lesions it is impossible to say.

Tuberculous Bronchopneumonia.—This is altogether the most frequent form of tuberculosis seen in young children. It may be primary in the lungs or it may be secondary to tuberculosis elsewhere, most frequently in the bronchial glands. It may be preceded by constitutional symptoms such as those described under the head of general tuberculosis.

It may follow single or repeated attacks of what was apparently a simple acute bronchitis or bronchopneumonia, whether that occurred as a primary disease or was in turn a sequel to one of the infectious diseases, especially measles, whooping-cough, or influenza.

Tuberculous bronchopneumonia, as a rule, begins gradually, and its course is less rapid than simple bronchopneumonia, its progress being generally marked by weeks. When primary it is often preceded by symptoms described as tuberculous bronchitis. When it follows one of the infectious diseases, it is usually engrafted upon the original disease without any intervening symptoms. The early symptoms are cough, rapid respiration, fever, progressive weakness, and anemia. The weight may be at first stationary, but soon there is steady loss, which may continue until there is marked emaciation. At first the usual range of temperature is from 100° to 102° F.; later it is rather higher than this. In many of the cases it differs little from the temperature of simple bronchopneumonia. Sometimes the general symptoms are severe and the physical signs wide-spread, and yet the range of temperature is not high. To be sure, this is occasionally seen in simple bronchopneumonia, but it is more frequent in tuberculosis. The cough early in the disease is slight, but later becomes severe and often distressing. In infants and young children it may be of a paroxysmal character, resembling pertussis. Expectoration is not often seen in those under five years old. Bloody expectoration is very rare in children.

The conditions in the lungs which give physical signs are bronchitis of the smaller tubes with areas of complete or partial consolidation. In character, these signs are identical with those of simple bronchopneumonia. They may be scattered throughout the whole of both lungs; but when localized they are more frequently in the upper than in the lower lobes, and more frequently in front than behind. Although both lungs are involved, they are usually not affected to the same degree. The patient may die before signs of complete consolidation are present; more often there gradually develop areas of consolidation, as shown by bronchial breathing and voice and dulness. In some cases although wide-spread lesions are found at autopsy the physical signs during life are few and indefinite; sometimes there may be almost none. (See Fig. 176.)

From the beginning of acute symptoms the progress of the disease is steadily downward, death occurring as in simple bronchopneumonia. The end is marked by cyanosis, great dyspnea, weak pulse, and extreme prostration. In a few cases there develop cerebral symptoms, indicating tuberculous disease of the brain. Such symptoms may be the first to lead the physician to suspect the process to be a tuberculous one. But even this is not conclusive, for one may be dealing with an acute meningitis due to the pneumococcus. Lumbar puncture will decide.

In the more protracted cases there are found in the lungs caseous nodules, with larger areas of caseous pneumonia, and usually some areas of softening. The process is not usually so generalized as in the cases just described, but as in them there is always associated a certain amount of simple pneumonia. The pathological process may terminate (1) in diffuse caseation, or (2) in localized caseation and excavation, or (3)

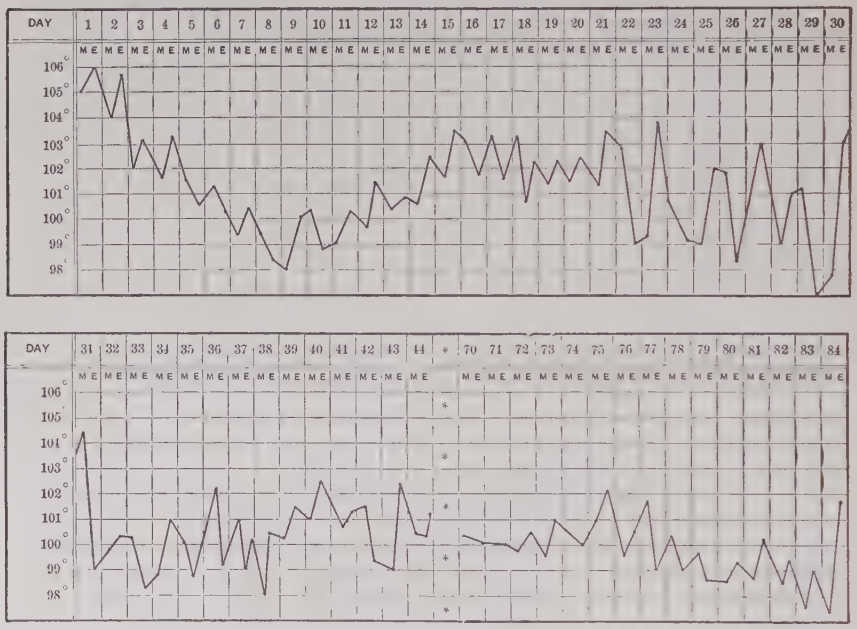


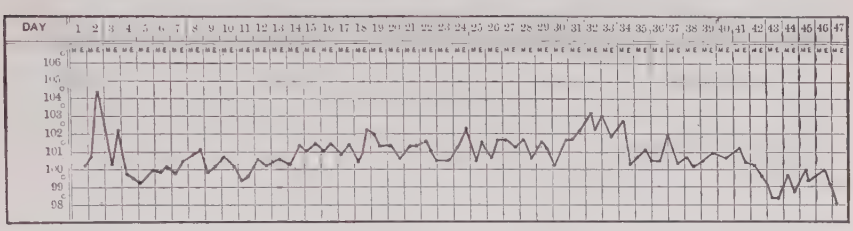
Fig. 177.—Tuberculosis Following Measles. Child sixteen months old, inmate of an institution. Chart begins on fifth day of a severe, but uncomplicated attack of measles, and shows a natural decline to normal. Fever then returned and continued till death, twelve weeks later. Record for the period which is omitted was much like that which immediately precedes and follows. Early symptoms not acute, only slow wasting, slight cough and fever, with scattered râles throughout chest. Signs of consolidation not distinct till eighth week, then present in right upper lobe. Toward the end, rapid emaciation, marked pulmonary symptoms, and signs of cavity at right apex. *Autopsy* showed a large cavity, extensive tuberculous deposits throughout both lungs and in nearly all abdominal organs.

in partial resolution and the development of a chronic fibroid pneumonia. In the first two varieties the progress is as a rule steadily downward to a fatal termination, which takes place in from one to three months. In the third form, which is described later, there is partial recovery.

The mode of onset will depend upon the conditions under which the disease develops. When the general symptoms of tuberculosis have preceded those in the lungs, the evolution of the latter is gradual, with cough, rapid breathing, dyspnea, increased prostration, etc. When the

pulmonary symptoms are present from the beginning, they are the same as in simple bronchopneumonia, with the exception that they usually come on less acutely. The latter is true of cases which are secondary to some other form of tuberculosis in the bones, peritoneum, etc.

When pulmonary tuberculosis follows measles (Fig. 177) or whooping-cough which has been complicated by simple pneumonia, the early symptoms may present no unusual features. After two or three weeks the temperature gradually falls, and the physical signs improve, but neither quite disappears. The cough continues, though its severity somewhat abates. In the course of a few weeks the child, who has meanwhile improved somewhat in his general condition, becomes distinctly worse, often without any assignable cause. The temperature rises to 102° or 103° F.; the cough increases, and an extension of the disease in the


DAY
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37 38 39 40 41 42 43 44 45 46 47
M E M E M E

and in many cases it is never so (Fig. 178). It frequently runs for several days between 99° and 102° F., and then, without evident cause, rises to 104° F. or over. In infants the morning temperature is frequently subnormal, although the evening temperature may be 102° or 103° F. Even toward the close of the disease, when softening and breaking down are actively going on, the regular hectic temperature of adults is rarely seen in a young child (Fig. 179). While the presence of fever is of great significance, its course has almost no diagnostic importance in early life. Especially should one beware of drawing the conclusion that, because the type of fever is not hectic, there is no breaking down of the lung.

Sweating belongs only to the late stage of the disease, and is usually

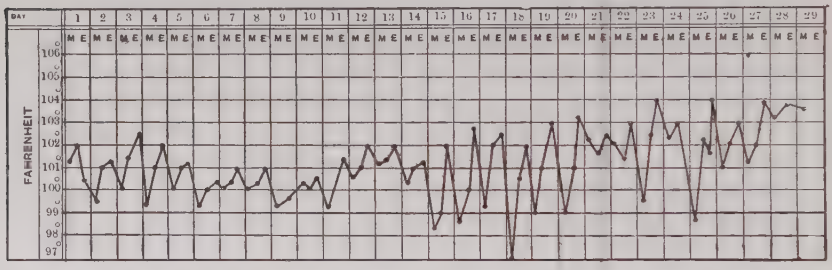


Fig. 179.—Tuberculous Pneumonia with Extensive Softening and Excavation. A delicate child, thirteen months old; weight, 10 pounds; came under observation four weeks before death, with consolidation at apex of right lung. Signs increased in intensity, and extended in area until there were heard, from clavicle to below the nipple, exaggerated bronchial voice and breathing and many moist râles; percussion note was flat; behind, the same signs at extreme apex. No distinct signs of a cavity; no hectic fever; no sweating. *Autopsy* showed large cavity (Fig. 173) at right apex partly filled with caseous masses; diffuse caseous pneumonia (Fig. 174) of the rest of right upper lobe, with scattered deposits in the other lobes, the opposite lung, and a few in the abdominal organs.

associated with the hectic type of fever; both these are regular symptoms in children over seven years old, but not in very young children.

Wasting, like fever, is characteristic of most active tuberculous processes. When fever and wasting are associated, tuberculosis should be suspected, no matter how obscure the other symptoms may be. The wasting is not always rapid, but it is usually continuous. In infants and very young children exceptions to this rule are not infrequent, the progress in weight depending more upon the feeding and condition of the digestive organs than upon the tuberculous process. In the early stage of the disease, wasting is especially suggestive when it continues without apparent cause after measles or pertussis, or when it persists under other circumstances in spite of a good appetite and apparently good digestion. It may at first be so slight as not to be noticed unless the scales are employed. In obscure cases this steady loss of weight is a point of much

diagnostic value, and is frequently overlooked. Toward the close of the disease there is rapid and frequently extreme emaciation.

Cough is almost invariably present; it may be hard, dry, or suppressed; it sometimes occurs in paroxysms resembling pertussis, which may or may not depend upon the presence of enlarged bronchial glands.

Expectoration is absent in infants, the material coughed up being swallowed. In children over seven years old there often is a profuse muco-purulent expectoration, but it is very exceptional below this age.

Hemoptysis is a rare symptom, but not unknown even in young children. Henoch has reported a case of fatal hemoptysis in a child ten months old, where the hemorrhage was due to the rupture of an aneurism in the wall of a cavity. Herz, in 247 clinical cases of tuberculosis in children, records 8 of hemoptysis—4 of them in children under five years, and the youngest only eighteen months old. The records of 131 autopsies on tuberculous children in the Pendlebury Hospital show that hemoptysis was four times a cause of death; two of these patients were under five years, and one was only twelve months old. We have never met with a case of hemoptysis in a child under five years old.

The respiration is accelerated, and usually out of proportion to the rise in temperature. As the lung becomes more and more extensively invaded there is constant dyspnea. The pulse is rapid in the early stage, and continues so throughout the disease; toward the end it becomes weak and irregular.

Pleuritic pains in the chest are not frequent in children. Gastro-intestinal symptoms, such as indigestion, vomiting, diarrhea, etc., are generally present, but are not peculiar to this disease. They usually depend upon the patient's general condition, only exceptionally upon tuberculous disease of the stomach or intestines. The characteristic symptoms of intestinal tuberculosis—abdominal pain, tenderness, uncontrollable diarrhea, and intestinal hemorrhage—are seldom met with in children under five years. Careful palpation of the abdomen may disclose the presence of enlarged mesenteric glands. When these are not readily felt through the abdominal walls, they may sometimes be discovered by a rectal examination.

The spleen is often enlarged, sometimes very much so, but this does not occur with sufficient frequency to be of much diagnostic value. It may be due to tuberculous deposits, to causes connected with the lungs or heart, or to fever. The liver is not often enlarged from tuberculous deposits, but may be so from amyloid or fatty degeneration, or from obstructed circulation, as in the case of the spleen.

Dropsy is rare. It may depend upon anemia, upon complicating nephritis, especially amyloid degeneration, upon cardiac or pulmonary conditions leading to interference with the return circulation, or upon

pressure of tuberculous retroperitoneal or mesenteric glands upon the inferior vena cava. Clubbing of the fingers is occasionally seen in cases running a very protracted course.

Anemia is commonly associated with wasting, and it is of special importance when the latter is slight or absent. It is a frequent sequel of acute disease in infancy when not dependent on tuberculosis; when, however, it is associated with low fever, cough, and persistence of râles in the chest, it should excite apprehension.

Chronic Tuberculous Pneumonia.—In young children this is a chronic interstitial pneumonia associated with tuberculous deposits. These cases have usually had their beginning in one of the acute forms. There is a slow convalescence and apparent recovery, although this is not complete. Often a slight cough remains, or returns from the slightest exposure or other exciting cause. The child does not regain his former weight or vigor, and careful examination of the lungs shows that some abnormal signs remain.

After a few months, possibly, the child has another attack resembling the first. It is accompanied by fever, cough, and perhaps there is a fresh consolidation of some part of the lung, generally in the neighborhood of the old disease. All active symptoms finally subside, and most of the signs of recent disease disappear; but it is then usually found that the condition of the lung is not quite so good as before this second illness. The acute attacks may be repeated several times and pass under the name of bronchitis, bronchopneumonia, or pleurisy. They may extend over a period of years. The general health in the interval is not good, there being present in most cases anemia, with the usual symptoms of malnutrition; these children are regarded as very delicate.

The course of this disease thus differs in no essential particulars from that of simple chronic bronchopneumonia; the physical signs likewise are identical in character, although they may differ in their location. They are generally found in the same conditions as are the signs in the more rapid forms of pulmonary tuberculosis in early childhood. A fatal result in these cases is usually brought about by the development of acute tuberculous pneumonia or miliary tuberculosis of the lungs, by tuberculous meningitis, or by a simple bronchopneumonia.

Physical Signs of Pulmonary Tuberculosis.—Speaking generally, except in situation there is little difference in a young child between the signs of a bronchitis or bronchopneumonia due to the tubercle bacillus, and those of the same lesions when due to other causes. Cavities, although present at autopsy in most of the advanced cases, are seldom of such size or so situated as to be recognized during life. In children over six or seven years old, the signs are essentially like those in adults.

The upper lobes are the seat of the most advanced disease twice as

frequently as the lower lobes, and the right lung rather more frequently than the left. The region most often involved is the middle zone of the lung. If the signs appear first behind they are usually in the interscapular space; if in the lateral part of the chest, they are in the middle or upper part of the axilla; if in front, they are in the mammary region. The explanation is found in the fact that the disease in infants and young children so often extends from the lymph nodes at the root of the lung to the lung itself. The physical signs themselves may be grouped under four heads, corresponding to the pathological conditions existing in the disease, viz., (1) bronchitis; (2) partial consolidation; (3) complete consolidation; (4) excavation. The early signs are almost identical with those described in bronchopneumonia. As a rule, however, the transition of the signs from one stage to another is much slower in tuberculous than in simple bronchopneumonia.

Tuberculous bronchitis gives râles which may be of all sizes and varieties, localized or general. If the process goes on to a partial consolidation there are gradually developed in addition slightly impaired resonance or even dulness, bronchovesicular respiration, and increased voice. These signs are usually over a localized area. Later the signs of complete consolidation are present—marked dulness, increased fremitus, bronchial respiration, and voice,—but still râles and friction sounds are generally heard.

The later signs depend upon what course the pathological process follows. If it terminates in a diffuse or localized caseation, the signs differ little from those of a lobar pneumonia with extensive and complete consolidation except that the dulness on percussion is usually greater. There may be even flatness so marked as to suggest the presence of a pleural effusion. Empyema is the diagnosis often made. These signs may persist until the death of the patient from exhaustion.

If the caseation is localized and followed by excavation, the signs of a cavity may be present. Cavities, however, are often so small and deeply seated as not to give definite physical signs. Furthermore, they are frequently filled with thick pus or cheesy matter, and rarely communicate freely with the bronchi. If large and superficial they give the same signs as in adults. Like the areas of tuberculous pneumonia, they are most frequent in the middle zone of the lung in front or laterally. In the young child similar signs are often present when there are only dilated bronchi associated with a fibroid condition, or when a superficial bronchus is surrounded by an area of diffuse caseation. Cavities are very often diagnosticated when they do not exist, and quite as often overlooked when present.

If the acute process terminates in a chronic tuberculous pneumonia the signs are those of an unresolved or slowly resolving pneumonia, in

which the area of consolidation gradually diminishes, but the signs do not altogether disappear. When recovery goes further there may remain only some dulness on percussion, bronchovesicular respiration, râles, and friction sounds. Such signs may last indefinitely, exacerbations and remissions occurring from time to time. These signs can not be distinguished from those of simple chronic bronchopneumonia.

Diagnosis of Pulmonary Tuberculosis.—The family history, surroundings and previous condition of the patient are important, also the mode of onset, the course of the disease, and the evidence afforded by the physical examination. Not only should the health of the parents and other children be investigated, but that of other members of the household and frequent visitors. The occurrence of bone and joint disease as well as pulmonary disease should be considered.. Surroundings favoring the development of tuberculosis are city residence, especially if in a tenement house, or an institution. One should regard as important, habitual underweight, anemia and general malnutrition. Of previous diseases in the patient the most significant are pneumonia, measles or pertussis with prolonged convalescence, and persistent or frequently recurring attacks of bronchitis. In the milder or early cases the two important symptoms are cough and fever; the cough is more significant when persistent and accompanied by mucopurulent expectoration. Hemoptysis among children is so rare as to aid little in diagnosis. Fever, to be of diagnostic value, should be at least 99.5° F. in the mouth or 100.5° F. rectal for a considerable period, usually several weeks. Wasting is important when present but its absence by no means excludes tuberculosis, since it depends more upon the condition of the digestive organs and the feeding than upon the local disease. Sweating is not a common symptom in children. The physical signs which are of especial diagnostic value are persistent localized râles anteriorly, either in the region of the nipples, or between the nipples and axillae, or at the apices. To these signs should be added a positive von Pirquet reaction, which test should be repeated at least once to avoid error. Additional information is sometimes afforded by the X-ray examination, though the plates need to be interpreted by one with much experience. These findings are always to be taken in conjunction with physical signs and rational symptoms.

In the more acute or more advanced cases, tuberculosis has to be distinguished chiefly from simple bronchopneumonia. The onset of simple pneumonia is usually rapid, often abrupt; tuberculous pneumonia usually develops more gradually. Constitutional symptoms may precede the local ones by several days or even weeks. In tuberculosis one is often struck by the disproportion between the general symptoms and the physical signs. One may see with tuberculosis, rapid wasting, prostration, cough and high fever with physical signs which are few, irregular and

inconstant. Again strongly suggestive of tuberculosis are very extensive physical signs, especially persistent generalized fine râles without consolidation, accompanied by severe dyspnea, even cyanosis, and yet with a temperature only moderately elevated.

The course of the temperature can not be depended upon in diagnosis. A high leucocyte count, e. g., above 25,000, especially when accompanied by a high polymorphonuclear percentage, strongly favors pneumonia. The X-ray examination is often of more value in these than in the less acute cases. The cutaneous tuberculin test gives positive results in nearly all cases except those which are extremely prostrated. Meningitis developing during a pulmonary disease of doubtful character is generally tuberculous. But acute pneumococcus meningitis may occur in very similar circumstances.

Examination for Bacilli.—Discovery of the bacilli in the sputum is of course conclusive and is by no means so difficult, even with very small patients, as has been supposed; but in most cases repeated examinations are necessary. Infants do not expectorate, but cough up the bronchial secretion into the pharynx and swallow it. Sputum must therefore usually be obtained from the pharynx. To obtain the sputum in an infant one should excite a cough by irritating the pharynx, and then catch upon a small swab the sputum brought up into view. By the procedure mentioned it is not usually more difficult to obtain good sputum in very young patients than in adults. Bacilli are seldom found in clear, glairy mucus, but in mucopurulent masses. Following the method described, bacilli have been found in the great majority of our hospital cases of pulmonary tuberculosis in infants, although in more than half of them the disease was not advanced, judging by symptoms and physical signs.

Bacilli may readily be found in the stools of many children suffering from tuberculosis. Their presence does not necessarily indicate a tuberculous lesion of the intestines, for their source is more frequently a pulmonary lesion, the bacilli being coughed up and swallowed. Hence, it is sometimes easier to find them in the stools than in the sputum. They must be carefully differentiated from the smegma bacilli.

III. Chronic Phthisis.—This form of tuberculosis, with its chronic hectic fever, slow cavity-formation, progressive emaciation, night sweats, etc., is very rarely seen before the fifth year, and it is not at all frequent until the tenth or twelfth year. In its symptoms, course, termination, and physical signs, it resembles the same disease in adults, and need not be described at length here.

IV. Tuberculosis of the Bronchial Lymph Nodes (Bronchial Glands).—This condition is usually associated with some form of pulmonary tuberculosis, but it may exist as altogether the most important tuberculous lesion.

The symptoms are usually associated with those of pulmonary or general tuberculosis; but they may occur when the pulmonary changes are too few to be recognized either by symptoms or physical signs. From the great frequency with which this lesion is found in infants and young children, it might be expected that local symptoms would be common in such patients. They are, however, in our experience, quite exceptional. Most of the cases in which well-marked symptoms occur are in children over two years old, and it is between the third and tenth years that they are usually seen. In infancy, death in most cases occurs from the pulmonary disease.

General symptoms may or may not precede the local ones. The latter are chiefly mechanical, and depend upon the size of the glands and upon their anatomical relations, and very little or not at all upon the nature of the changes in them. The most important relations, so far as the production of symptoms is concerned, are those which they bear to the pneumogastric and recurrent laryngeal nerves, the superior vena cava, the trachea, and bronchi; those less important are to the aorta, pulmonary artery, and esophagus.

Pressure upon or irritation of the pneumogastric or recurrent nerves produces cough, dyspnea, and sometimes a change in the voice. The cough is hoarse, persistent, and teasing, and frequently occurs in paroxysms which in many respects resemble those of pertussis, but it usually lacks the characteristic whoop, and is not accompanied by the expectoration of a mass of tenacious mucus. These paroxysms are severe and often prolonged. The dyspnea, like the cough, is paroxysmal, and sometimes strongly resembles ordinary spasmodic croup; at other times it is like a severe attack of asthma. Such symptoms may come and go, but they are frequently prolonged, and usually in the interval between the severe seizures the patient is not wholly free from dyspnea. Although the chief cause of dyspnea is no doubt nerve irritation, it may be due in part to pressure upon the trachea or one of the large bronchi. In dyspnea from pressure on the trachea the head is usually thrown back, and the obstruction is more frequently on expiration than on inspiration.

After such symptoms as those mentioned have existed for a few days or weeks, and in some cases without any warning, there may occur a sudden attack of asphyxia which may prove fatal. This is generally due to ulceration of a caseous gland into the trachea or a large bronchus and the escape of a large mass into the air passages, where it produces the same effects as does any other foreign body.

Of fifteen cases of this kind collected by Loeb, death by suffocation occurred in most in from five to ten minutes after the first definite symptoms; in some the fatal attack was preceded for some time by milder attacks or by a cough; in others no previous symptoms were present,

the child being apparently in perfect health. Rarely after ulceration into the trachea the patient has recovered after coughing up a large amount of pus.

Pressure upon the superior vena cava is usually associated with spasmodic dyspnea and cough, and causes cyanosis of the face and blueness of the lips. There is frequently a puffiness of the face, and there may be marked edema. The coexistence of cyanosis with such edema, when the urine is free from signs of renal disease, should always lead one to suspect pressure at the root of the lung. In some rare cases the interference with the return circulation has been so marked that meningeal hemorrhage has resulted. By a process of ulceration set up in these glands they may open, not only into the air passages, but into the pericardium, the esophagus, or any of the large vessels. The last mentioned is usually followed by instant death. Aldibert reports two cases in which the pulmonary artery was opened, death occurring from hemoptysis, as there was also a communication with one of the large bronchi. In Vogel's case the subclavian vein was perforated, and death resulted from the entrance of air. If ulceration takes place into the surrounding connective tissue, a mediastinal abscess may result, producing any of the pressure symptoms noted above, and, in addition, dysphagia from pressure on the esophagus. Such an abscess may point in the suprasternal notch; it may open through the chest anteriorly between the ribs or at the xiphoid cartilage; or it may burrow along the esophagus to the peritoneal cavity. As a rule, however, patients die of general tuberculosis before the local conditions have advanced so far.

Physical Signs.—In order to produce signs the mass of lymph nodes must be large enough to form a considerable mediastinal tumor, or be so situated as to produce pressure upon the trachea or bronchi. Only large packets of glands can be made out by physical signs. The large masses may give dulness over the first piece of the sternum, or, more frequently, behind in the interscapular space, usually between the third and seventh dorsal vertebrae. Normally, whispered bronchophony usually ceases at or just below the level of the fourth dorsal vertebra. When it extends below this point it is suggestive of enlarged bronchial glands (D'Espine's sign). It is usually more marked upon the right than the left side. There may be also voice and breathing of a somewhat amphoric but with a distinctly nasal quality. The signs are sometimes indistinguishable from those heard over a small cavity. Taken in connection with a positive cutaneous tuberculin test and X-ray findings, these signs are of much significance. If one of the primary bronchi or one of its lobar divisions is compressed, there may be very feeble respiration over one lung or one lobe; if the pressure is sufficient to prevent the entrance of air, or if one of these large tubes has been plugged by a caseous mass, there is an absence of

respiratory murmur over a single lobe or an entire lung. This sign is of great diagnostic value, but it is not often present.

Diagnosis.—Mediastinal glandular tumors may occur in Hodgkin's disease and in malignant disease; but both are relatively very rare and usually present other diagnostic symptoms. Practically, in almost every case, marked enlargement of the bronchial glands is due to tuberculosis. The only really trustworthy means of diagnosis in most cases is afforded by the X-ray, though considerable experience is requisite in the interpretation of the plates; the radiographic shadow usually shows better

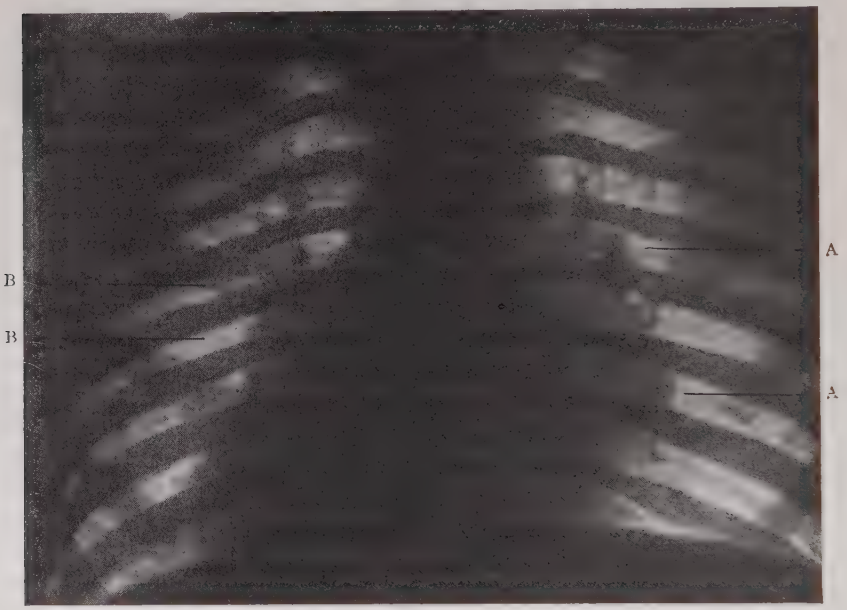


Fig. 180.—Tuberculous Bronchial Glands. A very large mass upon the right side, A, A; a smaller one upon the left side, B, B.

on the right side than on the left on account of the heart (Fig. 180). Especially significant are evidences of calcification, which may be found even in very young children. (We have autopsy records of such changes in infants only seven months old.) More stress is in some cases to be laid upon symptoms than physical signs for diagnosis: the most important symptoms are the association of a spasmodic cough with paroxysms of dyspnea resembling asthma or croup and severe congestion or edema of the face. The chief difficulty in diagnosis is found in those cases which present few or no other signs of tuberculosis, and which come first under observation with attacks of dyspnea or asphyxia resembling those seen in laryngeal stenosis. In many such cases tracheotomy has been done without finding any cause for the dyspnea, the autopsy

showing it to be due to the ulceration and impaction of a caseous gland. The development in a child of a chronic abscess in the anterior mediastinum is almost always due to tuberculous glands; and so is one in the posterior mediastinum, provided Pott's disease can be excluded.

The Tuberculin Tests.—For general diagnostic use in children von Pirquet's cutaneous test is so far superior to the other tests suggested that only this need be considered in detail.

The forearm is the most convenient part for applying the test. The skin is carefully washed with alcohol or ether. A small drop of pure tuberculin (Koch's O.T.) is placed upon the skin. With an instrument resembling a tiny chisel a very slight scarification for control is made at a distance of two or three inches from this drop. A similar scarification is then made through the drop. Linear scratches one-quarter inch in length lightly made with a sterile needle, serve equally well as a means of inoculation and control. The child should be watched, and if very young the arm should be held until the skin is quite dry to prevent infection by rubbing. As an added precaution it may be covered with a piece of sterile gauze. The reaction consists in a red areola about the point or along the scratch made. This generally begins in from twelve to eighteen hours, rarely later than twenty-four hours, and reaches its height during the next twenty-four hours. The size of the areola indicates the degree of reaction. It continues in most cases for from one to three days and slowly fades, often being followed by a slight local desquamation. Rarely there may be vesiculation. There is in most of the cases slight induration of the skin readily appreciable to the touch. The more marked reactions continue for from four to ten days. Any definite inflammatory reaction which follows this course may be regarded as positive. The arm should be observed daily to note the results. There seems to be no relation between the intensity of the reaction and the extent or the activity of the tuberculous disease.

The Significance of the Tuberculin Test.—The cutaneous test gives positive evidence if tuberculosis is present, in all except the most prostrated cases and those in the late stages of the disease, when diagnosis is rarely difficult from the other symptoms. Exceptions are, in our experience, extremely rare. Much importance is therefore to be attached to a negative reaction. For greater certainty the test should be repeated in suspicious cases. The interpretation of a positive reaction is much modified by the age of the patient. Under one year a positive reaction usually indicates an active tuberculous process. Many have even taken the ground that an infant under one year with a positive reaction is doomed. We do not think the outlook quite so hopeless; but such a reaction is certainly of grave import. During the second year a positive reaction is not so serious; it is often seen in infants who have not at the

time and do not develop active tuberculosis. After infancy the test becomes less and less an indication of an active tuberculosis and the interpretation of a positive reaction is more difficult. It is always to be taken in conjunction with the clinical symptoms. A negative reaction with clinical symptoms suggestive of tuberculosis is always to be regarded as significant. It almost certainly excludes tuberculosis except in conditions of extreme prostration. Great difficulty may exist in the interpretation of a positive reaction under two conditions. The first is in an apparently healthy child with a prolonged unexplained temperature but no physical signs of pulmonary disease. In such circumstances the existence of active tuberculosis is probable after other conditions have been excluded. The X-ray may shed light upon the case. The second condition includes the cases in which acute pulmonary disease is present in a patient who gives a positive reaction. The course and termination of the disease may ultimately establish the fact that the process in the lung was non-tuberculous. But because of the positive reaction grave suspicion of tuberculosis may exist. Much needless alarm may therefore be excited by a positive reaction, which really demonstrates only that the child has somewhere a tuberculous focus, but does not prove the existing disease to be a tuberculous process. The tuberculin reaction is always to be interpreted in conjunction with the general symptoms and the physical signs. As a rule, in older children a negative reaction is of more significance than a positive one. During active measles the test can not always be relied upon.

The tuberculin test should not be allowed to displace the examination for bacilli either in the sputum or cerebrospinal fluid, though the latter involves much more labor. The positive reaction furnishes reliable evidence of the existence of a tuberculous process, but as to whether this is active or latent gives no information.

Tuberculides of the Skin.—These are at times of considerable value in the diagnosis of general tuberculosis. Although seldom seen in the most acute varieties, they are not uncommon in the more slowly progressing forms. The distribution of the lesions is fairly constant. They are found chiefly on the buttocks, lower abdomen, genitalia and thighs. The number present is generally small, half a dozen to a dozen; but they are sometimes numerous and may be widely distributed. The lesion somewhat resembles that of varicella. It begins as a minute red papule, which is soon surmounted by a small vesicle. This dries to form a crust. If the crust is removed a small pit-like depression remains which heals quickly, leaving a white, glistening scar surrounded by a pigmented border. The lesion runs its entire course in two or three weeks. Tubercle bacilli are often present in the lesions but are difficult to demonstrate. Tuberculides of the skin in young children are evidence

of a widely disseminated process and are a very bad prognostic sign. Such patients rarely survive more than a few weeks.

General Prognosis of Tuberculosis.—The outlook for a child under two years with general or pulmonary tuberculosis is very bad. So long as the disease remains confined to the lymph nodes, the child is not usually in danger, except from accidents connected with their softening and ulceration, which after all are rare. Spontaneous cure may occur in these glands in the same way as in others in the body, viz., by encapsulation, calcification, etc. Such a result is no doubt a very frequent one; exactly how often it occurs it is impossible to say; but when once the disease has gained any headway in the lung itself, its steady advance is almost certain to be the course in a young child. In those who are older and have more resistance the chances of an arrest of the process are much greater.

If the bacilli have gained entrance into the body in any considerable numbers, even though they are shut up in an encapsulated, caseous, bronchial gland, the patient is never free from the danger of general infection.

Prophylaxis.—The prevention of tuberculosis must have constant reference to its cause. The first essential is the destruction of the tubercle bacilli wherever they exist. Since most of those existing in the air are derived from the sputum of patients affected with pulmonary tuberculosis, it should be insisted upon, everywhere and at all times, that the sputum from such cases should be collected in special cups or cloths and destroyed either by germicides or by fire. The next point is to avoid needless exposure. A tuberculous mother should on no account nurse her child nor kiss it upon the mouth. A wet-nurse likewise should be free from any tuberculous taint. No nurse or other care-taker should ever be employed about children who has, or ever has had, pulmonary tuberculosis. It is wise to exclude also those who suffered when children from tuberculosis of the bones or the cervical glands, although the danger from such persons is extremely slight. If active tuberculosis exists in any member of the family, a young child should be kept away from the room, and if possible should not reside in the house. On no account should infected persons be allowed to kiss children or sleep in the same bed with them. The danger from drinking-cups and other dishes should not be forgotten. A tuberculous person should either have his special dishes, or the utmost care should be taken to boil all those which he has used. Cows whose milk is used for children should be under regular veterinary inspection and should have passed the tuberculin test. In any case when the slightest doubt regarding the health of the cows exists, or when the source of the milk is unknown, the milk should be pasteurized. The danger of infection through the alimentary canal is very much less

than through the respiratory tract, and consequently the precautions first mentioned are much more important than those relating to the food, although the latter should on no account be neglected.

In the case of delicate children and those with tuberculous parents or with other tuberculous near relatives, everything possible should be done to fortify them against the disease. They should be kept under more or less constant medical supervision. Attacks of bronchitis or bronchopneumonia should be watched with the greatest solicitude. Exposure to influenza, measles or pertussis should especially be avoided. The country rather than the city should be chosen for residence, and the child should, if possible, spend the winter and spring in some warm, dry climate. Parents should be distinctly taught that watchfulness and care do not mean coddling or the keeping of children in the house the greater part of the time. Such children should live as much as possible in the open air, and every form of sport encouraged which tends to keep them there. Overheated houses are one of the most prolific agencies in perpetuating a delicate condition of health. Plenty of fresh air in sleeping apartments should always be insisted upon. All catarrhal troubles of the nose and pharynx should receive early and prompt attention, especially should hypertrophied tonsils and adenoid growths of the pharynx be removed, since these are conditions which form a most favorable nidus for the growth of tubercle bacilli.

Treatment of General and Pulmonary Tuberculosis.—If fresh air and a proper climate are necessary for the cure of this disease in adults, they are tenfold more necessary in the case of children. Without them there is little hope for a child with active pulmonary tuberculosis. Nowhere do these cases do so badly as in a hospital located in a city, and no class of hospital cases do worse than these. The same regions that are beneficial for adult cases usually agree with children, with the exception that the latter, as a rule, do better in a warm than in a cold climate. Plenty of fresh air and sunshine are essential. A child must be where he can be kept in the open air for the greater part of each day, in spite of fever, cough, or other acute symptoms.

For the most acute cases when the children are confined to the bed, the largest, best-ventilated, and sunniest room available should be secured, and the windows should be constantly open. The general management of such cases is the same as for those with acute pneumonia.

There is no specific remedy for tuberculosis. The diet is a matter of the utmost importance. Tuberculous patients must be fed like most other sick children, care being taken not to disturb the digestion by the unnecessary use of drugs. For a staple article of diet, milk is the best, and when this is not well borne some of its substitutes—buttermilk, kumyss, zoolak, etc.—may be tried. Cream is almost as useful as cod-

liver oil, and should be given in one form or another whenever the child's digestion can tolerate it.

Tuberculin in the treatment of this disease in young children has been most disappointing in its results. Its value has not yet been demonstrated. There always exists the possibility of lighting up a latent process in the lungs. It should be given with the greatest caution in active febrile cases. The method of using it is discussed under the treatment of Tuberculous Adenitis.

Cod-liver oil is usually best given in a fresh emulsion, although some children bear the pure oil better than its preparations. Inunctions of this or other oils are of some value when not well tolerated by the stomach. Arsenic and iron are useful as general tonics.

CHAPTER XI

SYPHILIS

Syphilis is a communicable disease due to a specific organism, the *spirocheta pallida* of Schaudinn. In acquired syphilis this is found in the primary lesion, in the mucous patches and in the lymph nodes. In hereditary syphilis it is found in the cutaneous lesions, in the fissures at the angle of the mouth and in the mucous patches of the buccal cavity, with less regularity in the internal organs, except the liver, which usually harbors the organism in immense numbers. While in the still-born child and in early cases, the number of organisms found is very great, they are not so numerous at a later period, and they diminish rapidly after treatment is begun. In the late lesions the spirochetae are not numerous, and are difficult to demonstrate.

In infancy and childhood both the acquired and the hereditary forms of syphilis are seen.

ACQUIRED SYPHILIS

While acquired syphilis is very much less frequent than the hereditary variety, it is by no means a very rare disease in early life. It is not improbable that some of the manifestations of syphilis in later childhood which are usually denominated "late hereditary syphilis," are really due to the acquired form.

Etiology.—An infant may be infected by the mother during parturition; but this is extremely rare and can take place only when there are lesions upon the mother's genitals. Infection is more likely to be from

a mother who contracts syphilis subsequent to the birth of the child, and may occur through nursing or accidental contact by kissing, etc. In either of these ways, or from a venereal sore upon the nipple, a child may be infected by a wet-nurse. Whether syphilis can be communicated through the milk when the nipple is perfectly healthy and free from fissures, is exceedingly doubtful.

Syphilis may be communicated directly from a syphilitic child to one who is healthy, by kissing, by sexual contact, or indirectly by means of bottles, spoons, cups, clothing, etc. The latter mode of infection is most likely to occur in institutions. Vaccination was formerly a not infrequent mode of communicating syphilis, but has been practically eliminated by the general introduction of bovine virus. Cases have been recorded where the disease has been conveyed by the rite of circumcision, either from the mouth or the instruments of the operator.

The relative frequency of the different sources of infection is shown by Fournier's statistics of 40 cases: The source of infection was the parents in 19; nurses, in 8; servants, in 4; sexual contact, in 4; vaccination, in 2; other children, in 2; a physician, in 1. The ages at which the disease was acquired in this series of cases were as follows: During the first year, 19; during the second year, 10; during the third and fourth years, 7; from the fifth to the fourteenth year, 6.

Symptoms.—The symptoms of acquired syphilis in children are in all respects similar to the same disease in the adult. A primary sore is present at the site of infection, which is most frequently the lips, the mouth, or some part of the face; very rarely is it seen on the genitals. There are few individual symptoms belonging to hereditary syphilis which may not also be present when the disease is acquired. Its course, however, is very much milder in the latter and a fatal termination is rare. Fournier states that of his forty-two cases only one died of marasmus. This marked contrast to hereditary syphilis is due chiefly to the fact that in the acquired variety the infant is rarely infected during the early months of life, a time when hereditary syphilis is so fatal.

Tertiary symptoms may appear at any time from three to twenty years after the original infection.

The treatment is the same as that of hereditary syphilis.

HEREDITARY SYPHILIS

Etiology.—If both parents are syphilitic, the child is usually but not invariably so. The symptoms, however, are not more severe than when the inheritance is from one parent only. The likelihood of transmission depends upon the stage of the disease in the parents. If the mother is suffering from secondary symptoms, transmission is almost certain.

If active treatment has been employed for several months, if the child is born at a period when no active symptoms are present, or if the symptoms are of a tertiary character, the offspring will probably escape. First-born children are more likely to suffer severely from syphilis than the later ones, provided infection of the parents has taken place prior to the birth of all the children.

The transmission of syphilis from the father without the intermediate infection of the mother was once held to be not only possible but frequent. At the present time, however, this question must be placed among those not yet definitely settled. There can be no doubt that in the vast majority of the cases the infection of the child is from the mother.

If both parents are healthy at the time of conception and the mother becomes infected during her pregnancy the child may or may not be syphilitic. Transmission to the child is much less likely to occur if the mother is infected during the last two months of her pregnancy than earlier, although, as Hutchinson's cases conclusively show, there is no certainty that the child will escape. Diday states that if the mother is infected before the fourth week and proper treatment is instituted, the child will usually escape on account of the relation of the embryo to the maternal circulation during this early period.

In 1837 Colles enunciated the following proposition, the truth of which has been abundantly verified since his time: "A new-born child affected with inherited syphilis, even although it may have symptoms in the mouth, never causes ulceration of the breasts which it sucks if it be the mother who suckles it, although continuing capable of infecting a strange nurse." From the careful analysis of many cases and with the great assistance derived from the Wassermann reaction the conclusion seems irresistible that the mother who bears a syphilitic child is immune to syphilis for the reason that she herself is suffering from syphilis, or a modification of that disease. The mother in these circumstances can not be inoculated either by her syphilitic nursing infant or artificially.

That hereditary syphilis is contagious is conclusively shown by a number of recorded instances in which a healthy wet-nurse has been infected by a syphilitic infant. We have ourselves seen one such instance. However, such examples of contagion are rare, and many writers of large experience state that they have never seen it. It is certainly true that the danger of spreading infection from a case of hereditary syphilis has been exaggerated.

Lesions.—Death may occur with syphilis, and yet the autopsy may reveal no characteristic anatomical changes, and in fact there may be no demonstrable changes in any of the organs except the presence of the spirochetae. This is particularly true of infants dying in the first weeks of life.

Bones.—In the case of a syphilitic fetus, a still-born child, or one dying soon after birth, the changes in the bones are more uniformly present than are any other lesions. They are, in fact, rarely wanting, and it is by them alone that syphilis is often recognized post mortem, but it may require a microscopical examination to establish the diagnosis. The long bones are principally affected, the most important changes being found at the junction of the shaft with the epiphyseal cartilage. The lesion is termed an epiphyseal osteochondritis. There are two varieties: in one there is an inhibition of bone formation around the columns of calcified cartilage, though the destruction of cartilage cells by the vessel loops and the formation of bone marrow goes on unchecked and in a normal manner. In the other, there is, in addition to the delay in bone formation, the development of granulation tissue that springs from the cartilage canals and that grows between the shaft and the epiphysis, and, perforating the column of cartilage cells, invades the epiphysis. The granulation tissue may grow so luxuriantly as to separate the epiphysis from the shaft and in either case the bone is so weakened at the epiphyseal line that fracture through it readily takes place as the result of slight traumatism, either in intra-uterine life or after birth. Thus results separation of the epiphysis, a frequent manifestation of severe hereditary syphilis. With either form of osteochondritis there is a broad yellow line to be made out macroscopically at the junction of the epiphysis and shaft; with the excessive formation of granulation tissue and the invasion of the epiphysis the line is an irregular one.

While the osseous changes are widely distributed throughout the body they are not of equal intensity. The lower end of the femur and radius and the upper end of the tibia and humerus are most severely affected. Complete recovery from the lesion is possible. Acute suppurative epiphysitis and arthritis may occur in syphilis but they are to be regarded as of pyemic rather than of syphilitic origin.

Osteoperiostitis is common in hereditary syphilis. In young infants it is found as a very generalized lesion, affecting the shafts of the long bones, especially those of the leg, forearm, and hands. The swelling is usually at the end of the shaft. With increasing age the tendency is to involve the shaft nearer its middle. The lesion in infants is largely periosteal. Later the bone participates more and more in the process; there is a formation of new bone which is firm and very compact or it may consist of a coarsely spongy structure. The periosteal swellings with appropriate treatment may entirely disappear by absorption. The new-formed bone largely persists.

Gummata of the bones are rare in infancy. With older children gummata may form on the long bones or the skull. They are not essentially different from those occurring with acquired syphilis.

Liver.—This is probably more frequently involved in the fetus and newly-born infant than any other organ. The syphilitic lesions of the liver consist in an interstitial hepatitis, a gummatous hepatitis, or a combination of the two varieties. In the interstitial form, which is most frequent in infancy, the liver is enlarged, frequently very much so, and firm. On cross section the markings are indistinct. Microscopically, there is a great increase in connective tissue which is diffusely scattered throughout the whole organ and even between the individual liver cells. There may be also bands of connective tissue invading the liver in different directions. As the connective tissue contracts an irregularity of the surface of the liver develops. Groups of miliary syphilomata may also be found.

The gummatous form is not frequent in early infancy, but belongs to a little later period. In this there may be miliary syphilomata with interstitial changes, and in addition the formation of small or large gummatous tumors which may be softened at the center. They are surrounded by zones of new connective tissue and the liver cells are atrophied. Amyloid changes may be present.

In the late form of hereditary syphilis, usually seen in children over four or five years old, the liver is occasionally affected. The lesions resemble those of the congenital variety. There are found cirrhotic changes, which may be diffuse or circumscribed, and gummatous deposits, which vary from a minute size to that of a cherry; there may be amyloid degeneration.

Spleen.—This is almost invariably much enlarged in newly-born children with syphilis and in syphilitic fetuses, but nothing characteristic is found under the microscope. In older children the enlargement of the spleen may be even greater. The organ may be the seat of interstitial changes, and sometimes there may be small gummatous deposits. These changes are rare in children under two years of age.

Respiratory System.—In syphilitic infants who are still-born and in those who die soon after birth, there is occasionally found in the lungs what is known as "white pneumonia." The lungs are nearly white or slightly red. They are firm and contain little or no air. The alveoli are filled with desquamated cells and leucocytes. There is an increase in the connective tissue of the alveolar walls, bronchi, and blood vessels. There may also be gummata scattered through the lungs. These are usually small.

The trachea and bronchi are in rare cases the seat of stenosis, which results from cicatrization following the softening of gummatous deposits in their walls. Lesions of the larynx other than a chronic catarrhal inflammation of the mucous membrane, are also infrequent. The lesion usually found is perichondritis, which more often involves the epiglottis

than any other part. Sometimes there is the formation of papillomatous masses; but ulceration and stenosis are both rare.

The nasal mucous membrane in the early stage of the disease is very constantly the seat of a chronic catarrhal inflammation, which may be accompanied by superficial ulceration. In the late cases there is deeper ulceration, from the breaking down of gummata, with extension to the periosteum, cartilages, and bones, causing perforation of the septum, necrosis of the bones, etc.

Nervous System.—Syphilis may affect the meninges, the blood vessels or the brain itself. There may be merely a diffuse thickening of the meninges with which there is usually associated a certain amount of encephalitis, or there may be miliary gummata scattered throughout the meninges but especially at the base. As the result of the chronic syphilitic meningitis, adhesions may form at the base, obliterating the foramen of Magendie and at times leading to hydrocephalus. Syphilitic endarteritis is very common and consists in a thickening of the vessel wall with proliferation of the intima and reduction in the caliber of the vessel. There is also a perivascular proliferation of connective tissue. The changes that have been described are found in direct proportion to the severity of the syphilitic infection. In infants dying *in utero* or shortly after birth they are frequent. In those with a mild infection, the lesions may be slight or absent. Large gummata are unusual at any time.

Later in childhood, syphilis of the brain is not very uncommon. The lesions are chiefly the result of the vascular changes and consist in localized or diffuse sclerosis with greater or less atrophy of the convolutions. The lesions of juvenile paresis and tabes do not differ from those that are the result of acquired syphilis.

Heart and Arteries.—These are very frequently affected, even in young infants. Adler, of four cases examined, found two in which well-marked lesions were present in infants under four months. Warthin has emphasized the importance of systematic study of the heart for evidences of syphilis. He has found lesions and has demonstrated the organism when no other evidences of syphilis were to be found in the body. The lesions consist of a diffuse or localized interstitial myocarditis with endarteritis of the coronary arteries and small blood vessels.

Digestive System.—Chronic catarrhal pharyngitis is almost a constant symptom of the early cases. Later there is seen superficial or deep ulceration of the pharynx, tonsils, or fauces, which may lead to perforation of the soft or hard palate.

There are no frequent lesions of the stomach or intestines either with early or late syphilis. In infants dying early with very extensive lesions ulcerations are sometimes found in the small intestine. They are

multiple and extend transversely across the intestine. They cause no symptoms. The rectum is occasionally the seat of ulceration, and condylomata may form about the anus even in young children.

Pancreas.—Changes in the pancreas are frequent with severe infections; with mild infections they are usually absent. They consist in a diffuse production of connective tissue which replaces, to a greater or less extent, the parenchyma of the organ. In the most extreme cases there may be no glandular tissue remaining. The islands of Langerhans are usually not destroyed.

Thymus.—Occasionally there are found in syphilis numerous small abscesses in the substance of the thymus gland. They are filled with a purulent material consisting of leucocytes with great numbers of spirochetes. The glandular tissue is also infiltrated with leucocytes. These abscesses of DuBois are very characteristic of syphilis.

Organs of Special Sense.—Otitis is a frequent accompaniment of the early syphilitic pharyngitis. It is very likely to become chronic, and in many cases results in a permanent impairment of hearing. Iritis is relatively rare in children, but it may occur even in intra-uterine life, as shown by the presence of adhesions in newly-born children. It is usually seen in infants four or five months old, and is always serious. Interstitial keratitis occurs frequently as a later manifestation of syphilis. Choroiditis and optic neuritis are both occasionally seen, but they are rare.

Genito-urinary Organs.—Nearly all these may be affected, but generally in the late period of the disease. There may be chronic interstitial nephritis and more rarely gummatous deposits in the kidney, interstitial changes in the suprarenal bodies, and orchitis, which usually affects the body of the organ, rarely the epididymis; it is generally an interstitial inflammation, with or without gummatous deposits.

Symptoms.—As the result of syphilis, abortion may take place at any period of pregnancy, with the discharge of a dead or macerated fetus, or the child may be still-born at term, or it may be born alive prematurely, but with so feeble a vitality that it survives but a few hours. In these circumstances it is often difficult and sometimes impossible to decide positively with reference to the existence of syphilis. Maceration of the fetus or peeling of the skin is no proof, and even the examination of the internal organs may not be conclusive, except for the presence of spirochetae. Lomer examined 43 fetuses, all dying before the thirtieth week of pregnancy; he found the spleen and liver enlarged in all, and marked bone changes in 21. Birch-Hirschfeld examined 108 newly-born syphilitic infants; he found the spleen invariably enlarged; typical bone changes were present in 35, but in many cases the bones were normal. More recent studies of the bones have shown them to be involved in a much larger proportion of cases than is given by these writers. Mervis,

from an examination of 92 syphilitic fetuses, states that no eruption upon the skin was found earlier than the eighth month.

Symptoms are present at birth in only a small number of cases. In such there is usually a very severe degree of infection, and the infants do not often live more than a few days. Upon the skin there may be seen an eruption of pustules, papules, or bullae. The bullae are usually upon the soles and palms, but may be found upon other parts of the body. The name "syphilitic pemphigus" is often given to this condition. The bullae are at first small, then may coalesce and form larger ones two inches or more in diameter. They contain a turbid serum which is sometimes tinged with blood, and sometimes yellow from pus. Pustules, when present, are usually seen upon the face or scalp. The general appearance of these infants is wretched in the extreme. The body is wasted, the skin wrinkled, and temperature subnormal. The spleen is usually enlarged and often the liver also. Death usually occurs from inanition within two weeks.

In the great majority of cases the infant appears healthy at birth, and continues so for a variable time before the manifestation of the characteristic symptoms of syphilis. As a rule, the more intense the infection, the earlier the symptoms make their appearance. The earliest symptoms are generally seen between the second and the sixth weeks. If three months pass without evidence of syphilis, the probabilities are that the child will escape. Miller (Moscow) gives the following statistics of the time of beginning of symptoms in 1,000 cases:

Symptoms appeared	during	the	first	week	85 cases.
"	"	"	second	week	138 "
"	"	"	third	week	240 "
"	"	"	fourth	week	177 "
"	"	"	fifth	week	86 "
"	"	"	sixth	week	54 "
"	"	"	seventh	week	50 "
"	"	"	eighth	week	30 "
"	"	after	the	eighth week	140 "

Sometimes the constitutional symptoms—wasting, cachexia, etc.—are noticed before the local ones, but usually this is not the case. Generally the first symptom is the coryza or "snuffles," which resembles an ordinary cold in the head, except that it persists. It is often accompanied by a hoarse cry, indicating that the larynx participates in the catarrhal inflammation. Soon the eruption makes its appearance, being generally first seen upon the hands, feet, and face. Fissures and mucous patches may be seen upon the lips, about the anus, and elsewhere. There is often slight fever, from 99° to 101° F. There may also be observed excessive tenderness and swelling about the shoulders, elbows, wrists, or ankles,

due to epiphysitis, which may cause the child to cry from the slightest amount of handling, and the limbs may be moved so little that paralysis is suspected.

In a severe case, as these local symptoms develop, the infant's general nutrition suffers. He loses steadily in weight, he becomes extremely anemic, and whines and frets almost continually, but especially at night. The features have a pitiful, drawn expression; the face is wrinkled, giving the infant a very old appearance. The skin has a peculiar sallow color, which has been well described as *café au lait*. The symptoms may continue until a condition of extreme marasmus is reached, or death may occur from some intercurrent affection of the lungs or digestive organs. Wasting is, however, very common in infants that are premature or very small at birth. Even without hereditary syphilis the question of nutrition is then a difficult one. Indirectly by causing prematurity, the syphilis is responsible. It is remarkable to see how well some children with extensive evidences of syphilis thrive, provided they were full-term infants and are breast fed.

FIG. 181.—EARLY ERUPTION OF HEREDITARY SYPHILIS. Infant two months old.

In the milder forms of infection the severe constitutional symptoms described are not seen, although the local evidences of disease are well marked. The severity of the symptoms is also much modified by treatment, especially when this is begun early.

The most important local symptoms are the coryza, eruption, fissures about the mouth and anus, mucous patches, painful swellings at the extremities of the long bones, pseudoparalysis, and onychia.

Coryza.—In most of the cases this is the first symptom. Beginning like an ordinary catarrh, it is distinguished by its severity and its persistence. There is a copious discharge of mucus and serum, often tinged with blood. Thick crusts form, which produce the usual symptoms of nasal obstruction; there is great difficulty in nursing; the infant breathes through the mouth, and the mucous membrane of the mouth is dry, causing great discomfort. If untreated, the process, which at first involves

the mucous membrane only, may extend to the submucous tissue, causing ulceration; but the cartilages and the bones of the nasal fossae are not often involved till a later period in the disease.

The nasal catarrh is associated with more or less laryngitis, causing hoarseness or aphonia, and rarely there may be laryngeal stenosis. Dillon Brown has reported one case in an infant six weeks old, which recovered after intubation.

Eruption.—The early eruption usually appears after the coryza has lasted about a week; but the two may come at the same time; or the

FIG. 182.—EARLY ERUPTION OF HEREDITARY SYPHILIS. Infant two and one-half months old.

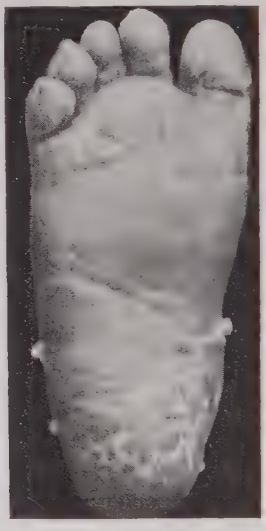


FIG. 183.—SYPHILITIC SCALING OF THE FOOT. From an infant eight weeks old.

coryza may be absent or so slight that the rash seems to be the first symptom.

Occasionally there is seen a diffuse blush or roseola, but usually the eruption is macular, occurring in small, dark-red spots about the size of the infant's finger nails, usually circular and often slightly elevated; there is no surrounding inflammation, and no itching. It is usually most abundant about the center of the face, the extensor surfaces of the upper and lower extremities and especially the hands and feet. It may extend over the entire body, but is generally absent over the chest and abdomen. At first the color is bright, but gradually becomes of a dusky-red or coppery hue. After a little time very fine scales may be seen upon the surface of the red macules. The rash comes out slowly, usually requiring from one to three weeks for its full development. It fades gradually, leaving a coppery discoloration of the skin, which continues for a long time. The

duration of the eruption is from three to eight weeks; less if active treatment is employed.

A papular eruption is rarely seen alone, but is usually associated with the macular variety. The papules are of a brownish color and are hard. They are seen most frequently upon the palms and soles.

A squamous eruption is frequently seen upon the palms and soles, but not often elsewhere. In a few cases this scaliness forms the most distinctive feature of the cutaneous lesion (see Fig. 183).

Fissures and Mucous Patches.—These are among the most diagnostic features of early hereditary syphilis. Fissures are most frequently seen on the lips and about the anus, but they may occur about the nostrils and occasionally elsewhere. The fissures of the lips are really linear ulcers, and are distinguished by their persistence in spite of local treatment.

FIG. 184. A LATER FORM OF ERUPTION IN HEREDITARY SYPHILIS.
Infant eight months old.

They are multiple, deep, painful, and bleed easily. After healing, these fissures may leave many cicatrices radiating from the mouth, the contraction of which produces the so-called "purse-string" deformity.

Mucous patches may develop from fissures, but more frequently from papules which are situated in regions where they are exposed to constant moisture and friction. They are very common upon the mucocutaneous surfaces and wherever the skin is especially thin. They are most apt to be seen about the lips, anus, scrotum, and vulva, but they may also be found behind the ears, between the toes, in the folds of the groin, axillae, or buttocks. They vary from an eighth to half an inch in diameter, are whitish in color, and are raised rather than excavated.

Ulcers may be present upon any of the mucous membranes, frequently in the mouth or on the genitals; they are seldom symmetrical, and while they may be broad they are never deep.

Hemorrhages.—They are generally associated with the lesions of the mucous membranes, especially of the nose. In young infants with severe

infection, bleeding may occur from the bullous eruption upon the skin, or from the fissures at any of the orifices, particularly the mouth and anus. Fischl has reported seven cases of multiple hemorrhages in the newly born, associated with other symptoms of congenital syphilis. Mracek noted hemorrhages in thirty-three per cent of 160 autopsies on syphilitic still-born infants or those dying soon after birth. Examination of the blood-vessels in some of these cases showed infiltration of their walls and narrowing of their lumen. The vascular changes were thought to be the cause of the bleeding.

Nails.—The nails present several peculiarities in syphilitic infants. There may be a disease of the matrix resulting in suppuration and exfoliation of the nail; frequently the dorsum is much arched, and the nail appears as if it had been pinched by a pair of forceps—i. e., claw-shaped; this is an early symptom of some diagnostic importance. The hair and eyebrows frequently fall out completely. This symptom is not usually present in very early infancy.

Pseudoparalysis.—This is due to acute epiphysitis, and it may be the first symptom of hereditary syphilis to attract attention. It is usually noticed when the infant is a few weeks old, that one or more extremities is not moved, and that the parts are tender when handled. The limb lies perfectly motionless, and any attempt at passive movement causes evident pain. A history will usually be obtained that the loss of power did not exist at birth but developed subsequently. If the arm is affected it is very frequently held in marked inward rotation with the palm looking outwards, resembling the position in Erb's palsy. There is tenderness on pressure, and soon swelling is seen, both being most marked at the epiphyseal line. If the bone affected is superficially situated, as the lower epiphysis of the humerus, radius, or tibia, swelling is very apparent, while it may be scarcely perceptible at the upper epiphysis of the humerus. The swelling is usually cylindrical and moderate in degree, being limited to the extremity of the bone. Separation of the epiphysis may take place, so that crepitation is obtained by moving the limb. With this there is sometimes suppuration due to secondary infection. The X-ray shows in many instances an increase in the width of the epiphyseal line which may be very irregular. (Fig. 185.)

In the milder cases, or those which have been subjected to active treatment, both the swelling and the tenderness subside rapidly without suppuration; and even though the epiphysis has separated from the shaft it speedily unites. When pseudoparalysis has been the chief symptom, very rapid improvement occurs under treatment, and usually there is complete recovery of function in two or three weeks. If secondary infection takes place the condition is usually fatal.

Syphilitic Osteoperiostitis.—This is usually found in infancy only

as the result of a severe infection. It chiefly affects the long bones, especially the tibia, fibula, radius, humerus, phalanges, and metacarpal and metatarsal bones. The lesions are multiple, often symmetrical, and at this age are principally periosteal. They are generally situated near the ends of the shaft. The swellings caused by the periostitis can be made out readily when they are but slightly covered by muscles or fat. It may, however, be impossible to demonstrate their presence except by means of the X-ray. The swellings are firm and often distinctly tender. They are frequently associated with the symptoms of syphilitic epiphys-

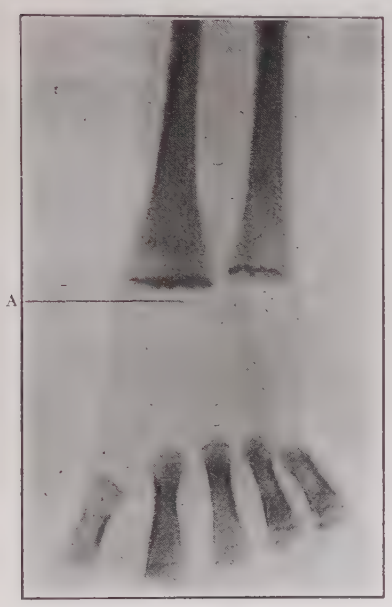


FIG. 185.—HEREDITARY SYPHILIS. Showing irregularity and exaggeration of line A. Infant two months old.

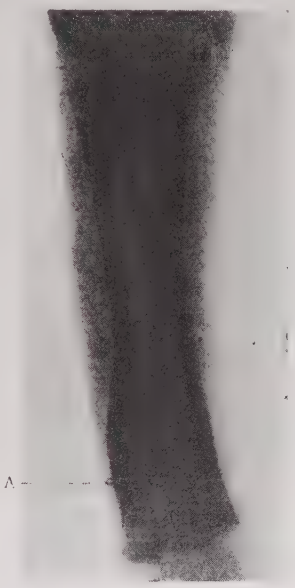


FIG. 186.—SYPHILITIC PERIOSTITIS OF THE FIBULA. Infant three months old. Same patient as Figs. 187–190 Right side affected; left side normal.

itis. The X-ray picture shows a fusiform swelling chiefly due to periosteal thickening. (Fig. 186.)

Syphilitic Dactylitis.—This is found in infants usually between the third and seventh months. It is not a frequent manifestation of syphilis. When present there are usually other evidences of bone syphilis, such as periosteal swellings, for the dactylitis is an osteoperiostitis but usually differs from that affecting other bones in that the involvement of the bone, even at this early age, is considerable and the periostitis rather slight. By means of the X-ray it can be seen that the phalanx involved is much thickened and of denser structure than the normal. Except for the fact that more than one and frequently several phalanges are in-

volved, the symptoms closely resemble the tuberculous form. The enlargement is spindle-shaped, involving the entire phalanx. It is usually not painful. It slowly increases in size and but rarely goes on to suppuration or necrosis. The disease may be arrested and cured by constitutional treatment.

Lymph Nodes.—These are often palpable. Marked enlargement is uncommon. No aid in diagnosis can be obtained from any but the epitrochlear glands. If these are considerably enlarged in infancy without evident adequate explanation, a suspicion of syphilis should always be aroused. They may be at times almost the only evidences of the disease.

The only visceral symptoms of importance are, enlargement of the

Fig. 187. Fig. 188.

Figs. 187, 188.—Syphilitic Dactylitis. On right hand first phalanges of forefinger and little finger affected; on left hand first phalanx of thumb and second phalanx of second finger.

spleen, which is almost invariably present in the active stage of hereditary syphilis, and jaundice with or without enlargement of the liver (see Icterus of the Newly Born).

Late Hereditary Syphilis.—The symptoms may come on at any period during childhood or about the time of puberty, but rarely at a later time than this. They are seen both in those who have had the usual symptoms of hereditary syphilis in early infancy, and in others where the most careful examination into the history fails to disclose any symptoms whatever of early syphilis. It is fair to assume in such cases either that early symptoms were absent or that they were of trivial importance.

Late hereditary syphilis shows itself by symptoms which in acquired disease would be classed as tertiary. The most characteristic are the affections of the teeth, the bones, the eyes, gummatous deposits in the solid

viscera, the skin or mucous membranes, the breaking down of which may lead to ulceration, and, finally, symptoms of disease of the nervous system.

Teeth.—There are no peculiarities in the first teeth of syphilitic chil-

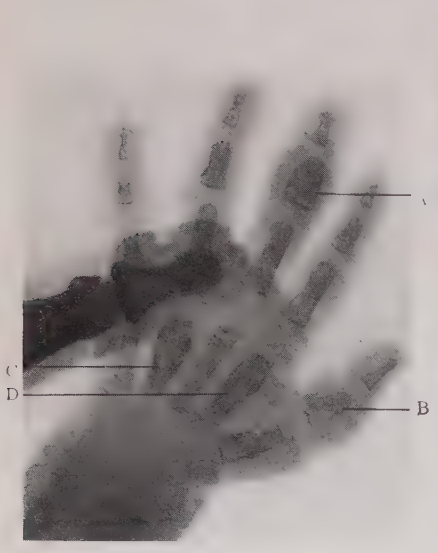


FIG. 189.

FIG. 190.

FIGS. 189, 190.—SAME HANDS AS FIGS. 187, 188. Note that besides the bones shown in the other pictures, two metacarpal bones (*C*, *D*) are affected in the left hand and the lower end of the radius (*G*) in the right hand.

dren except their proneness to early decay. They are rather more likely to appear early than late.

The characteristic teeth of syphilis are those of the second set. In estimating the diagnostic value of these changes, only the upper central incisors are to be relied upon; these are the test teeth. Although changes are frequently seen in other teeth, they are not always diagnostic. Typical syphilitic teeth, according to Hutchinson, have each a single notch in the center of the edge (Fig. 191). The notch is usually shallow and more or less crescentic in shape. The enamel is generally deficient in the center of the notch, and the tooth here is apt to be discolored. The teeth in other cases are variously dwarfed and deformed (Fig. 192). They often taper regularly from the base to the edge, giving rise to the term "screw-driver teeth." The teeth often are not so flat as the normal incisors, but often rounded and peg-like. They are not properly placed, but incline either

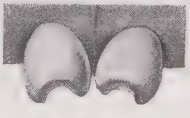


FIG. 191.—TYPICAL "HUTCHINSON'S TEETH." (After Fournier.)

toward or away from each other. They are seldom large enough to touch the adjacent teeth on both sides.

Although Hutchinson's teeth may generally be taken as conclusive evidence of syphilis, they are not invariably so, as Keyes and others have shown. It is to be remembered in this connection that the absence of changes in the teeth is of no importance whatever as evidence that syphilis is not present. Hutchinson states that they are wanting in more than half the cases.

Bones.—The form of disease which is usually seen at this period is an osteoperiostitis, affecting principally the shaft of the long bones and the cranium. Chronic osteoperiostitis is more frequent after the third year, and most of the cases occur between the fifth and fourteenth years. The most common seat of disease is the tibia, and next to this the bones of the forearm and the cranium. The following is the frequency with which the different bones were affected in the series of cases reported by Fournier: tibia in 91 cases, ulna in 22, radius in 15, cranium in 16, humerus in 12, all others in 37. The process may result either in a diffuse or a localized hyperplasia of bone or in necrosis.

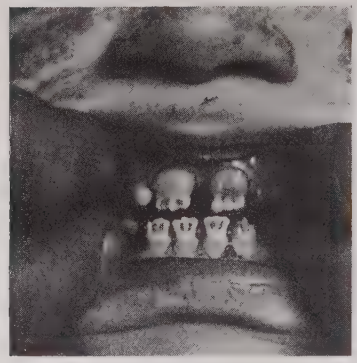


FIG. 192.—SYPHILITIC TEETH. Boy eight years old; under observation several years with various syphilitic manifestations.

The typical changes are seen in the tibia. The shaft of the bone is principally or solely affected. There is often produced a very characteristic deformity, consisting of a forward curve of the anterior border of the tibia, which has been compared to a saber blade (Figs. 193, 194). In some cases the bone is bent inward at its lower third, resembling somewhat a rachitic curvature. Sometimes the entire shaft of the bone is affected, and it may be greatly enlarged. At other times the swelling is chiefly near the epiphysis, where large bosses may form of sufficient size to interfere with the functions of the joint. Instead of affecting the bone uniformly, the disease often affects only certain parts, leading to the formation of large nodes which are more likely to be followed by necrosis than are the other lesions. In most of the cases the process is purely a hyperplastic one, leaving the bone permanently enlarged and the limb often lengthened. Less frequently, there occur gummatous deposits in or beneath the periosteum, which may soften, suppurate, and lead to superficial necrosis, with the formation of sinuses that remain open until the sequestrum is exfoliated. Syphilitic deposits sometimes take place in the interior of the bones, generally

near the articular ends (Fig. 194); these may soften and break down with abscesses, sinuses, etc., very much after the manner of a tuberculous inflammation.

The lesions of the other long bones are essentially the same as of the tibia. They are nearly always symmetrical and often multiple. The course of syphilitic osteoperiostitis is very chronic, and some permanent deformity is the rule, unless cases come very early under treatment.

When affecting the bones of the cranium the disease usually takes the form of a gummatous periostitis, which leads to the formation of large

FIG. 193.—SYPHILITIC OSTEOPERIOSTITIS OF THE TIBIA. Left tibia greatly enlarged; 1½ inches longer than the right, and leg 2 inches larger in circumference; saber-like anterior border. Right tibia normal; lesion of long standing. Patient 13 years old.

nodes. These may remain as permanent deformities, or they may break down and suppurate, with necrosis of one or both tables of the skull. This may be followed by inflammation of the dura, the pia, and even of the brain itself.

When the long bones are affected, the symptoms are pain, tenderness, and deformity. These come on very gradually, and often the deformity is noticed before either pain or tenderness is sufficiently marked to attract attention. The pain is regularly worse at night, and often felt only at that time; it may be mild and occasional, or so severe as virtually to prevent sleep. There is tenderness on pressure over the bones affected,

the acuteness of which will depend upon the activity of the process. When suppuration occurs, it comes very slowly, and never with symptoms of acute inflammation. Sinuses usually continue to discharge until a sequestrum is exfoliated. The course of the disease is very tedious, and the whole duration is usually several years.

Fig. 194.—Syphilitic Osteoperiostitis of the Left Tibia. Similar lesion to that shown in Fig. 193; patient 8 years old. The right tibia is normal.

When the cranium is affected, there are seen irregular nodes, especially upon the frontal and parietal bones. They are from one to two inches in diameter, and project from one-eighth to one-fourth of an inch above the general outline of the skull. There may be pain, tenderness, softening, suppuration, and necrosis, as in the long bones.

It is rare that disease of the bones of the cranium is due in childhood to any other cause than syphilis, and this disease may usually be assumed to exist if traumatism can be excluded. The bosses upon the cranium

in rickets are always large, smooth, and regular in position, and belong to infancy.

Syphilitic disease of the long bones is recognized by the nocturnal pain, the tenderness and peculiar deformity, and by the association of other late manifestations of syphilis—i. e., the peculiar notched teeth, the interstitial keratitis, the enlarged epitrochlear glands, etc. Tuberculous disease generally affects the articular ends of the bones; syphilis nearly always the shaft. The diffuse hyperplasia of the tibia and the saber-like deformity of its anterior border are rarely, if ever, due to any other cause than syphilis. The deformities of the long bones have in some cases a certain resemblance to those due to rickets, but the two conditions can hardly be confused if a careful examination is made.

Arthritis.—This may occur in a subacute or even acute form. It is most common in the knee, though any of the large joints may be involved. The lesion is chiefly synovial. The onset may be sudden with pain and marked tenderness. Effusion into the joint occurs and there is local heat and often a rise in temperature to 101° F. or more. The process usually remains limited to one joint and resists obstinately all methods of treatment except antisyphilitic treatment, to which it readily yields.

Lymph Nodes.—They are less frequently affected than in adults. In most cases there may be found a moderate degree of enlargement of the postcervical and epitrochlear glands, swelling of the latter having considerable diagnostic value. Under normal conditions these can scarcely be felt; but in syphilitic children they may be as large as a pea or a small bean; sometimes two or three of them can be distinguished. Provided no local cause for the swelling exists, they should always create a suspicion of syphilis. The postcervical glands are frequently affected, but are not so diagnostic. The degree of enlargement is rarely great. Occasionally there are seen in the neck large masses of swollen lymph glands which resemble tuberculous swellings. They are, however, very rare.

Special Senses.—The most frequent affection of the eye in late syphilis is interstitial keratitis, the close connection of which with hereditary syphilis was first pointed out by Hutchinson. It is often found associated with the typical notched teeth. The diagnostic value of keratitis in syphilis is denied by Fournier, who states that, while often syphilitic, it is not infrequently due simply to malnutrition. We cannot subscribe to this statement. In our experience it is almost always due to syphilis. Both eyes are usually affected, and in all degrees of severity, from a slight haziness of the cornea to complete opacity. However, with an early diagnosis and prompt treatment, a marked degree of improvement may be expected in most cases.

Chronic otitis may be a result of the acute process seen in early infancy. There is nothing peculiar about the inflammation in these cases. A form of deafness occurs in older children, which Hutchinson states is almost invariably due to syphilis. Its onset is quite sudden, without pain. The loss of hearing is apt to be permanent, and if it occurs early in childhood it is a cause of deaf-mutism.

Skin.—The most important of the later manifestations of syphilis consists in the formation of subcutaneous gummata. In the early stage they are indurated, elastic, of a grayish color, with red borders. Under treatment they disappear quite rapidly by absorption; but when neglected they break down, leaving large deep ulcers. These ulcers are quite characteristic in appearance, but may be confounded with those due to tuberculosis. The syphilitic ulcer has rounded, thickened, indurated borders, and a base which is depressed and has the appearance of being scooped out. It is sometimes covered by hard crusts and is surrounded by a red areola. It leaves a smooth white scar. The most frequent situation is upon the face and upper part of the legs or thighs. Tuberculous ulcers have usually soft, flat edges, and do not extend so deeply; they are more irregular in outline; the cicatrix left is of a purplish color, which becomes red and slowly fades; and tubercle bacilli may be found.

Nose and Palate.—Disease of these parts generally begins as the breaking down of gummatous deposits in the mucous membrane. The nose may in consequence be the seat of a protracted fetid discharge (ozena). The disease may take on a destructive form of ulceration which is at times phagedenic, and may cause rapid destruction of the nasal cartilages and bones, perforation of the septum, and occasionally of the floor of the nasal fossae. There may be necrosis of the turbinated bones, the vomer, or the ethmoid. In the most severe forms the nose may be almost destroyed in the course of a few weeks. There may be at the same time deep ulceration of the soft palate, leading to perforation. In a young person this is almost invariably due to syphilis. In many particulars these ulcerations of the nose and palate resemble lupus; they are distinguished by the rapidity of their progress, syphilis often doing as much damage in weeks as is done by lupus in years.

Other Symptoms.—Syphilitic disease of the larynx and bronchi is rare in childhood. The former may give rise to hoarseness or aphonia and occasionally to stenosis; the latter to a chronic cough and asthmatic attacks. There are no characteristic symptoms belonging to syphilis of the lungs.

The only visceral changes which aid much in diagnosis are those of the liver and spleen. The liver is often enlarged, sometimes to a marked degree, and occasionally there is ascites, but very seldom jaundice.

Enlargement of the spleen is a very frequent symptom—in fact, it is

almost constant during active syphilitic disease. It is occasionally so swollen as to form an abdominal tumor of considerable size. In one case under our observation, in a boy three years old, the spleen extended five inches below the free border of the ribs, quite to the crest of the ileum. It was associated with moderate enlargement of the liver, as is usually the case.

In addition to the local symptoms of late hereditary syphilis enumerated, there are others of a general character which are quite as important. The body is usually undersized; the constitution is delicate, and shows but little resistance to all forms of disease; puberty is frequently delayed, and the development of the breasts and the genital organs often imperfect; anemia is usually present, and the skin has a sallow appearance. Mentally, many of these children are somewhat deficient.

Syphilis of the Nervous System.—This may show itself in a great variety of ways. There may be a combination of symptoms giving rise to a more or less distinct clinical picture, indicating diffuse involvement of one or more parts of the brain or cord, or the lesion may apparently be limited to a strikingly small area.

Paralysis of single nerves, particularly the cranial nerves, is not uncommon. There may be only failure of one or both pupils to react to light, or there may be strabismus. Sudden deafness may occur. There may be a gradually developing optic atrophy.

Mention has been made of syphilis as a cause of hydrocephalus. In our experience the association between the two diseases is unusual, but many clinicians with large experience emphasize the fact that hydrocephalus may often be due to syphilis. Epilepsy, also, may depend, but in our experience very infrequently, upon syphilis. Statistics vary much as to the rôle of syphilis in producing feeblemindedness. Studies upon inmates in institutions for the feebleminded in this country have shown that not more than two or three per cent have clear clinical evidences of syphilis, while not more than ten per cent without physical symptoms of the disease give a positive Wassermann reaction. This, of course, does not indicate that syphilis is in the ten per cent the cause of the feeblemindedness. The association of the two conditions may be merely accidental. It is probable that the part of syphilis in the production of mental deficiency has been exaggerated. Lesions of the cord due to syphilis are distinctly uncommon.

Juvenile paresis is occasionally seen, but it is rare before the fifth year. There is no doubt of its dependence upon hereditary syphilis. The symptoms usually appear shortly before or about the time of puberty. They are quite characteristic. A child that has developed in a practically normal way gradually begins to lose his ability to do certain things.

There is loss of memory and a difficulty in speech, which consists in dropping a syllable or a whole word. If he has been able to write, the capacity to do this is gradually lost. Eventually speech is impossible and the intelligence is reduced to a minimum. Walking becomes difficult and later almost impossible. The child loses all sense of cleanliness and remains in a demented condition often for years until death occurs from inanition, bed-sores or from intercurrent disease. There is usually loss of reaction of the pupils to light, irregularity of the pupils, and often some degree of optic atrophy. The cerebrospinal fluid contains an excess of cells and globulin and gives a strongly positive Wassermann reaction. The course is slowly but progressively downwards.

It is at times difficult to differentiate from juvenile paresis a form of cerebral syphilis, which in our experience is more common than the paretic form. The history often gives valuable aid, showing that the child has ever appeared entirely normal. There has usually been, almost from the beginning, some, often a marked, degree of mental impairment and speech has been slowly and imperfectly acquired. The children are oftentimes restless and disobedient. They may have screaming attacks. The reflexes may be exaggerated or absent. Attacks of headache and vertigo with vomiting are not uncommon. There may be unequal pupils or failure to react to light. Some degree of optic atrophy is generally present. Hemiplegic attacks may occur in the course of the disease or they may appear as the first evidence of cerebral involvement. These attacks may occur first on one side and then on the other, and the paralysis often improves to a marked degree, even without treatment. With this form of cerebral syphilis there is not the same tendency to mental and physical deterioration as with paresis. The children may live many years in about the same mental condition. Sometimes with treatment, especially if it is begun early, considerable improvement occurs. The cerebrospinal fluid shows in these cases also an excess of cells and globulin and always gives a strongly positive Wassermann reaction. As is the case with paresis, it is exceedingly difficult to diminish the intensity of or to abolish the Wassermann reaction in the spinal fluid by antisyphilitic treatment of any kind, no matter how vigorously given or how often repeated.

Tabes may be found in childhood as the result of hereditary syphilis but is very uncommon. The symptoms are similar to those of the adult form of the disease, but some of them may be absent. The Argyll-Robertson pupil is constant, but the patellar reflexes may not be lost and Romberg's symptom may not be marked. Incontinence of urine is frequent. The course of the disease is exceedingly slow. It may last for fifteen or twenty years or even more.

Diagnosis.—The diagnosis of early syphilis in most cases is not diffi-

cult. The coryza, eruption, labial fissures, mucous patches about the anus and genitals, enlarged spleen, and later the general cachexia—all unite to form a picture which it is difficult to mistake. In irregular cases the diagnosis is easy just in proportion to the number of the foregoing symptoms which are present. Special care should be taken not to confound the moist papules of simple intertrigo upon the buttocks or thighs with those of syphilis. Much assistance may be obtained, especially in early cases, from the discovery of the spirochetae in the external lesions. This is a means of diagnosis which is too seldom employed. In a series of 34 cases, mostly early ones, in the hospital service of one of us, there were external lesions in 22, in all but one of which the spirochetae were demonstrated. The dark field is useful but not essential. They can easily be demonstrated by the India ink method. The Wassermann reaction has the same value as in adults.

In late syphilis the following symptoms are the most reliable for diagnosis: notching of the teeth, falling in of the bridge of the nose, interstitial keratitis, deafness not traceable to ordinary otitis, enlargement of the spleen and epitrochlear glands, ulceration of the palate or nose, the saber-like deformity of the tibia, and nodes upon the tibia or cranium. There are often found in older children indefinite symptoms in regard to which a suspicion of syphilis exists. For such cases the Wassermann test is of very great value.

It becomes at times important to distinguish hereditary from acquired syphilis. Visceral lesions in acquired syphilis are not common and belong to the late period of the disease; in the hereditary form they are well-nigh constant and occur early, often being present at birth. The acute epiphysitis, sometimes accompanied by pseudoparalysis, seldom if ever occurs in acquired syphilis, though frequent in the hereditary form. Symptoms due to defects in development, like the misshapen finger-nails, are seen only in hereditary syphilis. The early symptoms referable to the mucous membranes and mucocutaneous surfaces—coryza, hoarseness, hemorrhages, labial fissures, etc.—so characteristic of hereditary syphilis, have no place in the acquired form, while the single primary lesion sometimes found in the acquired form does not exist in the hereditary disease.

The value of Noguchi's cutaneous "luetin" test has not yet been finally settled. Considerable experience is needed to interpret results.

Prognosis.—Generally speaking, the prognosis is worse in infantile syphilis than in that of adults. In infancy it is much worse when hereditary than when acquired, for the reason that often the child who is the subject of hereditary syphilis has been affected by the poison from the very beginning of his existence, and this has modified his entire development.

The results of 206 syphilitic pregnancies observed by Julien (Paris) were as follows: abortion occurred in 36, stillbirths in 8, and 69 children died soon after birth, making a total mortality of 55 per cent; 50 were living and syphilitic; only 43 living and in good health. Still worse were the results in cases observed by Le Pileur: of 154 pregnancies in syphilitic women, there were 120 abortions or stillbirths, 26 children died soon after birth, and only 8 survived. The statistics of the Foundling Asylum in Moscow for ten years showed that of 2,038 syphilitic infants the mortality was over 70 per cent.

Such a mortality as that indicated in the above statistics is seen only in institutions where little or no previous treatment has been employed. In private practice certainly nothing approaching it occurs.

In addition to those who die early as the result of syphilitic infection, there must be added many whose constitutions are so impaired by syphilis that they fall an easy prey in infancy to pneumonia, diarrhea, or other forms of acute disease. The remote effects of syphilis in infancy it is hard to estimate; it may exert an injurious influence upon the constitution in childhood and even throughout the life of the individual.

The prognosis in an individual case depends upon the age at which the symptoms develop, the time when treatment is begun, upon its thoroughness, and upon the surroundings and mode of nourishment of the child. The outlook is better the longer after birth the first symptoms appear; it is also very much better in infants who are nursed than in those who are artificially fed.

As compared with syphilis of the adult, relapses are less frequent, and when they occur early they are nearly always the result of insufficient treatment. If proper treatment is carried out, these severe late symptoms are not common; patients are usually free from all symptoms until six or seven years old, or until near the time of puberty—two periods when they are likely to develop. We must conclude that treatment persisted in only for a short time and not energetic enough to influence in any way the Wassermann reaction has, nevertheless, a great influence in preventing the further ravages of the disease. We have observed children after an interval of several years that had been treated in this unsatisfactory way and could find no evidence of the disease but a positive Wassermann reaction. It is a fact also that most of the patients that apply for treatment for late hereditary syphilis have never received any treatment.

The prognosis is better in the later children of syphilitic parents than in the earlier ones, provided infection has preceded the birth of all the children. This fact illustrates the general tendency of the syphilitic poison to diminish in virulence as time passes, even without treatment. The following instance cited by Bertin well illustrates this point:

In the first pregnancy, the mother aborted with a dead child at the

sixth month; in the second, at the seventh month; in the third, at seven and a half months; in the fourth the child was born at term, and lived eighteen days; in the fifth it lived six weeks; in the sixth the child lived four months, without treatment.

The prognosis of syphilis of the nervous system should be considered by itself. Certain of the manifestations, such as localized paralyses, may yield promptly to treatment. It is also reported that many cases of syphilitic epilepsy and hydrocephalus have been greatly improved or cured. Gummatous lesions usually disappear promptly with appropriate treatment as in acquired syphilis. But the lesions of the nervous system are usually the result of arterial disease or of meningitis and encephalitis. These are very little influenced by treatment. In cases of diffuse involvement of the brain and in juvenile paresis, we have not seen lasting benefit from even the most energetic and long-continued treatment with salvarsan or with mercury and iodids.

Prophylaxis.—No infected person should be allowed to marry until at least two years have passed after the initial sore, treatment being continued meanwhile; nor if there are any active symptoms, no matter how long a time has elapsed since infection, nor if the Wassermann reaction is positive.

The mother should be treated during her pregnancy: (1) If she is syphilitic, whether the disease was acquired at the time of conception or subsequently; (2) if the father is known to be suffering from syphilis, whether the mother has symptoms or not; (3) if the mother has ever previously shown signs of syphilis, even if she has had no active symptoms for a considerable period. In all these conditions if efficient treatment is carried on throughout pregnancy there is a strong probability, but in no case a certainty, that the child will escape. The third condition mentioned is the one in which treatment is most likely to be neglected, especially if the mother has previously borne a child who was not syphilitic. Syphilis, however, shows a strong tendency to reappear and become active during pregnancy, even though it has been long quiescent, as the following case cited by Diday shows:

A woman who had lost seven children from syphilis was put under treatment during the eighth pregnancy; result—child born healthy, and continued so. In the ninth pregnancy treatment was continued with a like result; in the tenth pregnancy, no treatment, child syphilitic, dying when six months old; in the eleventh pregnancy, treatment repeated, child healthy.

The danger of infection during labor is slight. As the greatest danger of infecting a child after birth is from his parents or a wet-nurse, syphilitic parents should be duly warned of the danger to their children, and especially should be cautioned against kissing them or sleeping in

the same bed with them. The utmost care should be exercised to prevent a healthy child from being infected by a syphilitic nurse. A nurse should never be accepted without a thorough physical examination, no matter how clear a history may be given. As a syphilitic child in the household may be the means of infecting other children, the same precautions should be taken as in the case of other contagious diseases. The chief danger to other children comes from kissing or from using bottles, spoons, or cups which have been infected; as the syphilitic infant is chiefly dangerous on account of the lesions in the mouth. Trouble most frequently occurs because of ignorance regarding the nature of the disease. It is possible for a syphilitic child to nurse a healthy woman without communicating syphilis, if the child's mouth contains no lesions and the nipple not allowed to become fissured; but it is an experiment which should never be tried.

Treatment.—This should always be begun as soon as the first positive symptoms of syphilis appear. In certain circumstances it may be advisable not to wait for symptoms; as, for example, when both parents have recently suffered from active symptoms, when previous children have died soon after birth, or when, with marked symptoms in the parents, the child exhibits the cachexia of syphilis, but no definite local symptoms. Such anticipatory treatment need not be continued after a negative Wassermann reaction is obtained. It should be remembered, however, that even a syphilitic infant may give a negative Wassermann reaction for the first two or three weeks of life.

The indirect treatment, designed to reach the child through the mother's milk, has fallen into deserved disuse, as it is very uncertain and altogether unsatisfactory.

The two drugs most useful in treatment are mercury and salvarsan. Mercury is as much a specific for hereditary as for acquired syphilis. There are many ways of introducing it into the system—by inunctions, by mouth, by fumigations, baths, or hypodermically. In most cases, inunction is the manner to be preferred with children. Mercurial ointment in doses of from ten to twenty grains, depending upon the size of the child, diluted with an equal amount of vaseline may be rubbed into the abdomen, axillae, or the inner surface of the thighs. It is advisable to change the place of inunction from time to time and if this is done it is extremely rare that erythema is produced. It may advantageously be placed, with small infants, upon the inner surface of an abdominal binder. If for any reason inunctions are objectionable, as they may be when the family are to be kept in ignorance, either the gray powder or the bichlorid may be given by mouth. The usual dose of the gray powder is gr. ½, three times a day, and that of the bichlorid, gr. 1-60 three times a day, always well diluted. It is rare that larger doses are advisable. Calomel

in doses of 1-10 gr. four times a day is oftentimes a rapid method of bringing the system under the influence of mercury. Other methods of administration and other preparations offer no advantages and have some very obvious disadvantages. The duration of mercurial treatment should be at least one year. The doses during the last six months may be reduced to one half or one third of those employed while active symptoms were present. It is well to repeat two or three months of mercurial treatment during the second and third years, even if no symptoms are present. Treatment should always be employed longer than a year if symptoms exist. It is often better not to give the mercury continuously, but with short periods of intermission.

Salvarsan is quite as efficacious in infants as in older patients. Single doses of salvarsan do not cure syphilis and several doses may not do so. A repetition is always necessary and the best results are obtained when salvarsan is combined with the mercurial treatment. In such circumstances, it is wise to omit the mercury for a few days before and after the injection of salvarsan. The intravenous method of administration of salvarsan is altogether to be preferred on account of its irritating effects when injected into the tissues. The usual dose is .05 gram for very young infants and 0.1 gram for those who are five or six months old. More exactly it may be calculated as 0.01 gram for each kilogram (.005 per pound) of body weight. With infants, the injection may be made into a vein of the scalp or the external jugular vein. No dissection is necessary but care should be taken that none of the injected fluid is allowed to escape into the surrounding tissue, otherwise sloughing may result. Neosalvarsan has the advantages of being more readily prepared, much less irritating in its effect and consequently much less likely to cause necrosis if any escapes into the tissues. It is, however, less active and the dose should be one and one-half times that of salvarsan. The usual doses of neosalvarsan required by infants are readily given in 5 c.c. of freshly distilled water. The intravenous use of this preparation is greatly to be preferred. If, however, for any reason this is not practicable, neosalvarsan may be given intramuscularly, dissolved in some bland oil such as benzoinol. Salvarsan should not be so given.

Injections of salvarsan should not be made more frequently than once in two weeks, usually repeated four or five times and controlled by the Wassermann reaction. It is uncommon for a negative reaction to be obtained after less than three injections; we have used as many as eight and have found at times the reaction persistently positive.

The iodid of potassium may be used in combination with mercury whenever such lesions exist as are classed among adults as tertiary. This includes all the late manifestations and the earlier ones whenever the

bones or viscera are affected. The iodid is usually well borne by children and may be given in almost any desired dosage. In infancy, not more than gr. xx daily are required, but in older children one or two drams daily may be given, always largely diluted.

Syphilis of the nervous system is often but slightly affected by treatment, as has been mentioned previously. The symptoms of sharply localized disease, including the gummatous lesions, are usually promptly affected, but diffuse cerebrospinal syphilis, including paresis and tabes, is hardly benefited at all. The Wassermann reaction in the blood may sometimes be made negative, but the Wassermann reaction of the cerebrospinal fluid remains positive and the symptoms are in almost all instances entirely unaffected.

The general treatment of syphilis is important and should not be neglected. After specific treatment has been carried on for a time, particularly if rapidly pushed, the child often becomes anemic and suffers greatly from malnutrition. In such circumstances, it is usually wise to discontinue mercury altogether for a time, or at least to reduce the dose very much. Such a change is frequently found to act most beneficially.

Local Treatment.—Ulcerative lesions of the skin require cleanliness, dusting with calomel or iodoform, or bathing with the black wash. Mucous patches should be dusted with equal parts of calomel and bismuth. Fissures and ulcers of the mucous membranes should be treated by nitrate of silver. Phagedenic ulcers of the palate or nose should be cauterized with nitric acid or the acid nitrate of mercury. The late syphilitic ulcers of the skin, due to the breaking down of gummata, should be treated aseptically.

CHAPTER XII

INFLUENZA

In 1892 a bacillus was described by Pfeiffer which he believed to be the cause of epidemic influenza or what is commonly known as the grippe. It seems evident by the studies of the last half dozen years that this organism is not the cause of the grippe, although it is a pathogenic organism of considerable importance in respiratory diseases, and is associated with a pretty definite group of clinical symptoms. In this chapter we shall include under the term Influenza only the disease or diseases due to Pfeiffer's bacillus.

Etiology.—Pfeiffer's bacillus, or the influenza bacillus as it is known

in literature, is chiefly found in the secretions of the lower respiratory tract; less often in those of the upper tract—the rhinopharynx and discharges from the ears. As it usually occurs, it has been shown by Wollstein to be an organism of low virulence. It produces few immune bodies and consequently complement fixation cannot be demonstrated in the serum of these patients. It does not agglutinate except in very low dilutions. No immunity is developed from such attacks and hence patients are continually liable to recurrent influenza infection. Like the pneumococcus, Pfeiffer's bacillus may be present in the respiratory secretions without producing any symptoms whatever. It may be of no significance. At times very virulent strains of the influenza bacillus are met with. These produce antibodies and cause immunity; but unfortunately because of their virulence the patient is likely to be overpowered before this has occurred. The organism may quickly find its way from the respiratory tract into the blood stream, producing an intense septicemia and leading to the development of a severe form of pneumonia, to cerebrospinal meningitis, and rarely to inflammation of the large joints. Pfeiffer's bacillus belongs to the hemoglobinophilic group, growing only on a medium containing hemoglobin. It can be demonstrated in the sputum with certainty only by cultivation, smears being entirely unsatisfactory. In acute cases it may disappear very early; but in protracted cases its presence can often be demonstrated for weeks or even months. In the respiratory inflammations in which the organism occurs, although it may be found in pure culture, it is usually associated with the pneumococcus or the staphylococcus aureus, less frequently with the streptococcus. In routine cultures made from the sputum in acute respiratory infections in the winter and spring in the Babies' Hospital during a period of six years the influenza bacillus was found in different years in from 28 to 42 per cent of the cases.

Influenza may be ranked among moderately contagious diseases. It is rather more communicable than pneumococcus infections, but much less so than epidemic catarrh or the grippe. The influenza bacillus is regularly found in New York in the cold season, beginning early in November, but most years is not frequently found till after January. It usually disappears completely about the end of May with the advent of very warm weather. Its prevalence in the winter and spring of some seasons is so great that it may often be said to be epidemic. All ages are liable to the disease, infants especially so.

Lesions.—The influenza bacillus is much less frequently associated with the inflammations of the upper than the lower respiratory tract. It is found in comparatively few of the cases of acute rhinopharyngitis, in the severe inflammations which invade the antrum, the frontal or ethmoidal sinus or the middle ear. It is much more frequently associated with

inflammations of the trachea, bronchi, and lungs. There are no characteristic lesions of influenza. Those found in the respiratory tract differ little from the same inflammations when due to other organisms. The pneumonia is nearly always of the bronchopneumonia type. In certain cases resolution is much delayed or is incomplete and the inflammation may then develop into a chronic interstitial type which may continue indefinitely, with the later development of fibrosis in the lung of considerable extent with bronchiectasis, etc.

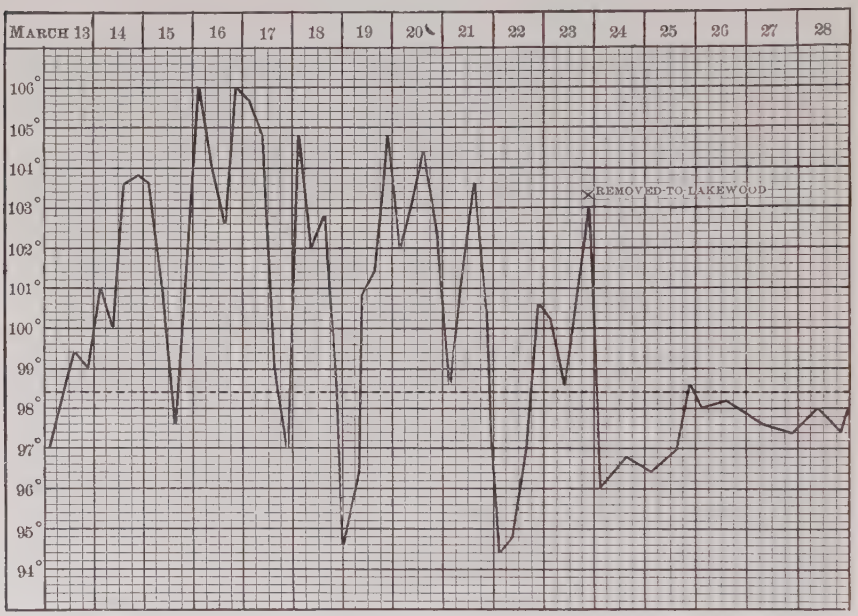


FIG. 195.—TEMPERATURE CHART OF UNCOMPLICATED INFLUENZA. Infant fourteen months old. No local signs of disease; repeated blood examinations for malaria negative; the wide fluctuations of the temperature independent of therapeutic measures. Prompt cessation of fever on removal from the city.

Symptoms.—The symptoms of influenza are in part due to the general infection and in part to the local inflammations which are excited. These may be regarded either in the light of manifestations or possibly as complications. The clinical manifestations of influenza are numerous and often exceedingly puzzling in diagnosis. Those most frequently met with are the following:

1. There may be only symptoms of a general infection of moderate severity, often with a high temperature but with few or no respiratory symptoms.

2. There are cases with symptoms of mild respiratory infections—bronchitis, otitis, etc.—or others with severe bronchitis or bronchopneu-

monia which present little unusual in their symptoms except quite extraordinary fluctuations of temperature.

3. A protracted form of bronchopneumonia or recurring attacks of acute bronchopneumonia with incomplete resolution, often mistaken for tuberculosis.

4. A protracted mild respiratory catarrh with little fever but with a paroxysmal cough which is almost indistinguishable from whooping-cough.

5. An especially severe form of infection with general blood infection often terminating in meningitis.

The chart (Fig. 195) well illustrates the first group of cases. There are often no local symptoms of importance to be found on the most careful examination; there is a high and widely-fluctuating temperature which is quite out of proportion to the other symptoms. The child does not appear to be seriously ill, yet the height of the temperature and its wide fluctuations are most alarming. Sometimes at the height of the fever there may be marked nervous symptoms — irritability, hyperesthesia, rigidity, stupor, etc., strongly suggestive of cerebrospinal meningitis; but with the fall in the temperature all these symptoms pass off in a few hours. In most of the cases the only symptoms present are such as accompany high temperature from any cause. In some there is an acute erythematous blush of the fauces and in many there is a slight cough. Often such a temperature as that shown in the chart may continue for several days, subside without treatment, and all symptoms recur after an interval of a few days or one or two weeks; finally a small area of pneumonia may be discovered, or perhaps otitis may develop as a later complication. The improvement in symptoms by change in climate is sometimes most surprising and occasionally an equally abrupt ending of the attack may occur without it. More frequently, however, the symptoms subside gradually. Malaria or some hidden focus of suppuration are most frequently diagnosticated.

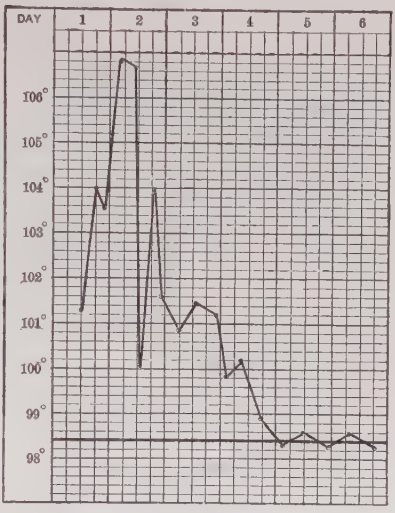


FIG. 196.—ACUTE BRONCHOPNEUMONIA, ABORTIVE TYPE, COMPLICATING INFLUENZA IN AN INFANT SIX MONTHS OLD. The entire left lung posteriorly was involved.

The cases of pneumonia associated with influenza are sometimes of such brief duration as to be classed as abortive (Fig. 196). The attack

begins like an ordinary pneumonia of perhaps more than usual severity; but after two or three days, generally before signs of complete consolidation have appeared, a rapid subsidence of symptoms and signs takes place with a speedy convalescence. In other cases of pneumonia more often seen the physical signs and general symptoms do not differ essentially from those of an ordinary pneumonia, but the temperature shows the same tendency to high and irregular fluctuations without evident reason, similar to those seen in the first group considered (see Fig. 62, Chapter on Pneumonia).

Influenza complicated by otitis often presents a most difficult problem in diagnosis. The early part of the attack may be with general symp-

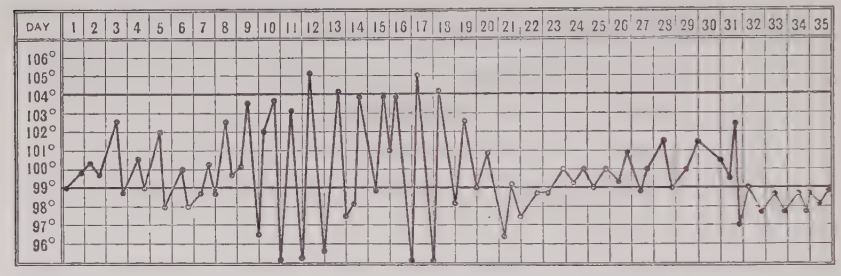


Fig. 197.—Influenza-bronchitis; Double Otitis; Late Bronchopneumonia; Autopsy. Infant, nine months old, admitted with influenza-bronchitis; double paracentesis fourth day, repeated on tenth day; the left ear opened again on twelfth and twenty-fourth days. The only signs in the chest were those of bronchitis until the eighteenth day, then bronchopneumonia which persisted until death. On account of the wide fluctuations in temperature from the eighth to the eighteenth day, mastoiditis and sinus thrombosis suspected. Operation not permitted, partly because of the child's poor condition, but chiefly because the bacillus influenzae was constantly present in the bronchial secretion and this was regarded as a sufficient explanation of the temperature. *Autopsy.*—Moderate bronchopneumonia; cultures from the lungs showed the influenza bacillus and pneumococcus. Careful examination of the mastoid and sinus showed no trace of disease.

toms which are not particularly characteristic. Otitis develops after a time as a complication; the ears are opened, the temperature does not subside, however, but assumes the widely fluctuating character seen in many cases of influenza. It is often assumed that the continuance of the temperature is due to some grave condition associated with the otitis —mastoiditis, sinus thrombosis, etc.—and serious operations have often been performed in these circumstances; whereas the fever was simply a manifestation of the general influenza infection upon which the paracentesis has of course had no effect (Fig. 197). Intercurrent attacks of influenza occurring in surgical cases with few or no respiratory symptoms may also be very puzzling.

The most characteristic forms of pneumonia accompanying influenza are the cases which in the early part of the attack may show little that

is unusual except the very irregular temperature curve. The signs are like those of ordinary bronchopneumonia, often with a lobar type of consolidation. The course is a very protracted one. The signs clear up very slowly and imperfectly. The children get better, but they do not get well. One attack often succeeds another, separated sometimes by an interval of only a few days, and sometimes of several weeks, and so a patient may go on for the greater part of a season. Tuberculosis is usually suspected, and no doubt it is frequently the explanation of similar symptoms. But we see many cases which are not tuberculous; the von Pirquet test is negative and tubercle bacilli are not found in the sputum, but the influenza bacillus is often regularly found for months. The persistence of the organism in the lungs and smaller bronchi is exceeded only by that of the tubercle bacillus. Many of these cases recover slowly and recover completely so far as can be determined clinically. There are some, however, which go on to chronic interstitial pneumonia and a few which develop bronchiectasis.

Influenza may be accompanied by a paroxysmal cough which is hard to distinguish from pertussis. There is a mild degree of laryngotracheitis or tracheobronchitis with few constitutional symptoms. Such a cough we have seen continue for from four to six weeks with paroxysms so severe as to excite vomiting. We have observed it in families of children who had previously had pertussis. Bordet's bacillus could not be discovered in the sputum but the influenza bacillus was present. There was no lymphocytosis but only a moderate polymorphonuclear leucocytosis. We believe that many of the reported instances of second attacks of pertussis are of this nature.

The very virulent forms of influenza are not common. It is usually only on account of the pulmonary complications that the attacks are serious. Every now and then, however, one encounters the especially severe type. The early symptoms often are not grave and for two or three days the patient's condition may excite no apprehension, when there develops, often quite rapidly, a state of profound general septicemia with great prostration and a severe pneumonia; or there are seen convulsions, drowsiness and stupor, hyperesthesia and rigidity; in short the symptoms of an acute meningitis which in our experience has been invariably fatal. The blood cultures in these cases regularly show the presence of the influenza bacillus.

Suppuration in the large joints we have in a few instances seen in influenza, in which this organism was found in the pus in pure culture. Usually this occurs as a late symptom. We have in one case seen it as the first definite local symptom. A boy of eight months after five days of general febrile symptoms developed swelling of an elbow and ankle. When first seen one week later there was general prostration, and the in-

fluenza bacillus was grown from pus aspirated from both joints. The following day convulsions occurred and the cerebrospinal fluid was turbid and contained the same organism. It was also found in the blood culture. Death from meningitis occurred three days later and at autopsy the influenza bacillus was obtained from brain, lungs, and blood. This proved to be one of the most virulent strains of the influenza bacillus ever tested in the hospital laboratory.

The influenza bacillus is associated chiefly with inflammations of the lower respiratory tract; in which respect it closely resembles the pneumococcus. The two organisms are often associated in inflammations of the lungs and bronchi. It has also the same tendency as the pneumococcus when in virulent form to excite a general septicemia, cerebrospinal meningitis and occasionally joint suppuration. It differs from it in being much less frequently associated with inflammations of the upper respiratory tract, and in occurring almost solely in the cold season, while pneumococcus infections prevail throughout the entire year.

Gastro-intestinal symptoms associated with the influenza bacillus we have not seen other than those that may occur with any form of acute febrile illness.

Complications and Sequelae.—The most frequent complications are bronchitis, pneumonia, otitis, and adenitis. In most of the cases with high temperature the urine contains albumin, and acute nephritis is occasionally seen. We have seen three cases of hemorrhagic nephritis in a single season. All recovered promptly. In one case the influenza bacillus was obtained from the urine by culture. One of the most frequent sequelae is anemia; this may be severe. Following the inflammation of the mucous membranes, there may be chronic enlargement of the cervical lymph glands. Attacks of influenza bear the same relation to the development of tuberculosis as do those of measles.

Convalescence after influenza is often very slow, and it may be months before the full effects of a severe attack have disappeared. For a long time the mucous membranes are in an extremely sensitive condition. Relapses are often brought about by slight exposure before the symptoms have quite disappeared.

Diagnosis.—The ordinary head colds even when severe and epidemic are very rarely due to influenza infection. The features which distinguish influenza infections of the respiratory tract from those due to other causes are, the peculiar range of temperature, the tendency to chronicity, to relapses, and to recurrences. A very high and widely-fluctuating temperature accompanied by few constitutional symptoms in the winter season is always suggestive. Influenza can be diagnosticated with certainty only by cultures which should be made upon blood agar. These should be

made from the bronchial secretion which is obtained as in cases of tuberculosis (q. v.). Cultures from the pharyngeal secretion are not to be depended upon. It is somewhat difficult to obtain the organism from the bronchial secretion and repeated examinations are usually necessary. In some typical cases we have been unable to find it at all during life, though it was found in the lungs at autopsy. Influenza may be confounded with malaria or cerebrospinal meningitis; from both of these it is distinguished by the methods of diagnosis used to identify these diseases. In the absence of cultures the diagnosis in many cases must be made by exclusion.

Prognosis.—Uncomplicated cases are seldom fatal, even in infants. Though the temperature is very high, recovery may be predicted as long as there is no evidence of important complications. The prognosis of the pneumonia of influenza is rather worse than that of simple bronchopneumonia. In a word, influenza is serious when there are pulmonary complications, but rarely otherwise, except in its virulent form, which, however, is infrequent. In this, general blood infection and meningitis are likely to occur.

Treatment.—The communicability of the disease makes it desirable that cases of influenza should be isolated whenever practicable, and particularly that delicate children, or those prone to pulmonary disease, should not be exposed. As there is no specific for influenza, the treatment is symptomatic and conducted along the same general lines as in other respiratory infections. The temperature rarely calls for antipyretic measures; for, although very high at times, there is very rarely a sustained high temperature. In our experience patients with influenza infections are not benefited by very cold air, but on the contrary are not infrequently made worse by it. Fresh air is, however, indispensable in the treatment of these cases, but at a moderate temperature, i. e., 60° to 65° F. The cough which so often persists after influenza is best controlled by cod-liver oil and creosote, used as after acute bronchitis. With persistent bronchitis which resists ordinary remedies, a patient should be sent to a warm, dry climate.

The complications of influenza are to be treated as they arise, in the same manner as when they occur under other conditions. Especial care should be exercised to avoid exposure during convalescence. One should be particularly anxious about patients who have a strong tendency to tuberculosis, and such cases should be watched with the greatest care. In prolonged or constantly recurring attacks nothing is of much avail except a removal to a warm climate. If this is impossible, a young or delicate child should be kept indoors during the cold season, but frequently moved from one apartment to another.

EPIDEMIC CATARRH—LA GRIPPE

To this disease the term Influenza has often been given. With our present knowledge it seems to us best to restrict the latter term to the disease or diseases just described with which Pfeiffer's bacillus is associated, instead of using it as a general name to cover contagious epidemic catarrh. Pfeiffer's bacillus was originally put forward as the cause of epidemic catarrh. Studies of the last few years have made this extremely doubtful. It is evident, however, that it is found in a certain proportion of cases; but it seems to play the rôle rather of an associated organism, exactly as, under the same circumstances, do the pneumococcus and the staphylococcus. But all these organisms are frequently found when no epidemic exists. The final solution of this question must wait on the discovery of the actual cause of the grippe. Meanwhile, there are many important reasons for believing that Pfeiffer's bacillus is not its cause:

(1) The highly contagious character of the disease, in which respect it is comparable to measles. The disease due to Pfeiffer's bacillus is only moderately communicable. (2) When the grippe is prevailing epidemically, Pfeiffer's bacillus is found in only a small proportion of the cases; and, *per contra,* Pfeiffer's bacillus is often found in groups of cases when the grippe is not prevalent. (3) The most striking clinical symptom of the disease induced by Pfeiffer's bacillus is a very high temperature without other general or local symptoms of corresponding severity; while in the grippe exactly the opposite is often the case, i. e., severe general symptoms with only a moderate elevation of temperature. (4) The intense general prostration, especially symptoms relating to the heart and nervous system, so common in the grippe, are not found in the disease due to Pfeiffer's bacillus, except in those rare cases of bacteriemia and meningitis. (5) Although a prolonged convalescence due to general prostration is not uncommon, the grippe is usually a short acute infection with little tendency to become protracted as are the inflammations due to Pfeiffer's bacillus. (6) With either form of infection any part of the respiratory tract may be involved in inflammation; but it is characteristic of the grippe that it is so often complicated by inflammations of the upper respiratory tract—rhinopharyngitis with extension to the adjacent sinuses, otitis, mastoiditis, adenitis, etc.—complications which are relatively infrequent with infections due to Pfeiffer's bacillus, whose complications are rather those of the lower respiratory tract—bronchitis and bronchopneumonia.

Clinically the grippe is manifested in children as in adults by two main groups of symptoms. In one there are quite marked symptoms of general prostration, chilly sensations, general aching pains in the muscles and sometimes in the joints, with only a moderate elevation of tempera-

ture—101° to 103° F. A few respiratory symptoms are usually present, but in most cases there is only a moderate cough and perhaps coarse râles in the chest. In infants and young children gastro-intestinal symptoms are frequently seen accompanying these symptoms. There may be vomiting or acute diarrhea or marked indigestion with quite a prolonged loss in weight without either vomiting or diarrhea.

In the second group of cases the respiratory symptoms are especially pronounced. In many these are only of the upper respiratory tract; there is a severe inflammation of the rhinopharynx, with sneezing, copious discharge from nose and eyes, followed by the development of hoarseness and cough. The inflammation does not extend beyond the trachea or possibly the larger bronchi. The chief complications of these cases are adenitis, otitis frequently followed by mastoiditis, extension from the nose to the neighboring sinuses, etc. These cases seldom have high temperature except when complicated. In others the temperature is higher and acute bronchitis or bronchopneumonia develops early. Although at the onset the pneumonia often seems particularly severe, it is not infrequently of short duration, resolution taking place before complete consolidation of the lungs has occurred. In other cases the type of pneumonia is of special severity, spreads rapidly, usually with a fatal outcome.

The treatment of the grippe is the treatment of its special symptoms and complications, which should be managed along the same general lines as when these occur under other conditions.

CHAPTER XIII

MALARIA

Malaria is an infectious disease due to the presence in the blood of a specific organism often called the *plasmodium,* but more exactly the *hematocytozoön malariae.* It manifests itself in children by the ordinary acute febrile attacks which are seen in adults and by chronic malarial poisoning. Both of these forms may present certain peculiar symptoms dependent upon the age of the patient.

Etiology.—The malarial organism was discovered by Laveran in 1881; it is a parasite of the blood and belongs to the group of protozoa. It is now well established that the parasite enters the blood through the bite of certain forms of mosquito, those belonging to the genus *Anopheles,* and probably in no other way. For this knowledge we are indebted chiefly to the work of Ronald Ross, in India, in 1897. For a general

discussion of the malarial parasite, its methods of staining, etc., the reader is referred to works on clinical medicine.

Malaria affects all ages, even the newly-born infant. We must accept with some allowance the statements made by the older writers upon the subject of intra-uterine infection, but in the following case reported by Crandall, there seems little doubt that the disease was contracted *in utero*: For ten days before delivery the mother had suffered from a tertian intermittent of moderate severity. Eighteen hours after birth the child was noticed to have cold hands and feet, blue lips and nails, and a pinched face. These symptoms lasted about half an hour and were followed by a distinct fever. Upon the following day the paroxysm was repeated. Examination of the blood of the mother and the child revealed the malarial organisms in both cases.

Malaria is more frequently overlooked in young children than in later life, from the fact that its forms are more irregular, and this has led to the belief that young children are less liable than adults to the disease. We believe, however, the opposite to be the case. In a large number of instances where families have been exposed to malarial poisoning we have noted that the young children were frequently the first to show the symptoms of the disease.

Malaria is an endemic disease prevailing in certain localities. Exact knowledge regarding the mode of infection has cleared up many obscure points in its etiology. The rôle of the mosquito explains the greater liability to contract malaria after sunset and during the night, the danger from stagnant ponds and pools of water, the peculiar susceptibility of infants and young children, and the greater frequency of the disease in the spring and summer. Malarial attacks may, however, occur at any season, since the organism may be latent in the body for an indefinite time; how long it is impossible to say, but there seems to be conclusive proof that it may be for many months. Attacks of malaria very often occur when the general health has been reduced by some other cause, particularly by disturbances of digestion.

Lesions.—Opportunities for a study of the peculiarities of the lesions of malaria in children are infrequent, especially in New York, as fatal cases are extremely rare. We have seen but two. As observed by others, the lesions do not differ in any marked way from those of the adult form of the disease. The most important changes are the destruction of the red corpuscles of the blood, enlargement, and in chronic cases hyperplasia with pigmentation of the spleen; less frequently pigmentation of the liver, kidneys, and brain. Pneumonia and gastro-enteritis are occasional complications.

Symptoms.—The clinical forms of malarial fever in children from six to ten years old, do not differ essentially from the same disease in adults.

Both tertian (Fig. 199) and estivo-autumnal (Fig. 200) attacks occur with considerable frequency, the former being the type most often seen. Double tertian infection (Fig. 198) is not uncommon but along the middle Atlantic coast the quartan type, unless imported, is unknown. The stages of the paroxysm are generally well marked. The cold stage begins with a chill or vomiting, with headache, lassitude, and general

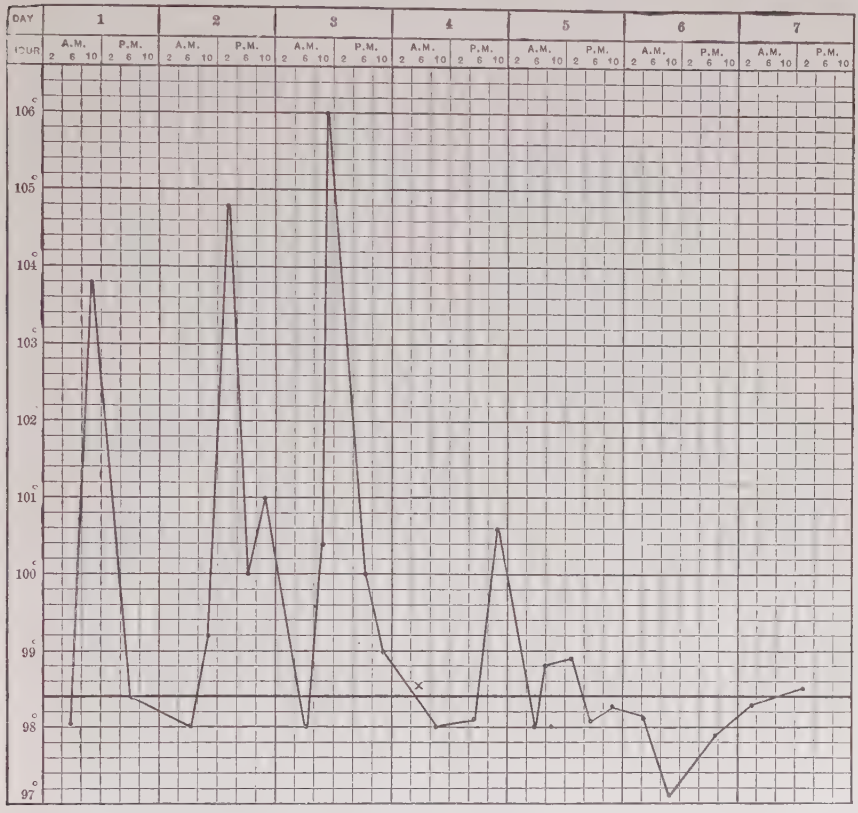


FIG. 198.—TYPICAL MALARIAL TEMPERATURE, DOUBLE TERTIAN TYPE, IN A BOY SIX YEARS OLD. Each paroxysm preceded by a chill. It will be noticed that the temperature rose higher with each succeeding paroxysm; × marks the time when quinin was begun.

pains. The hot stage is usually characterized by a higher temperature than in adults, and this is followed by the sweating stage, which is generally marked. The paroxysm may be repeated every other day or every day, depending upon whether there is a single or double tertian infection, until controlled by quinin. Less frequently there is an estivo-autumnal infection and the fever is remittent from the beginning and the constitutional symptoms are of greater severity. In this form there is

marked prostration, the tongue is thickly coated, there are often tenderness and pain in the region of the liver, and occasionally there is slight jaundice.

In infants and very young children peculiar types of malaria are seen. A well-marked intermittent fever with distinct stages is often absent, many cases assuming more of a remittent type or an irregular

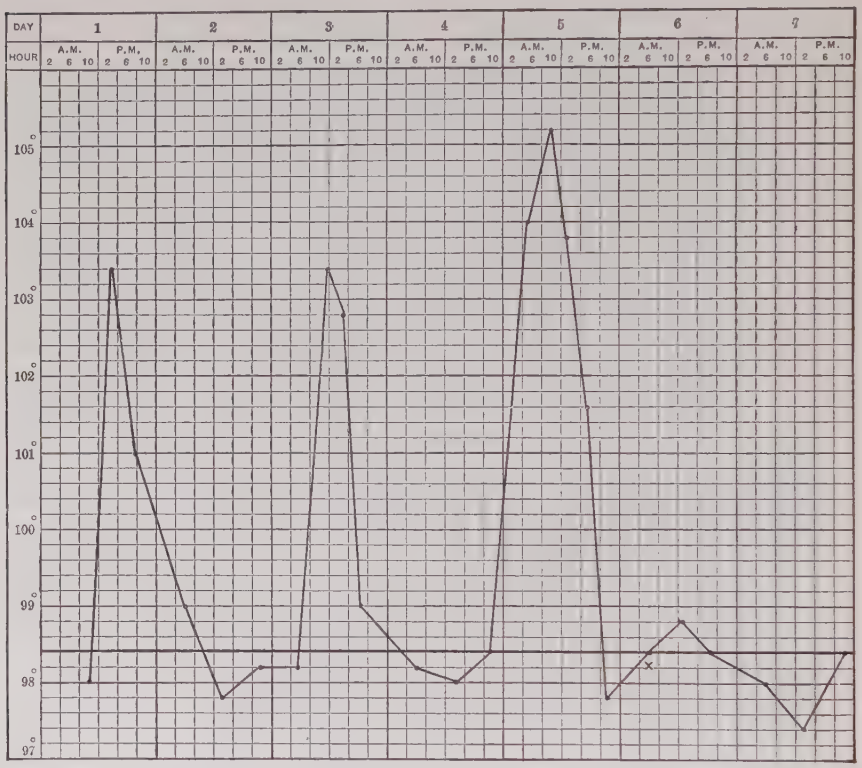


FIG. 199.—TYPICAL MALARIAL TEMPERATURE, TERTIAN TYPE, IN A BOY FIVE YEARS OLD. Onset with vomiting and drowsiness, but no chill. This was an anticipating tertian, the first paroxysm occurring at 3 P.M., the second at 12 M., the third at 10 A.M.; × marks the time when quinin was begun.

form of intermittent (Fig. 200). The onset is usually abrupt with vomiting, a well-marked chill being rare. Malarial chills are not often witnessed in children under five years old. They are replaced in infants by cold hands and feet, blue lips and nails, sometimes slight general cyanosis, pallor, drowsiness, and prostration. Vomiting has been present in two-thirds of our own cases. Several times we have seen a malarial attack ushered in by convulsions.

The fever is relatively higher than in adults, rising rapidly to 104° or 105° F., occasionally to 106° or 106.5° F. This continues from four

to twelve hours and gradually falls, usually to normal. The other constitutional symptoms of the febrile stage are much less severe than in most diseases with the same elevation of temperature. The sweating stage is only slightly marked and is often absent altogether. With the fall in the temperature there is a gradual subsidence of all the other symptoms of the febrile stage.

After the first paroxysm the patient may be quite well for several

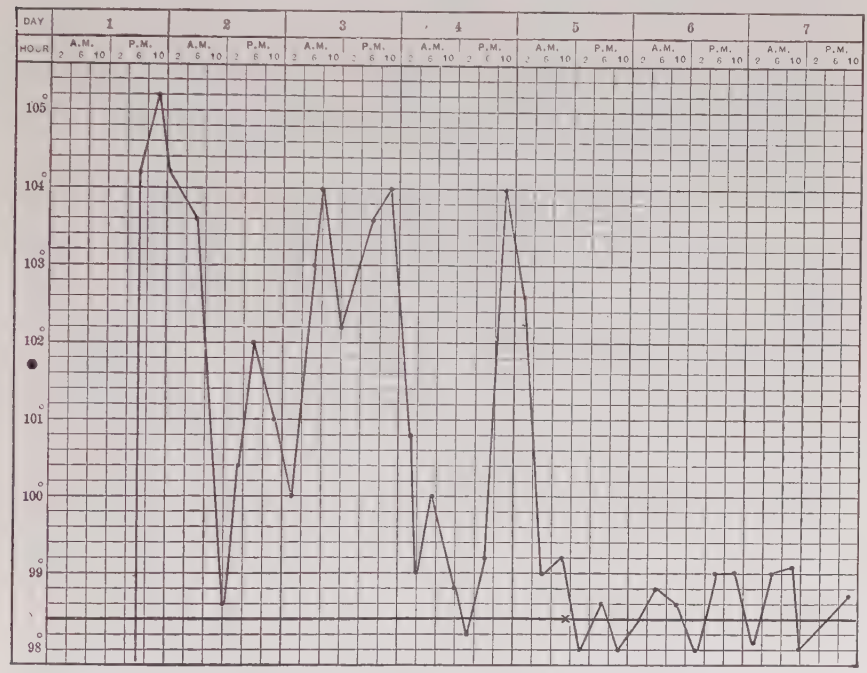


FIG. 200.—AN IRREGULAR MALARIAL TEMPERATURE (DUE TO ESTIVO-AUTUMNAL INFECTION) IN A CHILD NINE MONTHS OLD. The paroxysm on the fourth day was accompanied by an attack of acute pulmonary congestion which came near being fatal; × marks the time when quinin was begun. Although the course of the temperature is irregular, it touched the normal line both on the second and fourth days.

hours or even for a day, when the second paroxysm occurs. This is generally not so well marked as the first one, the third may be even less so, and the case may resemble more and more one of continuous fever with wide oscillations in the temperature. In some cases it is remittent at first and later becomes intermittent, but it is very rare in any circumstances that the temperature does not touch the normal point at some time in the twenty-four hours.

Enlargement of the spleen is present in the great majority of cases, and usually to a sufficient degree to be readily appreciated by examination. The most satisfactory method of examination is by palpation.

A spleen which can be easily felt below the ribs (except in the rare cases in which the organ is displaced downward by some condition in the thorax) is enlarged. When it is not sufficiently enlarged to be readily felt by a practiced observer under favorable conditions for examination, it is not large enough to be of any diagnostic importance. None of the other symptoms occurring in malarial fever are characteristic; they are quite similar to those which are seen in almost all febrile attacks. They are anorexia, coated tongue, constipation, and restlessness.

Masked or Irregular Forms of Malaria.—These are quite frequent in young children, and are due to the presence of certain special or uncommon symptoms which may readily lead to a mistake in diagnosis. They are more often seen than cases of true malarial cachexia.

Among the most frequent of the irregular forms are those relating to the nervous system. Headache is exceedingly common and is usually frontal. When severe and associated with continuous drowsiness, vomiting, and constipation, it may lead to a strong suspicion of tuberculous meningitis. Vertigo is not a frequent symptom, but it is sometimes very prominent. Pains in various parts of the body are very common. A sharp, severe pain at the epigastrium is frequent at the beginning of a paroxysm. It is often associated with tenderness, but has no relation to meals. Less frequently, pain is localized in the region of the spleen or liver. Aching or dragging pains in the muscles of the lower extremities are frequent symptoms during acute attacks, but may be of short duration, disappearing with the fever. The pain is accompanied by tenderness of the muscles and nerve trunks, and by loss of power, which is usually partial.

Accompanying the paroxysm of malaria there is occasionally seen, more often in infants than in older children, acute pulmonary congestion (Fig. 200), which may give rise to obscure and often very alarming symptoms. There is an acute onset with vomiting and prostration, high temperature, cough, rapid respiration, and often slight cyanosis. On examination of the chest there is found feeble or rude respiration over one lung, or over both lungs behind, and sometimes coarse moist râles: these signs and symptoms may disappear in the course of a few hours with the fall in temperature, to return with the next paroxysm, or if quinin is given they may disappear entirely.[1] This group of symptoms

[1] The following case is a good example of this condition in its more severe form, and illustrates the difficulties in the diagnosis of malaria in infancy: A fairly nourished child, nine months old, who had been under observation in an institution for two weeks, was suddenly taken with vomiting and fever (Fig. 200). A cathartic was followed by a large undigested stool, and as the temperature then fell to normal, the attack was regarded as one of indigestion. On

has sometimes led to the mistaken opinion that the disease was pneumonia, which had been aborted by the administration of quinin.

Subacute or Chronic Forms of Malaria.—The most constant symptoms are anemia, enlargement of the spleen, and slight fever. The anemia is usually marked, often being extreme. The enlargement of the spleen is distinct, easily made out by palpation, and sometimes is very great. The fever is often so slight as to be discovered only when the temperature is taken five or six times in the twenty-four hours. The other symptoms are of a very indefinite character; there may be slight edema of the lower extremities, general muscular weakness, so that the child is easily fatigued, loss of appetite, coated tongue, constipation, headache, muscular pains, and often cough from a slight bronchitis. These symptoms may depend upon many conditions other than malaria, even when they are seen in a malarial district. The only positive evidence of malaria in such cases is the presence of the malarial organisms in the blood. Even the swollen spleen, anemia, and slight fever, which are often looked upon as diagnostic, may be present in cases of anemia with which malaria has nothing whatever to do.

Diagnosis.—The positive diagnosis of malaria rests upon the demonstration of the malarial organisms in the blood. They will be found in nearly all the cases provided a careful examination is made a few hours before the paroxysm, and also that no quinin has been administered. When their number is small they may be missed at the height of the fever, although they may readily be found just before the temperature begins to rise. While a positive result is conclusive, a negative one is not always so because of the impossibility of fulfilling all the above conditions. This fact and lack of experience in blood examinations make it necessary for a large part of the profession to make the diagnosis by the

---

the third day the temperature was again high and accompanied by cough; coarse râles were found throughout the chest, and fine râles at the right base; it was then thought that pneumonia was developing. On the fourth day all the symptoms were so much improved that the infant was regarded as convalescent. At 6 P.M. the temperature was normal, and the infant went to sleep quietly. At 9.30 P.M. he awoke with a temperature of 104° F., extreme restlessness, and marked dyspnea. In half an hour his symptoms had increased to a point where he seemed likely to die. He became cyanotic, the respirations were of a panting character and rose nearly to one hundred a minute, and he coughed with almost every breath; the pulse was scarcely perceptible. The severe symptoms continued for about an hour, then passed away gradually, and at the end of two and a half hours they had completely disappeared, and the child was in a quiet sleep which continued until morning. Malaria was now suspected, and the diagnosis established by the discovery of the organisms in the blood. The spleen was at this time much enlarged; the signs in the chest were those only of bronchitis of the large tubes. Quinin was given in full doses, and immediately controlled the temperature and the pulmonary symptoms.

other symptoms. These, in the order of their importance, we would place as follows: Prompt curability (especially in cases of fever) by quinin; distinct periodicity in the symptoms; enlargement of the spleen; and a history of an exposure in a district known to be malarial. Particular importance is to be attached to the therapeutic test. Recent experience emphasizes more and more strongly the fact that quinin has very little influence upon fevers which are not malarial, and, conversely, that a fever immediately and permanently controlled by quinin is pretty certain to be malarial.

The fever and recurring chills of pyelitis are often attributed to malaria. Many conditions accompanied by an enlarged spleen may be confounded with malaria, especially simple anemia, leukemia, rickets, and syphilis. While malaria may be multiform in its manifestations, the physician can fall into no more serious error, even in a malarial district, than to regard all ailments with obscure or indefinite symptoms as malarial, neglecting careful physical and blood examinations, by which means alone an accurate diagnosis is reached.

Prognosis.—Although it is seldom fatal in itself, an attack of malaria in a young child may so undermine his constitution that he may succumb to some other acute disease. Cases are often difficult to cure while the patient remains in the malarial district, and when frequent re-infection occurs. In other circumstances and with proper treatment the prognosis of malaria is good.

Treatment.—*Prophylaxis.*—More exact knowledge regarding the etiology of malaria makes it possible for much to be done in the way of prevention. Besides the general measures proposed for the extermination of the mosquitoes concerned, emphasis should be laid upon the necessity, in the case of young children, of protecting them against the bites of mosquitoes in localities which are or which may possibly be malarial. This can be done by a more thorough use of mosquito netting and by using upon exposed parts of the body lotions or ointments containing menthol, pennyroyal, turpentine, or other substances which keep these pests away. The general treatment is symptomatic, and is to be conducted as in all acute febrile diseases. In the cold stage, stimulants or a hot bath may be required; in the hot stage, ice to the head and frequent sponging.

Methods of Administration of Quinin.—For infants our own preference is to give the sulphate in an aqueous solution, two or five grains to the teaspoonful, according to the age of the patient. Most infants take such a solution with less difficulty and vomit it less frequently than the combinations with the various vehicles supposed to cover its taste. If the quinin is given at night upon an empty stomach, vomiting seldom occurs. If repeated vomiting makes it impossible to give quinin by

mouth it may be given hypodermically. For this purpose the bimuriate of quinin and urea is perhaps the most satisfactory preparation; but the bisulphate may be used. Both are more or less irritating and there usually follows some induration at the site of the injection, which may last a long time. While the hypodermic use of quinin is sometimes invaluable it should not be employed in infants except in serious attacks and when the diagnosis has been established. The frequent repetition of the hypodermic injections should be avoided; in most cases, two or three good doses are sufficient, the effect being continued by quinin given by other methods.

For children from two to seven years old the taste of quinin must be concealed. An aqueous solution of the bisulphate may be mixed with the syrup of sarsaparilla, orange, or yerba santa; or the sulphate may be given in suspension in one of the same vehicles, the mixture being made just before the dose is taken; otherwise the partial solution of the drug will render the whole dose exceedingly bitter. When the dose required is not large, as in the milder cases, the lozenges of the tannate of quinin combined with chocolate answer the purpose admirably, for these are so nearly tasteless that children will take them without difficulty. Each lozenge usually contains one grain of the tannate, which is equivalent to about one-third of a grain of the sulphate of quinin. A similar lozenge containing one grain of the sulphate may be made, which is often taken by children without the slightest objection.

For children over seven years old, the same methods of administration may usually be employed as in adults. It is always preferable to give quinin in solution, or if not so, in capsule, but not in pill form.

In a case with well-marked paroxysms the quinin should if possible be given in the interval, with the largest dose about four hours before the expected paroxysm. With infants this plan is sometimes impracticable, as frequent small doses are usually better borne by the stomach than a few large ones. In them also vomiting seems less likely to occur when it is given on an empty stomach. For this reason it is advantageous to give the drug at regular two- or three-hour intervals during the night, and omit all medication during the day.

Dosage.—Relatively much larger doses of quinin are required for young children than for adults. Except for its tendency to disturb the stomach, quinin is borne remarkably well by little patients. Generally too small doses are given. An infant of a year with a sharp attack of malarial fever will usually require from eight to twelve grains of the sulphate (ten to fourteen grains of the bisulphate) daily. Occasionally we have found it necessary to give double the quantity referred to. It is useless to expect to control an acute attack of malaria by such doses as one grain three or four times a day. Children from five to ten years

old require almost as large doses as do adults. None of the substitutes for quinin are to be relied upon in acute cases.

In chronic cases, arsenic and iron are usually required in combination with smaller doses of the quinin than those mentioned. For children over seven years old, Warburg's tincture may be employed. In most chronic cases a cure can be effected only by a change of climate.

The masked and irregular manifestations of malaria are to be treated in the same manner as cases of malarial fever.

SECTION X

OTHER GENERAL DISEASES

CHAPTER I

RHEUMATISM

Rheumatism manifests itself in children by quite a different group of symptoms from those seen in adults; for this reason the disease was formerly supposed to be a rare one in early life. It is only within recent years that its frequency and its peculiarities have come to be appreciated. For our present understanding of the subject we are indebted largely to the work of English physicians, especially Cheadle, who has brought out more fully than any one else the close connection existing between many conditions formerly not regarded as rheumatic. One who has in mind only the adult types of articular rheumatism, and regards arthritis as a necessary symptom for a diagnosis, will overlook in early life many manifestations which are clearly the result of the rheumatic poison. There is seen at this period a group of clinical phenomena, which often occur in combination or in succession, whose association was not understood until they were all discovered to be related to rheumatism. Sometimes one member of the group and sometimes another is first seen, but when one has appeared others are likely soon to follow.

Rheumatism in childhood, then, is manifested not alone by arthritis with acute or subacute symptoms, but by a large number of other conditions which are not to be regarded in the light of complications, but rather as forms of the disease.

Etiology.—It is not in the province of this work to discuss the various theories regarding the nature of rheumatism and its exciting cause. The drift of medical opinion to-day is strongly toward the view that acute rheumatism is an infectious disease, probably of microbic origin. Although the character of the microörganism is not yet satisfactorily determined, the observations of Poynton and Paine, Wassermann and others point to a diplococcus. Under five years of age articular rheumatism is not common, and in infancy it is extremely rare. We once saw, however, in a nursing infant, a typical attack of rheumatic fever with

multiple joint lesions. The condition is, however, so exceptional that one should be cautious in making the diagnosis of rheumatism in infancy. Most of the cases so regarded are examples of scurvy. After the fifth year both the articular and the other manifestations of rheumatism become very common, and occur with increasing frequency up to the time of puberty.

Heredity is a very important etiological factor, and in fully two-thirds of the cases that have come under our care, a rheumatic family history was obtained. Of the other important causes, the most frequent are living in damp dwellings, direct exposure to cold and wet, poor hygienic surroundings, and insufficient food. While seen among all classes, rheumatism is more common among those who are badly housed. Attacks of rheumatism occur at all seasons, but are much more frequent in the spring months. One attack strongly predisposes to a second, and in most cases there is a history of a large number of attacks of greater or less severity. Among our own patients, girls have been affected with greater frequency than boys.

Symptoms.—*The General and Articular Manifestations.*—The clinical types of rheumatism in children present very notable contrasts to those seen in adults. A typical attack of acute articular rheumatism such as is seen in adult life, with a sudden onset, high temperature, severe inflammation of several joints, profuse acid perspiration, and occasional delirium, is rarely seen in a child under eight or ten years old. In most of the attacks in childhood the onset is not very acute, the temperature is but slightly elevated—only 100° or 101.5° F.—the swelling and pain are moderate, and the redness is often absent. The number of joints involved is generally small, those most frequently affected being the ankles, the knees, the small joints of the foot, the wrists, or the elbows. These symptoms are often not severe enough to keep the patient in bed, and only the pain in the joints of the lower extremities prevents him from walking. The duration of these attacks is from one to three weeks, and in the course of a month most of them recover even without treatment.

Not infrequently the symptoms are limited to a single joint, usually the hip, knee, or ankle. Possibly the joints of the upper extremity are affected oftener than would appear, but disease here is much more likely to be overlooked than when lameness is present. The swelling is moderate and may not be evident except on a close examination; in some cases there is none. There is stiffness of the joint, as shown by lameness, and there may be so much pain and soreness that the child refuses to walk altogether. Muscular spasm about the affected joint is often marked, and may be the most striking objective symptom. The tenderness is sometimes localized, but it may affect the ligaments, tendons, and even

the muscles. These symptoms may persist for two or three weeks and lead to a suspicion of incipient tuberculous disease of the joint. Rheumatism is distinguished by its more acute onset and usually by the presence of slight fever; some elevation of temperature being the rule, though it is not often much over 100° F. A family history of rheumatism, or a history of previous similar attacks in the patient affecting the same or other joints, or other manifestations of rheumatism, are also of assistance in the diagnosis. Occasionally all doubt is removed by the disease extending to other joints, or by the development of endocarditis. In some cases the symptoms are less in the joints themselves than in the muscles, and they are frequently dismissed as simply "growing pains," having nothing characteristic about them except their occurrence in damp weather.

Cardiac Manifestations.—These may occur when the articular symptoms are very mild, and in some cases when they are entirely absent. The most frequent is endocarditis. This is much more often seen in the acute rheumatism of children than of adults, and probably occurs in the majority of all severe cases; if it does not come in the first attack, it is likely to be seen in the later ones. It frequently occurs with a mild rheumatic arthritis, often being unnoticed until valvular disease of considerable severity has developed. Sometimes there is only high fever with severe constitutional symptoms of an indefinite character, but no arthritis, and no suspicion that the attack is rheumatic until endocarditis is discovered. Such cases are not infrequent. If the patients are kept under observation, articular symptoms are almost certain to develop later, and often there are other manifestations of rheumatism, especially chorea.

Pericarditis is much less frequent than endocarditis, and usually occurs in children over seven years old. It is often associated with endocarditis. The most characteristic form of inflammation in early life is a sub-acute, dry, fibrous form, often resulting in great thickening with extensive adhesions, and frequently in obliteration of the pericardial sac. When once started it shows a strong tendency to recurrence and persistence.

The heart is so frequently affected in the rheumatism of childhood that it should be closely watched whenever articular symptoms are present, no matter how mild they may be; and not only in these cases, but in all the conditions hereafter enumerated with which rheumatism is likely to be associated.

Inflammations of other serous membranes—the pleura, peritoneum, and pia mater—were much more frequently ascribed to rheumatism in the past than now. There is reason for believing that on rare occasions the pleura may be involved, but very exceptionally in young children.

There is no evidence that the peritoneum and meninges are directly affected by rheumatism.

Torticollis when it occurs acutely is frequently rheumatic. This form is characterized by its sudden development, continuous spasm, the great amount of muscular soreness, the moderate pain, and the fact that it usually disappears spontaneously after a few days. Other manifestations of muscular rheumatism are less characteristic and usually affect the muscles of the extremities.

Anemia is almost invariably seen in rheumatic patients, both during and between the attacks. The effect of the rheumatic poison upon the blood resembles that of malaria. A secondary anemia develops, often of considerable severity.

Chorea.—In the chapter upon Chorea we have already discussed the association of that disease with rheumatism and expressed our belief in a very close relationship existing between them. Not infrequently chorea is the first manifestation of a rheumatic diathesis, to be followed soon by articular symptoms or by endocarditis without such symptoms. In other cases chorea and acute endocarditis occur together without articular symptoms, or all three may be associated. Whichever of the three conditions is first seen, the physician should always be on the lookout for the others. The frequency of rheumatism in choreic patients has been variously estimated by different observers; in our own cases over fifty per cent have given unmistakable evidence of a rheumatic diathesis.

Tonsillitis.—The association of tonsillitis and pharyngitis with rheumatism appears in many cases to be a close one. Children who are the subjects of frequent attacks should be regarded as probably rheumatic, and closely watched for other signs of that disease. Acute tonsillitis often ushers in an attack of rheumatic arthritis, and occasionally acute endocarditis without articular symptoms. The nature of the relationship is not yet fully explained; by many the tonsils are regarded as the structures in which the organisms of rheumatism first obtain a foothold.

Subcutaneous Tendinous Nodules.—General attention was first drawn to these as a manifestation of rheumatism by Barlow and Warner, in 1881, who described them as "oval, semi-transparent, fibrous bodies like boiled sago grains." They are most frequently found at the back of the elbow, over the malleoli, at the margin of the patella; occasionally on the extensor tendons of the hands, fingers, or toes, or over the spinous processes of the vertebrae or the scapulae. They are composed of fibrin, cells, and fibrous tissue, and vary in size from a large pin's head to a small bean, sometimes being as large as an almond. The nodules may come in crops, lasting for a few weeks and then disappear, or they may last for months. An eruption of nodules is usually coincident with other

rheumatic manifestations. These nodules are better felt than seen, although they may be visible if the skin is tightly drawn. They are certainly not common in this country; and although we have made it a rule to examine rheumatic patients for them, we have seen them but seldom, and they have been prominent in only eight or ten cases. This has also been the experience of most observers in this country. From published reports, however, they appear to be much more frequent in England. There can be no doubt regarding the connection of these nodules with rheumatism.

Erythema.—The connection between rheumatism and the various forms of erythema—marginatum, papulatum, and nodosum—has been very clearly shown by Cheadle. None of these is a frequent condition in childhood, but when seen it should always suggest rheumatism.

Purpura.—The association of purpura with rheumatism is at times so close that there can be little doubt of the close connection between the two conditions. Rheumatic purpura, however, is quite distinct from the other forms of purpura, and is a much less frequent disease.

Diagnosis.—In order to recognize rheumatism in a child, one must free his mind from preconceived notions of the disease drawn from its manifestations in adults, as very few cases correspond to the adult type of acute rheumatism. In early life the disease is recognized not by any one or two special symptoms, but by the association or combination of a number of conditions which may appear unrelated. In determining whether or not any given set of symptoms is due to rheumatism, one should consider: (1) the family history, since in early life heredity is so important an etiological factor; (2) the previous history of the patient, not only as regards articular pains and swellings, the slight joint-stiffness without swelling, the indefinite wandering pains in damp weather, and the so-called growing pains, but also the previous existence of chorea, frequent attacks of tonsillitis, torticollis, or erythema; (3) the examination of the patient, which should include a careful search for tendinous nodules, as well as a thorough examination of the heart for signs of endocarditis or pericarditis, and, in cases which are at all acute, the temperature. In doubtful cases with monarticular symptoms much importance is to be attached to the presence of slight fever, the abrupt onset, and tenderness of the neighboring muscles and tendons—all occurring without a history of traumatism. Rheumatism is more often overlooked than confounded with other diseases; although in childhood multiple neuritis and tuberculous and syphilitic bone disease are often mistaken for it, and in infancy the same is true of scurvy. The extreme infrequency of rheumatism during the first two years of life should always make one sceptical regarding it. In an infant, when the symptoms are confined to the legs and are not accompanied by fever, they

are almost certain to be due to scurvy, even though the gums are normal and ecchymoses have not appeared. Multiple gonococcus arthritis has often been diagnosticated rheumatism. Many cases of general sepsis, especially such as originate from the tonsils or the teeth, may be accompanied by joint swellings resembling rheumatism.

Prognosis.—Rheumatism in a child is in itself seldom if ever dangerous to life. In the great majority of cases the articular symptoms soon disappear, even without special treatment. The danger from the disease consists in its cardiac complications. One attack of rheumatism is almost certain to be followed by others, and when once the heart has been affected its lesions are likely to increase with each recurrence of the disease.

Treatment.—Rheumatism in children derives its chief importance from its relation to cardiac disease. Cardiac complications are so frequent and so serious that everything possible should be done to avert rheumatism from those who by inheritance are especially predisposed to it, to prevent its recurrence in a child who has once had the disease, and during an attack to prevent the heart from becoming involved. The relation of diet to rheumatism is very imperfectly understood. Our own opinion is that there is no close connection between the two. The underclothing should be of wool during the entire year, in summer the lightest weight being worn. The feet should be carefully protected, and exposure in damp weather avoided. Indoor occupations should be chosen for rheumatic boys.

The tendency to recurrence is so strong in this disease that a child of rheumatic antecedents, who has shown in the various ways mentioned a marked predisposition to rheumatism, and who has had an attack, even though a mild one, should, if possible, spend the winter and spring in some warm, dry climate, or even remain there permanently. Otherwise in most such children, it is only a question of time when, with the repeated attacks, the heart will become involved.

To avert the danger of cardiac complications during an attack of rheumatism, or to limit their extent, there are two things which should invariably be insisted on: first, to confine to the house and in a warm room every child with rheumatic pains, no matter how mild; secondly, if fever is also present, to keep the child in bed while it continues, even though it may not be above 100.5° F. Absolute rest and the equable temperature thus secured are unquestionably of more importance than anything else in protecting the heart during a rheumatic attack. With these precautions must be combined an early diagnosis. In very many, perhaps in most cases, the harm is done before the true nature of the disease is suspected, the symptoms being dismissed as of slight importance because the articular manifestations are not very severe. Children

who have once had rheumatism should be closely watched during chorea and other diseases related to rheumatism, the heart should be frequently examined, and the physician should be on the alert for the first articular symptoms.

Aside from the measures just mentioned, the treatment of rheumatism in childhood is to be conducted very much like that of adult life. In most acute attacks either salicylate of soda (gr. v every three hours to a child of five years), aspirin, oil of wintergreen, or salicin should be given; as the majority of cases are not very acute, marked improvement is by no means always obtained by these drugs. Alkalis should be given in all cases in combination with the salicylates, but particularly in those in which there is hyperacidity of the urine. Either the acetate or citrate of potassium or the bicarbonate of sodium may be used, a sufficient quantity being administered to render the urine alkaline.

Quite as necessary as these drugs is the use of general tonics, particularly iron and cod-liver oil. These should be given not only between attacks to fortify patients against their recurrence, but also in subacute cases which are sometimes influenced very little or not at all either by salicylates or alkalis.

The importance of attention to pathological conditions in the tonsils and mouth in all children with recurring rheumatic attacks should not be overlooked; especially should diseased tonsils be removed and carious teeth and diseased gums receive appropriate treatment.

CHAPTER II

DIABETES MELLITUS

In this chapter will be attempted only a description of the peculiar features which diabetes presents when affecting young patients. It is a rather infrequent disease in children. Of 1,360 cases of diabetes collected by Pavy, only eight were under ten years of age. In a series of 700 cases collected by Prout, only one case was under ten years. In a series of 380 cases collected by Meyer, only one case was under ten years of age. More recent statistics have shown that the proportion of children under ten among diabetics is not so small as would be indicated by these figures. Von Noorden has reported 84 cases in children under ten in about 3,000 cases of diabetes. We have ourselves seen more than thirty cases.

Etiology.—Stern, in a series of 117 collected cases of diabetes in children, states that 47 were females and 31 males, the sex in the other

cases not being given. Of 26 of the cases observed by us, 16 were in females and 10 in males. It seems that females are rather more frequently affected, in contrast with the marked preponderance of cases in males in adult life. Although extremely rare, cases have been observed during the first year of life. Statistics on this point are not altogether trustworthy, since some cases of temporary glycosuria have certainly been included. The youngest case that has come under our observation was in a boy of twenty-six months.

Among the etiological factors heredity is one of the most important. Pavy reports the case of a child dying of diabetes at two years in whose family the disease had existed for three generations. Instances have been recorded of the occurrence of diabetes in four or five children of the same family. There was a family history of the disease in 11 out of 26 patients under our care. Several of the cases reported in children have been preceded by injuries received upon the head. In a number of our own cases the disease has followed the consumption of large quantities of sugar for a long time. Often no adequate cause can be found.

Symptoms.—The most important early symptoms are thirst, polyuria, and wasting; their development is often quite rapid. The thirst is intense, often leading children to drink four or five pints of fluid a day, or even more. The amount of urine passed varies from one to eight quarts daily. The specific gravity is from 1.026 to 1.040, and the amount of sugar usually large. Acetone, diacetic and β-oxybutyric acids are also present in greater or less amount. Albumin is not infrequently found. Incontinence of urine is an important symptom, and often one of the earliest to be noticed. The wasting is usually quite rapid, so that a child may lose as much as six or eight pounds in a month. It is generally accompanied by anemia. The appetite may be poor; at times, however, it is voracious. Other symptoms of less importance are a dry mouth, scanty perspiration, irregular sleep, occasional epistaxis, furuncles and abscesses, decayed teeth, and genital irritation.

The course of the disease is much more rapid in children than in adults, and, as a rule, the younger the child the more rapid its progress. Without proper treatment, the great majority of the cases prove fatal in from three to six months from the time the symptoms are sufficiently marked to make the diagnosis possible. Occasionally, however, one of the milder type may be prolonged from one to two years.

The progress of the disease is marked by continuous wasting, which may result in a striking degree of malnutrition and prove fatal. Some are carried off by intercurrent pneumonia or tuberculosis, but the majority die comatose. When coma develops, the case may be considered hopeless, and death is likely to be postponed but a few days. The cause

of diabetic coma has not been explained with entire satisfaction. It occurs when there has been a prolonged and severe drain upon the alkaline defenses of the body by the abnormal acids which are not, in themselves, directly poisonous. Acidosis is a regular accompaniment of coma. Whether it is the sole cause is at the present time not entirely clear.

Diagnosis.—Diabetes is apt to be overlooked, because of the common neglect of urinary examinations in children. The prominent symptoms,—thirst, polyuria, and wasting—when associated, should always attract attention. Enuresis, accompanied by marked wasting, is always suspicious. In some cases genital irritation may be the most prominent early symptom. A positive diagnosis is made only by an examination of the urine.

Prognosis.—In few diseases has the prognosis been so bad as in diabetes in children. Senator has declared that diabetes in childhood is hopeless and all treatment useless. Von Noorden has said that with rare exceptions diabetes of childhood allows no respite. Such has also been our experience. From the more recent methods of treatment, especially that recommended and elaborated by Allen, much more is to be expected. It has been sufficiently demonstrated that children can be maintained in a satisfactory condition, free from sugar and gaining gradually in weight for many months. We have now under observation five children who are doing well. Whether it will be possible for them to continue in this way and to reach adult life properly developed is a matter which only the future can decide. The outlook is, however, not so immediately dark as it has been. Intelligent observation and unremitting care are required both by the physician and parents. Without them good results are impossible.

Treatment.—The indications for treatment are the same in children as in adults. Nothing more can be indicated here than the principles to be followed. In diabetes the carbohydrate tolerance is always very greatly diminished but usually not entirely lost. The purpose is to increase this tolerance. It can only be accomplished by protecting the carbohydrate mechanism from overstrain. If the tolerance is exceeded and sugar is excreted in the urine, the carbohydrate mechanism becomes less and less capable and the tolerance sinks. By preventing sugar excretion the mechanism improves and the tolerance rises. Patients should therefore be rendered sugar free at the earliest possible moment and constantly maintained sugar-free. This may be accomplished by temporary starvation until no sugar appears in the urine. Nothing whatever by mouth should be allowed but clear broth and water. When there is no sugar excreted, well-cooked vegetables may be given, at first those containing but little carbohydrate, such as asparagus, spinach,

cabbage, onions and celery. After two or three days, nitrogenous foods, eggs, meat or fish may be allowed and later bacon, butter, olive oil and other fats. All of these are to be given in small amount at first and gradually increased until the nitrogenous and caloric needs of the body are satisfied. Loss of weight at first is to be expected and is not to be feared. Carbohydrate tolerance and not the weight curve is the index of progress. It is important that a record should be kept of the amount of carbohydrate taken and of the amount of sugar and acetone bodies excreted in the urine. Not more than 10 grams of carbohydrate a day should be given at first and any increase should be slowly made. Even if no sugar appears in the urine, it is advisable in severe cases to introduce a day of only broth feeding every ten days or two weeks, after which a low carbohydrate diet should be instituted and the carbohydrates again gradually increased. From time to time an attempt should be made to introduce articles of food such as oatmeal and milk in small quantities but never in amount sufficient to cause glycosuria. If sugar appears, a rapid and great reduction in the carbohydrates of the food is to be made and any increase should be instituted with caution, and not for several weeks should the amount be reached which was formerly followed by glycosuria.

CHAPTER III

PELLAGRA

Although it is only recently that pellagra has attracted much attention in this country, it is not likely that it has existed here for only a few years, but rather that it has not been recognized. At the present time its etiology is not understood. Three theories as to its cause have been advanced. The first and the one longest held is that it is due to the eating of spoiled corn (maize). In this, toxic products are supposed to be produced by the growth of fungi or of bacteria. The second is that it is a parasitic disease transmitted by the bite of an insect (the gnat). The third, and the view which is becoming more and more widely accepted, is that it is due to a diet deficient in certain important constituents (vitamins), which places it in the same group as scurvy and beriberi. The recent observations of Goldberger have shown that recurrences of the disease may be prevented by a reduction in the amount of carbohydrate food, and by considerable increase in vegetable and animal proteins, especially fresh milk, eggs, meat and leguminous vegetables. His observations indicate that pellagra may be produced by giving a diet which, though abundant, may consist chiefly of carbohydrates

and from which fresh animal and vegetable proteins have been excluded.

Pellagra is seen at all ages although it is comparatively rare in very young infants. After two years of age it is much more common. It is found with greatest frequency in the states of the South Atlantic Coast, although cases have been reported from almost every state in the Union and even from Canada. Pellagra is a disease preëminently of the warm months,—spring, summer and autumn. As soon as cool weather comes it usually diminishes much in severity and in frequency, but cases sometimes develop even during the winter. It is found chiefly among the poor living in unsanitary surroundings, but no class is entirely exempt. While it is found in cities as well as towns, it occurs more often in country districts.

There are no characteristic anatomical lesions in pellagra. Cellular change in the brain is common. In the cord degeneration of the lateral and posterior columns is frequently found, but usually only in cases that have existed for many months or years.

Symptoms.—The symptoms in a well-marked case are easy to recognize, but in the mild form the disease may be almost impossible to detect, and it may be a long time before a definite conclusion as to the diagnosis can be reached. There are three chief symptoms—the cutaneous lesions, the gastro-intestinal symptoms and those of the nervous system. The cutaneous or the gastro-intestinal symptoms are those first in evidence. The eruption is found chiefly on exposed surfaces and for this reason and because it often begins with the advent of warm weather, it is frequently mistaken for sunburn. The eruption begins as an erythema, but after a variable length of time exfoliation takes place, desquamation being in some cases very marked. The skin is thickened, rough and dry, although in exceptional circumstances vesicles and bullae may be found and ulceration even may take place. The eruption (Fig. 201) is found upon the hands, neck, face and feet, although it may spread far up the arms and legs and involve even portions of the trunk as well. It is strikingly symmetrical and the lesions are sharply outlined; when they are not so it usually indicates that the eruption is receding. There is a certain amount of brownish discoloration, its intensity depending somewhat upon the complexion of the person affected. No itching is complained of, but a slight burning or tingling sensation. The nails are unaffected. The tongue is oftentimes red; it may be coated, with clear edges, or it may be dry and glazed. The papillae are often somewhat enlarged. The tongue may be swollen. In addition to the glossitis there may be also stomatitis and gingivitis. Burning in the mouth is an occasional complaint.

The gastric symptoms are few. Vomiting is rare. Anorexia may be marked but at times there is a craving for unusual food. Diarrhea is

the rule. The stools are from two or three to as many as fifteen a day. They may be watery, but at times mucus and even blood are present. Prolonged constipation is rare, but the diarrhea often alternates with periods of constipation.

The mental symptoms are not so marked in children as in adults.

FIG. 201.—PELLAGRA. Boy, five years old; died of the disease five months later.

Depression is often present. There is frequently a change in disposition, the children becoming dull, morose and peevish. An anxious, distressed facial expression is characteristic of marked cases. The reflexes are usually exaggerated. Ankle clonus is frequently present and there may be a decided tremor upon exertion. If the intestinal symptoms are marked, there may be great loss of weight. The progress of the symptoms is not usually continuous, but there are marked remissions and exacerbations. The disease often disappears in the fall and winter to return

again the following spring and this may be repeated many times. It is for this reason difficult to say when the disease is really cured. The prognosis in children is better than that in adults but death may occur from a continuance of the diarrhea, from the development of marked malnutrition or from intercurrent infections.

Treatment.—No specific remedy for the disease has yet been discovered. The gastro-intestinal condition should be treated symptomatically. Pellagrous mothers should not nurse their infants. They should be artificially fed or a wet-nurse should be secured. In children beyond the nursing age the diet should be a mixed one, suited to the age of the child so far as the gastro-intestinal symptoms will allow. Following the suggestions derived from Goldberger's observations, careful attention should be given to the food. A faulty diet in which carbohydrates, especially corn meal, have been excessive should be replaced by one with an abundance of milk, eggs, fresh meat, peas and beans. The patient should be put in the best hygienic surroundings possible. Arsenic is believed to be of special value. It may be given by mouth in the form of Fowler's solution, but it is thought by many to be more effective when given hypodermatically. Sodium cacodylate may be used in doses of 1/12 to ¼ grain repeated two or three times at intervals of several days.

INDEX

Abdomen, examination of, 39; growth of, 25; in rickets, 254.
Abscess, alveolar, 273; cerebral, 759; cerebral, in acute otitis, 943; hepatic, 439; ischiorectal, 434; mammary, 117; multiple, in newly born, 86; peritoneal, 445; peritonsillar, 307; psoas, in spinal caries, 911; retro-esophageal, 312; retro-pharyngeal, in Pott's disease, 293, 908; subphrenic, 455.
Acid, hydrochloric, increased by lavage, 340; hydrochloric, in stomach digestion, 317; in chronic gastric digestion, 341.
Acidosis, 217, 365; treatment of, 372.
Adenitis, simple, acute, 862; simple, chronic, 865.
Adenoid vegetations of pharynx, 294; asthma from, 488; causing chronic nasal catarrh, 460; chronic laryngitis with, 472; in rickets, 255; with adenitis, 865.
Adenoma, of umbilicus, 114.
Agenesis, cortical, 779.
Airing, when allowed out of doors, 8.
Air-space required by infants, 10.
Alalia, 711.
Albumin water, preparation of, 164.
Albuminuria, orthostatic or cyclic, 617; in chronic cardiac disease, 598; in chronic nephritis, 637; in measles, 986; in scarlet fever, 966.
Alcohol, as stimulant, 54; as tonic, 55; effect of, on breast milk, 175; use of, in diet of nurse, 143.
Amaurotic family idiocy, 788.
Amebic colitis, 390.
Amyloid degeneration, 441; in chronic bone disease, 907; of the intestines, 391; of the liver, 391; of the spleen, 391.
Anemia, cardiac murmurs in, 607; pernicious, 846; pseudoleukemic, of infancy, 844; secondary, 841; treatment, 847.
Anesthetics, 66.
Aneurism, 612.
Antipyretic drugs, 53.
Antipyretics, 51.
Antitoxin, in tetanus, 92; eliminated by human milk, 143.
Anuria, 621.
Anus, fissure of the, 431; imperforate, 118.
Aorta, abnormal origin of, 582; aneurism of, 612; atheroma of, 612; hypoplasia of, 611; thrombosis of, 611.
Aortic insufficiency, 600; stenosis, 600.
Aphasia, functional, 711; in acquired cerebral paralysis, 786; after typhoid fever, 1064.
Appendicitis, 418; diagnosis of, 421; treatment of, 422.
Arm, paralysis of, at birth, 111.
Arsenic, as a tonic, 55; dosage of, in chorea, 699.
Arteries, hypogastric, in fetal circulation, 575; hypoplasia of, 611; umbilical, in fetal circulation, 575.
Arthritis, acute, of infants, 900; atrophic, 902; chronic, 902; gonococcus, 653, 659, 900; rheumatic, 1150.
Arthrogryposis (see TETANY), 677.
Artificial feeding, 179; versus wet-nursing, 168.
Ascaris lumbricoides (see WORMS, INTESTINAL), 425.
Ascites, 454; chylous, 455; in acute diffuse nephritis, 631; in cirrhosis of liver, 440; with chronic peritonitis, 448; with tuberculosis of the peritoneum, 450.
Asphyxia, death from, in young children, 46; from overlying, 48; from aspiration of food, 48; from enlarged thymus, 49; in convulsions, 675; in retropharyngeal abscess, 292; in the newly born, 69; from tuberculous bronchial lymph nodes, 1098; methods of resuscitation, 72; sudden, in retro-esophageal abscess, 313.
Aspiration, of chest, in empyema, 570.
Asthma, 487; with adenoids, 297; simulated by tuberculous bronchial glands, 1096; treatment, 491.
Ataxia, Friedreich's, 820; in multiple neuritis, 831.
Atelectasis, acquired, 553; in delicate infants, 554; causing sudden death, 49; congenital, 74.
Atheroma, 612.
Athetoid movements, 701; in acquired cerebral paralysis, 786; in birth paralysis, 783.
Athetosis, 701.
Atrophy, infantile (see MARASMUS), 227; muscular spinal, types of, 822; muscular neural, types of, 822.

Babcock's centrifugal machine, 149.
Bacillus, of diphtheria, 1020, 1041; distribution of, in the body, 1023; in milk, 146; in healthy throats, 1041; in laryngeal diphtheria, 1041; nonvirulent, 1041; of dysentery (Shiga), in ileocolitis, acute, 374; in gastrointestinal intoxication, acute, 374; of Eberth, in typhoid fever, 1058; Klebs-Loeffler (see B. DIPHTHERIE), 1020; lactis aerogenes, 319; of Pfeiffer, in influenza, 1130; pseudodiphtheria, 301; of tuberculosis, 1067; in acute bronchopneumonia, 495.
Backwardness, 792.
Bacterium coli communis, 319; in appendicitis, 419; in peritonitis, 445.
Bacterium lactis aerogenes, 319.
Balanitis, 653.
Band, abdominal, 1, 3.
Barley water, directions for making, 164; use of, during first year, 198.
Barlow's disease (see SCORBUTUS) 231.
Bath, at birth, 1, 2; cold, 53; in acute bronchopneumonia, 524; in asphyxia, of newly born, 72; evaporation, 53; hot, 59; hot air, 59; vapor, 56; mustard, 59; bran, 60; tepid, 60; shower, 60; cold sponge, 60; hot, in asphyxia of newly born, 72; in typhoid fever, 1067.
Bed-wetting, 662.
Beef, raw scraped, 163.
Beef broth, 163.
Beef extracts, 163.
Beef juice, 159.
Beef preparations, 159.
Belladonna, 56; elimination of, in milk, 143; scarlatiniform rash from, 970.
Bile, physiological action of, 318.
Bile ducts, congenital malformations of, 78.
Birth paralyses, 106.
Bladder, control of, acquired, 663; exstrophy of, 651; hemorrhage from, in newly born, 105; stone in, 667; training to control, 4.
Bleeders, 852.
Blindness, hysterical, 705; transient, in pertussis, 1010.
Blood, circulation of, in early life, 575; diseases of, 839; in chlorosis, 843; in leukemia, 849; in pernicious anemia, 846; in pseudoleukemic anemia, 844; in secondary anemia, 841; transfusion of, 68.
Blood vessels, diseases of, 611; aneurism, 612; coarctation of the arch of the aorta, 611.
Boil (see FURUNCULOSIS), 930.
Bones, diseases of, 895; in hereditary syphilis, 1115; in late syphilis, 1118; lesions of, in rickets, 244; microscopical changes of, in rickets, 245; syphilitic diseases of, 1118; tuberculous diseases of, 905.
Bothriocephalus latus, 424.
Bottles, nursing, choice and care of, 196.
Bow-legs, in rickets, 253.
Bradycardia, 609.
Brain, diseases of, 719; abscess of, 759; atrophy and sclerosis of, 780, 784; atrophy and sclerosis of, in acquired cerebral paralysis, 780; cysts of, in infantile cerebral paralysis, 781; malformations of, 719; tuberculosis of, 1080; tumor of, 762; weight of, 669.
Bran bath, 60.
Breast, abscess of, in newly born, 117.
Breast-feeding, 166; schedule for, 171.
Breast milk (see MILK, WOMAN'S).
Breath, offensive, in ulcerative stomatitis, 279.
Breathing, noisy, with adenoids, 296; stridulous, in diseases of the larynx, 463, 469, 473; in retro-esophageal abscess, 314.
Bright's disease (see NEPHRITIS), 629.
Bromids, elimination of, in milk, 143.
Bronchi, catarrhal spasm of, 489; diphtheria of, 1026; foreign bodies in, 475, lesions of, in acute bronchopneumonia, 498; lymph nodes of, in tuberculosis, 1073, 1078; tube casts from, 486.
Bronchial glands (see also LYMPH NODES, BRONCHIAL), 1095; enlarged, cause of asthma, 488; in acute bronchopneumonia, 509.
Bronchitis, acute catarrhal, 479; symptoms of, 480, 483; treatment of, 483, 485; capillary (see BRONCHOPNEUMONIA, ACUTE), 497, 506; attacks of asthma resembling, 489; chronic, 486; chronic, in rickets, 246; diphtheritic, bronchopneumonia in, 517; fibrinous, 485; treatment, 486; in pertussis, 1008; in typhoid fever, 1063; spasmodic (see ASTHMA), 489; tuberculous, 1087.
Bronchiectasis, in chronic bronchitis, 487; in bronchopneumonia, chronic, 547.
Bronchopneumonia, acute, 497; bacteriology of, 495, 496; complications in, 518; complicating influenza, 517; complicating diphtheria, 517; complicating measles, 517; complicating pertussis, 515; complicating rickets, 246; diagnosis of, 519; etiology of, 497; lesions in, 498; associated in the lung, 504; physical signs of, illustrated, 513; protracted or persistent form of, 515; secondary pneumonia with measles, 984; ileocolitis, associated with, 381; influenza, associated with, 1136; pertussis, associated with, 1008; diphtheria, associated with, 1036; prognosis of, 520; protracted cases of, 515; symptoms of, 505; temperature charts of, 510; termi-

nations of, 503; treatment of, 523; prophylaxis in, 523.
Bronchopneumonia, chronic, 540, 542.
Bronchopneumonia, tuberculous, 1086; rapid cases, 1087; protracted cases, 1088; (see also TUBERCULOUS PNEUMONIA), 1086, 1092.
Broths, directions for making, 163.
Buhl's disease, 93.
Buttermilk, 159, 207, 388.

Calamine lotion, 929.
Calculi, biliary, 443; renal, 646; pyelitis with, 647; vesical, 667.
Calories, required daily by healthy infants, 180; method of calculating, 181; value of different foodstuffs in, 129, 181.
Cancrum oris (see STOMATITIS GANGRENOUS), 285.
Carbohydrates, function of, in diet, 131.
Carcinoma, of brain, 762; of stomach, 345.
Casein, 150, 183.
Caseinogen, 150.
Casts, in urine, of chronic nephritis, 637.
Catarrh, of eustachian tube, in hypertrophy of tonsils, 308; gastric, 337; nasal acute, 457; prophylaxis in, 459; chronic, 460; with adenoid growths, 296; foreign bodies in nose, 460; nasal polypi, 461; rhinitis, simple chronic, 461; epidemic, 1138; syphilitic, 462; rhinopharyngeal, with adenoids, 296.
Catheters, sizes required for infants, 615.
Cellulitis, of abdominal wall with peritonitis, 445; of neck, in scarlet fever, 964.
Cephalhematoma, 97.
Cereals, 164; allowed from third to sixth year, 212.
Cerebral abscess, 761.
Cerebral tumor, 762.
Cerebral paralysis, infantile, 779; from hemorrhage, 106; etiology of, 106.
Cerebrospinal meningitis (see MENINGITIS, ACUTE CEREBROSPINAL), 727.
Chest, circumference of, 24; development of, 24; "funnel" chest, 24; lateral depressions of, in adenoids, 296.
Chicken-pox (see VARICELLA), 994.
Chloral, dosage and administration of, 56.
Chlorosis, 843; treatment of, 847.
Cholera infantum, 363; treatment of, 367, 373.
Chondrodystrophy, 897.
Chorea, 694; endocarditis in, 596, 697; diagnosis of, 698; etiology of, 694; following birth paralysis, 783; typhoid fever, associated with, 1064; heart murmurs in, 697; prognosis of, 698; hysterical, 707; with adenoids, 297; in rheumatism, 695, 1152; pathology of, 696; posthemiplegic, 701; in cerebral palsy, 783; prognosis of, 698; relation of, to rheumatism, 695; speech in, 697, 711; symptoms of, 696; treatment of, 698.
Circulation, changes in, at birth, 575; fetal, 575; in early life, 575.
Circulatory system, diseases of, 575.
Cleft palate, 265.
Clothing, at birth, 2; in summer, 3; at night, 3; in summer diarrhea, 368.
Club-foot, with spina bifida, 799.
Codein, dosage of, 56.
Cod liver oil, as tonic, 54.
Cold, antipyretic methods in use of:—ice cap, 52; sponging, 52; pack, 50; bath, 53; colon irrigation, 53.
Cold sores, 269.
Colic, habitual, 194; intestinal, 401; renal, 647.
Colitis, acute (see ILEOCOLITIS, ACUTE), 373; amebic, 390; membranous, 379; membranous gastritis, with, 338.
Collapse, in acute bronchopneumonia, treatment of, 525; in acute peritonitis, 447; in ulcer of stomach, 343.
Collapse, pulmonary (see ATELECTASIS, ACQUIRED), 553.
Colles's law, 1105.
Colon, abnormal position of, 350; dilatation of, 408; in rickets, 254; follicular ulcer of, 377; hypertrophy of, 408; irrigation of, 53, 64; gastro-enteric intoxication, 371; in acute ileocolitis, 388; membranous inflammation of, 385.
Colostrum, 134.
Coma, in diabetes mellitus, 1156.
Compression-myelitis (see MYELITIS), 804.
Condensed milk, as a cause of rickets, 241; composition of, 158; dilution of, for infants, 158; fresh, 158.
Congenital diseases, ichthyosis, 875; myatonia, 827; myotonia, 702; rickets, 256; syphilis, 1104; tuberculosis, 1069.
Conjunctiva, catarrhal inflammation of, in measles, 986; hemorrhage from, in newly born, 105.
Constipation, in rickets, 254; chronic, 403; treatment of, 405; dilatation of colon in, 408; anal fissure from, 431; early symptom of rickets, 248; from deficient fat in food, 241; in intussusception, 416.
Contractures, hysterical, 706.
Convulsions, 671; symptoms of, 672; treatment of, 675; causing death without other symptoms, 47; epileptic, 687; hysterical, 707; in acquired cerebral paralysis, 785; in cerebral hemorrhages, 109; in congenital atelectasis, 75; in pertussis, 1009; in rickets, 248.
Cord, spinal, diseases of, 796; malformations of, 796; meningitis, 802; myelitis, 803; pressure-paralysis of, 808; tumors of, 819; weight of, 669.

Cord, umbilical, care of, 1; separation of, 2.
Corpuscles, of blood, 839.
Coryza, 457; early symptoms of measles, 978; syphilitic, 462, 1111.
Cough, hysterical, 707; from tuberculous bronchial glands, 1096; whooping (see Pertussis), 1003.
Counterirritants, 57.
Cow's milk (see Milk), 144.
Craniotabes, early symptom in rickets, 248.
Cranium, syphilitic nodes on, 1119.
Cream, 151; to secure different percentages of, 151, 152.
Cream-gauge, 139, 149.
Credé's method of preventing ophthalmia neonatorum, 1; treatment of ophthalmia, 90.
Cretinism sporadic, 881.
Croup, bronchial, 485; catarrhal, 465; spasmodic, 465.
Croupous tonsillitis, 300.
Cry, causes and varieties of, 34; in diseases, 35; in colic, 402.
Cryptorchidism, 652.
Cups, dry, indications for, 58.
Cyanosis, in acute bronchopneumonia, 506, 508; in acute inanition, 220; in chronic cardiac disease, 598; in congenital atelectasis, 74; in congenital disease of heart, 583; in diphtheritic paralysis, 834; in malaria, 1142, 1144; of face, from pressure at root of lung, 1097.
Cyclic vomiting, 331.
Cyst, of brain, 762; of brain, in infantile cerebral paralysis, 781.
Cysticerci, 423.

Dactylitis, syphilitic, 1116; tuberculous, 918.
Deaf-mutism, 795.
Deafness, following mumps, 1019; with adenoids, 296; with hypertrophy of tonsils, 309; sudden, in late syphilis, 1122.
Death, most frequent causes of, at different ages, 46; sudden, causes of, 48.
Deformities, hysterical, 706; in rickets, 248.
Dental caries, 272.
Dentition, 28; eruption of first teeth, 28; eruption of permanent teeth, 29, 30; delayed, 29; before birth, 28; difficult, 273; in rickets, 254.
Development, conditions interfering with, 30; muscular, 25; of body, 15.
Dew's method of inducing artificial respiration, 73.
Diabetes insipidus, 621.
Diabetes mellitus, 1155.
Diagnosis, general considerations in, 31.
Diapers, 3.
Diarrhea, general consideration of, 350; deaths from, in New York in five years, 350; prevalence of, during summer, 351; impure milk as a cause of, 352; observations of the Rockefeller Institute, on association of feeding impure milk and diarrheal disease, 343 et seq.; inflammatory (see Ileocolitis, Acute), 373; with acute intestinal indigestion, 357; with chronic intestinal indigestion, 395; summer, 357.
Diathesis, exudative, 261; neuropathic, 262.
Diet (see also Feeding), as cause of chronic constipation, 403; as cause of rickets, 241; in acute gastric-enteric infection, 368; in acute gastric indigestion, 328; in chronic constipation, 405; in chronic gastric indigestion, 340; in dental caries, 272; in eczema, 928; in intestinal indigestion, 388; in malnutrition, 230; in rickets, 258; in scurvy, 239; of nurse, effect of, on milk, 142.
Digestion, gastric, 315; duration of, 317; in infancy, 315; intestinal, 318.
Digestive system, diseases of, 267.
Digitalis, dosage of, 54; in cardiac disease, 593, 604.
Dilatation, of stomach, 341.
Diphtheria, 1020; bacillus (see Bacillus of Diphtheria), 1020; bronchopneumonia in, 517, 1028, 1036; blood in, 1028; cardiac failure in, 1038; cardiac thrombi in, 1028; catarrhal, 1024, 1030; complications and sequelæ, 1036; diagnosis, 1038; bacteriological, 1041; clinical, 1039; from pseudodiphtheria, 1041; distribution and mode of communication of, 1021; etiology of, 1020; fibrinous bronchitis in, 485; immunity to, 1022; immunization from, 1044; ileocolitis in, 1037; incubation, 1023; lesions, 1023; membrane, 973; proctitis in, 432; myocarditis in, 605, 1038; nasal syringing in, 1046; nephritis in, 1027, 1037; of esophagus, 311; otitis in, 1036; paralysis after, 1037; paralysis in, 832; prognosis of, 1042; prophylaxis in, 1043; quarantine in, 1043; simulated after tonsillotomy, 310; symptoms of, 1029; thrombosis in, 1036; toxins of, 1024; treatment of, 1045; local treatment of, 1046; serum treatment of, 1047; treatment of children exposed to, 1044; treatment of suspected cases of, 1043; laryngeal, 1029, 1033, 1052; nasal, 1030, 1031; pseudo- (see Pseudo-diphtheria), 300; scarlatiniform erythema in, 970; tonsillar, 1030.
Diphtheria antitoxin, dosage of, 1048; immunizing dose of, 1044; local and general effects of, 1049; other treatment with, 1045, 1046; real and alleged dangers from, 1050; strength of, 1048; time of administration of, 1048.

Diplegia, in birth paralysis, 783; from meningeal hemorrhage, 109; spastic, 779.
Disease, peculiarities of, in children, 30; etiology of, 30; symptomatology and diagnosis of, 31; pathology of, 41; prognosis of, 43; prophylaxis in, 50; therapeutics in, 51.
Dover's powder, dosage of, 56.
Dropsy (see also EDEMA), in acute diffuse nephritis, 632; in chronic cardiac disease, 591; in chronic nephritis, 636; in tuberculosis, 1091; without renal disease, 224.
Drugs, administration of, 51; antipyretics, 51; sedatives, dosage of, 56; stimulants, dosage of, 55; elimination of, in breast milk, 143; well borne by children, 57; not well borne by children, 57.
Duodenum, congenital atresia of, 118.
Dura mater, hematoma of, 724; thrombosis of the sinuses of, 757.
Dysentery (see ILEOCOLITIS, ACUTE), 373.
Dysphagia, hysterical, 708; in retropharyngeal abscess, 291.
Dyspnea, evidence of, 34; from tuberculous bronchial lymph nodes, 1096; in acute catarrhal laryngitis, 469; in catarrhal spasm of larynx, 466; in chronic cardiac disease, 597; inspiratory, in retro-esophageal abscess, 313; from pressure of abscess on pneumogastric, 314; spasmodic, in asthma, 489.
Dystrophy, muscular, 824.

Ear, middle, inflammation of (see OTITIS), 938; in measles, 986; in scarlet fever, 965.
Ecchymoses, on purpura, 857; in scurvy, 235; in leukemia, 851.
Echinococcus, of liver, 443.
Eclampsia (see CONVULSIONS), 671.
Ecthyma gangrenosa, 932.
Ectocardia, 582.
Eczema, 923; etiology of, 924; diagnosis of, 926; treatment of, 927; intertrigo, 926.
Edema, in acute diffuse nephritis, 632; in anemia, 842; in chronic nephritis, 636; in cardiac disease, 584; in leukemia, 851; of face, from pressure at root of lung, 1097; general, in marasmus, 224.
Edema glottidis, 441; in corrosive esophagitis, 312; in quinsy, 308.
Emboli, infections, in malignant endocarditis, 604.
Embolism, 612; in diphtheria, 1036.
Emphysema, 555; symptoms, 557; acute, in bronchitis of infants, 478; in acute bronchopneumonia, 505; in pertussis, 1009.
Empyema, 563; lesions, 563; symptoms, 566; diagnosis, 567; treatment, 570; in acute bronchopneumonia, 504.
Encephalocele, 720; symptoms of, 721; treatment of, 722.
Endarteritis, syphilitic, of brain, 1108; tuberculous, 748.
Endocarditis, acute simple, 594; lesions, 595; symptoms, 596; treatment of, 602; in chorea, 595; chronic (see also HEART, VALVULAR DISEASE), 597; fetal, 579; in chorea, 697; in rheumatism, 1152; malignant, 604.
Enemata, 65; nutrient, 66; drugs by, 66; astringent, in chronic ileocolitis, 389; in chronic constipation, 407; in colic, 402; injuries to rectum from, 431.
Enuresis, 662.
Epilepsy, 686; diagnosis of, 691; hysterical, 707; in acquired cerebral paralysis, 786; in birth paralysis, 783; Jacksonian, in cerebral tumor, 765; mental condition in, 690; prognosis of, 692; status epilepticus, 691; types of symptoms, 688; treatment of, 692.
Epiphyseal separation, in acute arthritis, 900; in scurvy, 237; in syphilis, 867.
Epiphyses, enlargement of, in rickets, 253; in syphilis, 1106.
Epiphysitis (see ARTHRITIS ACUTE), 900; syphilitic, 1106, 1118.
Epispadias, 651.
Epistaxis, 463; in anemia, 842; in pertussis, 1008; in purpura, 857.
Epitrochlear lymph nodes, in syphilis, 1125.
Erb's paralysis, 111.
Erysipelas, in newly born, 86.
Erythema, following diphtheria antitoxin, 1050; intertrigo, 926; in intestinal indigestion, 397; in rheumatism, 1153; scarlatiniform causes, 970.
Esophagitis, acute, 311; catarrhal, 311; corrosive, 312.
Esophagus, diseases of, 311; abscess behind, 312; congenital narrowing of, 311; congenital obstruction in, 311; diphtheria of, 1026; malformation of, 311; stricture of, 311; thrush in, 311.
Examination, of sick child, 34.
Exercise, importance of, 7; caution regarding, in heart disease, 603; in anemia, 848.
Exstrophy of bladder, 651; exudative diathesis, 261.
Eye, keratitis, interstitial, in syphilis, 1121; care of, at birth, 1, 3; diphtheritic paralysis of, 834; early use of, 26; ectropion of, in congenital ichthyosis, 920; inflammation of, in newly born, 89; in measles, 986; nystagmus, 701.

Face, expression of, in disease, 34; cyanosis and edema of, from pressure at root of lung, 1097.

Facial paralysis at birth, 110; acquired, peripheral, 836; in otitis, 944.
Feces, 319; of milk diet, 319; of mixed diet, 320; incontinence of, 435.
Fat, determination of, in milk, 140; in the feces, 319; lack of, a cause of rickets, 241; in woman's milk, 135; percentage of, in modification of cow's milk, 184, 187, 188; symptoms from deficiency of, in food, 193; symptoms from excess in food, 192, 194; function of, in diet, 131.
Fatty degeneration, of the newly born, 93.
Feeding, artificial, principles of, 181; rules for, 189, 197; indications for special variations in, 192, 195; schedule for first year, 189; *versus* wet nursing, 168; breast, schedule, for, 171; other than milk, first year, 198; daily dietary from fifteen to twenty months, 211; for healthy infants, second year, 209 *et seq.*; difficult cases, 200 *et seq.*; from third to sixth year, 211; articles allowed, 211; articles forbidden, 213; during acute illness, 214; in infants, 214; older children, 214; during periods of excessive heat, 367; by gavage, in acute illness, 214; nasal, 64; in acute intestinal indigestion and diarrhea, 357; methods of, in etiology of diarrhea, 351; mixed indications for, 179; simple rules in, 214.
Fever from insufficient nourishment, 172; inanition, 122 (see also TEMPERATURE).
Finger (see DACTYLITIS).
Fingers, clubbing of, in congenital heart disease, 584; food intoxication, 365.
Fissure of the anus, 431.
Flatulence, cause of colic, 402; in intestinal indigestion, 397.
Flexner's serum for cerebrospinal meningitis, 738.
Fetal circulation, 575; endocarditis, 579.
Fetus, evidences of syphilis in, 1106.
Follicular ulceration, of intestine, 377.
Fontanel, bulging of, in cerebrospinal meningitis, 735; bulging of, in meningeal hemorrhage, 109; bulging of, in tuberculous meningitis, 751; closure of, 22; in cretinism, 883; in rickets, 250.
Food, constituents, 128; protein, 129; fats, 131; carbohydrates, 131; mineral salts, 132; water, 133; farinaceous, a cause of eczema, 928; in chronic indigestion, 340; second year, 209; improper, in etiology of diarrhea, 351; of dental caries, 272; regurgitation of, causes and treatment, 193.
Foods, infant, 165; cause of rickets, 241; cause of scurvy, 232; indicated, in chronic constipation, 405.
Foreign bodies, swallowing of, 339; in the larynx, 471.
Fractures, green-stick, in rickets, 244, 252.
Freeman's pasteurizer, 155.
Friedreich's ataxia, 820.
Fröhlich's syndrome, 767.
Fruit, best time for giving, 211; during second year, 211; during third to sixth year, 211.
Furunculosis, 887.

Gangrene, of the face in noma, 286; of intestine, in intussusception, 413; of lung, 552; in acute bronchopneumonia, 505; in lobar pneumonia, 528; in scarlet fever, 969; in measles, 985.
Gangrenous stomatitis, 285.
Gastritis, acute, 335; symptoms, 337; treatment, 338; chronic, 339; ulcers in, 342; toxic (see GASTRITIS CORROSIVE), 338.
Gastro-enteritis (see ACUTE INTESTINAL INDIGESTION AND DIARRHEA), 357; in newly born, 85.
Gavage, 63; in acute illness, 215; in acute inanition, 221; in diphtheria, 1046; in premature infants, 13.
Genital organs, diseases of, 650; care of, in newly born, 4; malformations of, 650; female, gangrene of, 285; females, diseases of, 655; hemorrhage from, in newly born, 105; males, diseases of, 653.
Gingivitis, in dental caries, 272; in scurvy, 234, 235.
Glands, bronchial (see LYMPH NODES, BRONCHIAL), 1095.
Glands, lymphatic (see LYMPH NODES), 860.
Glioma of brain, 762; of spinal cord, 819.
Gliosarcoma of brain, 763.
Glossitis, 270.
Glottis, edema of the, 471.
Gonococcus, differentiation of, 658; in gonorrheal stomatitis, 284; in specific urethritis, 653; vaginitis, 656.
Grippe, 1138.
Growing pains, rheumatic, 1151.
Growth, conditions interfering with, 31; of body, 15; extremities, 21; trunk, 21.
Gumma, syphilitic (see SYPHILIS LESIONS), 1106; in syphilitic bone disease, 1118; of brain, 762.
Gums, abscess of, 272; bleeding in ulcerative stomatitis, 279; inspection of, 38; lancing, 275; spongy and bleeding, in scurvy, 234, 235; in ulcerative stomatitis, 279.

Habit-spasm, 700.
Habits, injurious, 714.
Hematemesis, 345.
Hematoma of the sternomastoid, 94.
Hematuria, 619; in newly born, 194; in

purpura, 856; in pyelitis, 645; in scurvy, 237; in tumors of kidney, 641.
Hemoglobinuria, 619; epidemic, 92; paroxysmal, 620.
Hemophilia, 852.
Hemorrhage, from stomach, 345; in hemophilia, 852; intra-alveolar, in acute bronchopneumonia, 500; internal, causing sudden death, 46; intestinal, from tuberculous ulcer, 394; in typhoid fever, 1063; meningeal, causing birth paralysis, 779; in acquired cerebral paralysis, 781; in acute bronchopneumonia, 519; in convulsions, 672; meningeal, in pertussis, 1008; meningeal, in purpura, 855; nasal, in diphtheria, 1037; pulmonary, in cardiac cases, 597; rectal, from ulcer, 433; in leukemia, 850; in measles, 986; in pertussis, 1008; in pernicious anemia, 847; in purpura, 855; in the newly born, 96; visceral, 100; in scurvy, 237; in syphilis, 1113.
Hemorrhagic disease of the newly born, 98.
Hemorrhoids, 435; in chronic constipation, 404.
Harelip, 266.
Hay fever, 490.
Head, circumference of, 22; closure of sutures, 22; closure of fontanels, 22; shape of, 23; in rickets, 248; examination of, 35; hydrocephalic, characteristics of, 772; nodding spasm of, 701.
Headache, varieties, 709; treatment, 710.
Head-banging, 719.
Hearing, when developed, 26.
Heart, diseases of, 575; aneurism of, 612; aortic disease, congenital, 581; auscultation of, 39, 578; diphtheritic paralysis of, 835; examination of, 577; hypertrophy of, in valvular diseases, 596; in measles, 986; in scarlet fever, 967; malformations of, 579; peculiarities of, in early life, 575; persistent fetal conditions, 580; position of apex beat, 577; in infancy, 577; size and growth of, 577; sounds of reduplication, 578; sudden failure of, in diphtheria, 1038; thrombus of, ante-mortem, 612; transposition of, 583; congenital anomalies of, 579; functional disturbances of, 608; murmurs of, 598; differential diagnosis of, 586; accidental, 606; in congenital diseases, 585; in chorea, 697; in marasmus, 224; valves, aortic insufficiency, 600; aortic stenosis, 600; mitral insufficiency, 599; mitral stenosis, 599; valvular diseases of (see also ENDOCARDITIS), 594; chronic valvular disease of, 597; ventricle, left, signs of dilatation, 599; signs of hypertrophy, 600; right, signs of hypertrophy, 599.
Heart block, in diphtheritic paralysis, 835.
Height, 19; from birth to sixteenth year, 20.
Heliotherapy, in tuberculous peritonitis, 453.
Hemichorea, 696.
Hemiplegia, in acquired cerebral paralysis, 782; in birth paralysis, 779; in meningeal hemorrhage, 109; in cerebral tumor, 766; spastic, 781.
Hepatitis, interstitial, 77; suppurative, 439.
Hermaphroditism, false, 651.
Hernia, cerebri, 720; diaphragmatic, 119; umbilical, 116.
Herpes labialis, 269.
Herpetic stomatitis, 277.
Hiccough, 702; in acute peritonitis, 447; in hysteria, 707.
Hip-joint disease, 912.
History-taking, 32.
Hives (see URTICARIA), 933.
Hodgkin's disease, 874.
Holding-breath spells, 681.
Home modification of milk, 196.
Hookworm, 429.
Hutchinson's teeth, in syphilis, 1117.
Hydatids, of liver, 443.
Hydrencephalocele, 719.
Hydrocele, 654.
Hydrocephalus, 769; in chronic basilar meningitis, 755; with spina bifida, 799, 801; acute (see MENINGITIS, TUBERCULOUS), 747, 754; chronic external, 769; internal, 770; congenital, 723; intra-uterine, 721; syphilitic, 1108.
Hydronephrosis, 624; with malformations of kidney, 623; with renal calculi, 647.
Hygiene, of infancy, 1.
Hyperesthesia, general, in cerebrospinal meningitis, 733; in acute poliomyelitis, 811; hysterical, 705; in multiple neuritis, 831; in scurvy, 236; in spinal meningitis, 802.
Hypertrophy, muscular pseudo-, 824.
Hypodermic medication, 66; dosage for, 55.
Hypodermoclysis, indications for, 66.
Hypospadias, 651.
Hysteria, 705; symptoms, 705; diagnosis, 707; treatment, 708.
Hystero-epilepsy, 707.

Ice bag, 59.
Ice cap, 52, 59.
Ice coil, 59.
Ichthyosis, congenital, 920.
Icterus, 438; in epidemic hemoglobinuria, 92; varieties in newly born, 77; in malformation of the bile ducts, 78;

interstitial hepatitis, 78; physiological or idiopathic, 78; differential diagnosis, 81.
Idiocy, 789; Mongolian, 793; amaurotic family, 788.
Idiosyncrasies to foodstuffs, 216.
Ileocolitis, acute, 373; catarrhal, 375; follicular, 377; membranous, 385; associated lesions, 381; with follicular ulceration, 377; membranous form, 378; treatment, 378, *et seq.*; bronchopneumonia complicating, 518; in diphtheria, 1037; in measles, 985.
Ileum, congenital atresia of, 118.
Imbecility, 789.
Impetigo, bullous, 94; in newly born, 94; contagiosa, 932.
Inanition, acute, 219.
Inanition fever, 122.
Incubators, 12.
Indican, in urine of chronic constipation, 404; of chronic intestinal indigestion, 398.
Indigestion, chronic gastric, 339; treatment, 340; acute intestinal, and diarrhea, 357; diagnosis, 366; treatment, 367; Finkelstein's "food intoxication," 365.
Indigestion, chronic intestinal, 395; treatment, 399.
Infant, care of newly born, 1; when premature or delicate, 11.
Infant feeding, 167.
Infant foods, 165.
Infantilism, intestinal, 398.
Infarctions, uric acid, in kidney, 627.
Infectious diseases, specific, 949.
Influenza, 1130; etiology, 1130; lesions, 1131; symptoms, 1132; bronchopulmonary complications, 1155; protracted cases, 1133; complications and sequelæ, 1136; anemia in, 1136; diagnosis, 1136; prognosis, 1137; treatment, 1137; bronchopneumonia in, 517, 1134; epidemic, acute otitis in, 938; scarlatiniform erythema in, 970; nephritis in, 1136.
Inhalations, 62; in bronchitis, 484.
Inheritance, a factor in disease, 30.
Injections, rectal, in ileocolitis, 388; subcutaneous, of saline solution in cholera infantum, 373.
Intertrigo, 926.
Intestinal obstruction in newly born, 118; acute, from intussusception, 410.
Intestines, diseases of, 348; amyloid degeneration of, 391; bacteria of, 318; digestion in, 317; hemorrhage from, in newly born, 104; in typhoid, 1063; in tuberculosis, 393; length, 318; malformations of, 348; obstruction, congenital of, 118; perforation of, in tuberculous ulcers, 393; in typhoid fever, 1063; tuberculosis of, 391, 1082; etiology, 392; lesions, 392; symptoms, 393; treatment, 394.
Intoxication, acute intestinal and diarrhea, 357; etiology, 357; lesions, 358; symptoms, mild form, 359; relapses, 360; cases without diarrhea, 362; diagnosis, 366; prognosis, 366; prophylaxis, 366; treatment, 367, 372; cholera infantum, 363; treatment, 367; acidosis in, 372.
Intubation, in acute catarrhal laryngitis, 468; in syphilitic laryngitis, 474; in pertussis, 1013.
Intussusception, 410; etiology, 412; lesions and mechanism, 412; symptoms, 414; diagnosis, 417; treatment, 418; laparotomy, 418; in the dying, 411.
Iodids, elimination of, in milk, 143.
Iritis, syphilitic, 1109.
Iron, preparations of, 55.
Irrigation, intestinal, in chronic indigestion, 400; as antipyretic, 53; of the colon, method of, 64.
Ischiorectal abscess, 434.

Jaundice (see also Icterus), 77; catarrhal, 437.
Jaw, necrosis of, from alveolar abscess, 273; in gangrenous stomatitis, 286; in ulcerative stomatitis, 279.
Jejunum, congenital atresia of, 118.
Joints, diseases of, 895; hysterical affections of, 706; in scarlet fever, 967; rheumatism of, 1150; suppuration of, in newly born, 85; swelling of, in scurvy, 236; ecchymoses about, in scurvy, 235; tuberculous diseases of, 905.
Junket, 162.

Kernig's sign, 733.
Keratitis, interstitial, in late syphilis, 1109, 1121.
Kidney, diseases of, 623; acute degeneration of, 628; calculi in, 646; chronic congestion of, 628; cystic, 624; movable, 627; granular (see Nephritis, Chronic), 636; horseshoe, 623; hydronephrosis, 624; malformations and malpositions of, 623; malignant tumors of, 639; nephritis, acute diffuse, 628; acute exudative, 629; chronic, 635; perinephritis, 648; pyelitis, 642; pyonephrosis, 645; tuberculosis of, 639, 1081; uric-acid infarction in, 627; in diphtheria, 1027; in scarlet fever, 966.
Klebs-Loeffler bacillus (see Bacillus of Diphtheria), 1020, 1039.
Knee, articular ostitis of, 916; subluxation of, in poliomyelitis, 815; swelling of, in scurvy, 234.
Knee-jerk, in acquired cerebral paralysis, 785; in birth paralysis, 783; lost, in

diphtheritic paralysis, 834; in multiple neuritis, 831; in tetany, 677.
Knock-knee in rickets, 253.
Koplik's sign in measles, 978.
Kumyss, 160.
Kyphosis, in rickets, 251; treatment, 258; in spinal caries, 907.

Lactalbumin, 138, 183.
Lactation, care of breasts during, 169.
Lactic acid milk, 160.
Lactometer, author's, 139.
Larynogospasm, 684; in rickets, 248; with tetany, 681.
Laryngitis, acute catarrhal, 468; catarrhal in measles, 984; chronic, 472; with adenoid vegetations of pharynx, 472; tuberculous, 472; syphilitic, 473; with new growths of larynx, 474; spasmodic, 465; submucous (edema of glottis), 471.
Laryngeal diphtheria, 1033; antitoxin in, 1048; intubation in, 1053; symptoms of, 1033.
Laryngotomy for foreign body in larynx, 475.
Larynx, diseases of, 465; foreign bodies in, 475; new growths of, 474; spasm of, 684.
Lavage (see STOMACH WASHING), 62.
Leukemia, 849.
Lichen, urticatus (see URTICARIA), 934; tropicus, 922.
Lip, eczema of, 269; perléche, 269; diseases of, 269; herpes of, 269; malformations of, 267.
Lisping, 711.
Liver, diseases of, 436; abscess of, 439; acute yellow atrophy of, 438; amyloid degeneration of, 441; biliary calculi, 443; cirrhosis of, 440; congestion of, 439; interstitial hepatitis, 78; enlarged in congestion, 439; in abscess, 439; in cirrhosis (early), 441; in chronic cardiac disease, 593; in marasmus, 222; hydatids of, 443; in rickets, 255; in syphilis, 1107, 1022; in tuberculosis, 1080; lardaceous, 441; malformations and malpositions of, 437; size and position of, 40, 436; tuberculosis of, 1080; waxy, 441; weight of, in infancy, 436.
Lumbar puncture, 737.
Lung, diseases of, 476; abscess of, 551; abscesses of, in acute bronchopneumonia, 505; acute congestion of, in malaria, 1140; calcareous nodules in, 1078; caseous degeneration of, 1077; collapse of, from compression, 553; from obstruction, 554; in acute bronchopneumonia, 500; congenital atelectasis of, 74; emphysema of, 555; acute, in bronchitis of infants, 482; gangrene of, 552; gangrene of, in lobar pneumonia, 528; hemorrhages into, in newly born, 100; inflation of, 73; miliary tuberculosis of, 1084; peculiarities in disease, 478; in infancy and early childhood, 476; physical examination of, 477; structure of, 477.
Lymph nodes, diseases of, 862; calcareous cervical, 869; bronchial, 1095; early infection in tuberculosis, 1072; enlarged in Hodgkin's disease, 874; in malnutrition, 227; frequency of disease of, 41; inflammation of (see ADENITIS), 862; in late hereditary syphilis, 1121; in measles, 986; in pseudodiphtheria, 301; in scarlet fever, 964; simple hyperplasia of, 865; situation and drainage areas of the groups of head and neck, 861; syphilitic disease of, 866; tuberculous bronchial, 1078; lesions, 1078, 1082; symptoms, 1031; cervical, tuberculosis of, 867; mesenteric, 393, 1081; in diphtheria, 1027; in rickets, 255; in tonsillitis, 301; epitrochlear, in syphilis, 1121; in typhoid fever, 1060; tuberculosis of, 867; retropharyngeal, abscess of, 290.
Lymphocytes, 840.

Malaria, 1139; symptoms, 1140; treatment, 1146; quinin, methods of administration, 1146; spleen in, 878.
Malnutrition, 226.
Maltose, in infant feeding, 208.
Malt soup, use of, with difficult feeding cases, 185, 208.
Mania, acute, following typhoid, 1064.
Marasmus, 221.
Massage, 66; in chronic constipation, 406; in malnutrition, 230; of breasts to increase milk, 174.
Mastitis, in the newly born, 117.
Mastoid disease, cerebral abscess following, 899; in acute otitis, 898.
Mastoiditis, 942; symptoms, 898; treatment, 946; dangers from operation, 946.
Masturbation, 715.
Matzoon, 161.
Measles, 975; bronchopneumonia complicating, 517; complications and sequelæ, 983; desquamation, 981; diagnosis, 987; digestive system, 985; diphtheria in, 987; duration of infective period, 977; ears in, 938, 986; eruption, 979; etiology, 975; eyes in, 986; German (see RUBELLA), 991; hemorrhage in, 986; hemorrhagic, 980; heart in, 986; ileocolitis, 985; incubation, 976; invasion, 978; kidneys in, 986; larynx in, 984; lesions, 977; lungs, 984; lymph nodes, 986; mode of infection, 977; mortality, 988; nervous system in, 986; other infectious diseases in, 987; otitis, 986; predisposition, 976; prognosis, 988; prophylaxis, 989; pseudodiphtheria in,

985; quarantine in, 989; skin in, 986; symptoms, 978; throat, 985; treatment, 989; tuberculosis following, 987.
Meats, from third to sixth year, 212.
Meckel's diverticulum, 114, 349.
Meconium, composition of, 319.
Mediastinum, anterior, abscess of, 1097; tumor of, due to tuberculous lymph nodes, 1096.
Mediastinitis, 589.
Melena, 104.
Meningeal hemorrhage, 102, 724, 779.
Meninges, diseases of, 719.
Meningitis, acute, 726; cerebrospinal, 727; complications and sequelæ, 736; course, duration and termination, 736; lesions, 728; lumbar puncture in, 737, 743; symptoms, 730, 736; diagnosis, 737; prognosis, 736; treatment, 738, 743.
Meningitis, acute, from other causes than the meningococcus, 743; pneumococcus, 744; influenza, 745; septic, 746; in newly born, 85; from otitis, 943; in pneumonia, 519, 539.
Meningitis, chronic basilar, 755; spinal, 802; syphilitic, 1108.
Meningitis, tuberculous, 747; symptoms, 749; diagnosis, 752; lumbar puncture in, 756.
Meningocele, of brain, 720; of cord, 797.
Meningo-encephalitis, 780.
Meningomyelocele, 798.
Menstruation, effect of, on breast milk, 142.
Mental deficiency, 789; diagnosis, 790, 793; treatment, 796.
Mercury, elimination of, in milk, 143; ulcerative stomatitis from, 278; in syphilis, 1128.
Microcephalus, 722.
Microörganisms in cow's milk, 145.
Micturition, difficult or painful, 667; frequency of, 616.
Miliaria, 921; papulosa, 922; treatment, 927; rubra, 922.
Milk, cow's, 144; composition of, 182; bacteriological standard for, 147; handling and transportation of, 144, 145; average percentages in, from different breeds, 148, 149; examination of, 149; cream, 151; contaminated, as cause of diarrhea, 352; differences from woman's milk, 150; dried milk, 159; essentials of, for infant feeding, 144; formulas from diluting, 187, *et seq.*; formulas reduced to percentages, 191, 192; frozen, 157; microörganisms in, 145; modification of, percentage, 187; at home, 186, 187, 196; top-milk, 152; formulas from whole milk, 187; schedule showing quantities and intervals of feeding, 189; modifications required by particular symptoms, 192; in difficult cases, 207; in summer diarrhea, 368; in chronic constipation, 405; pasteurization of, 153, 154, 155; protein of, 129; sterilization of, at 212° F., 155; tubercle bacilli in, 1020; condensed (see Condensed Milk), 158; fermented, 159, 207; protein, 161; skimmed, 207; peptonized, 157.
Milk laboratories, 197.
Milk-sugar, 132, 184; solution, how to prepare, 196.
Milk, woman's, 134; physical characters of, 134; colostrum of, 135; daily quantity of, 135; average quantity at one nursing, 137; composition of, 137; protein, 137, 138, 140; fat, 138; sugar, 138; salts, 138; reaction, 139; specific gravity, 139, 141; average percentages of, 182; conditions affecting composition of, 141, 144; menstruation, 142; diet, 142; drugs, 143; pregnancy, 143; elimination of antitoxin and other protective substances, 137; nervous impressions, 143; examination of, 139; variations in quality, 141; how to modify quantity and quality, 174, 175; indications of scanty supply, 171.
Modified milk, schedule for feeding from birth, 189.
Mongolian idiocy, 793.
Monoplegia, in birth paralysis, 781; in cerebral hemorrhage, 109; in cerebral tumor, 766.
Morphin, dosage of, 56; dosage in convulsions, 676; hypodermically in cholera infantum, 372.
Mortality, at different ages, 43, 44.
Mouth, diseases of (see also Stomatitis), 276, *et seq.*; care of, at birth, 1, 3; hemorrhages from scurvy, 236; malformations of, 267; mucous patches, in syphilis, 284; "tapir," 826.
Mouth breathing, with hypertrophy of tonsils, 309; with adenoids, 297; with retropharyngeal abscess, 291.
Mumps, 1015; complications and sequelæ, 1018; diagnosis, 1019; pathology and lesions, 1016; symptoms, 1017; treatment, 1019.
Murmurs, cardiac (see Heart Murmurs).
Muscles, atrophy of, 820; in multiple neuritis, 831; in myelitis, 803; in poliomyelitis, 815; contractures of, hysterical, 706; in infantile cerebral paralysis, 786; in birth paralysis, 782; development of, 25.
Muscular wasting diseases, different types of, 822.
Muscular pseudohypertrophy, 825.
Mustard bath, 59; paste, 57; pack, 58.
Myelitis, 802; symptoms, 803; treatment, 804; from Pott's disease, 804; diffuse, 803; transverse, 803.
Myocarditis, 605; aneurism in, 605; toxic,

in diphtheria, 835, 1036; in scarlet fever, 968; in syphilis, 1108.
Myatonia, congenital (Oppenheim's disease), 827.
Myotonia, congenital (Thomsen's disease), 702.

Nail-biting, 718.
Nails, in syphilis, 1114.
Neck, cellulitis of, in scarlatina, 964; congenital fistula of, 311; wry- (see Torticollis), 703.
Necrosis, of bone, in syphilis, 1106.
Nematodes (see Worms), 425.
Nephritis, acute diffuse, 629; etiology, 629; lesions, 629; symptoms, 630, 633; prognosis, 633; treatment, 633; acute parenchymatous type, 630.
Nephritis, chronic, 635; etiology, 635; lesions, 636; symptoms, 636; of the parenchymatous type, 636; of the interstitial type, 637; diagnosis, 638; prognosis, 637; treatment, 638; chronic, diffuse, with hydronephrosis, 625; chronic interstitial, syphilitic, 1109; in diphtheria, 1027, 1037; interstitial (see Nephritis, Chronic), 637; post-scarlatinal, 966.
Nerves, peripheral, diseases of, 828.
Nervous impressions, effect of, on breast milk, 144.
Nervous system, diseases of, 669; general hygiene of, 5; peculiarities of, in childhood, 669.
Neuritis, multiple, 828; after diphtheria, 833; optic, in acute meningitis, 734; in cerebral tumor, 767; with cerebral abscess, 761.
Neuropathic diathesis, 262; neuropathic child, 264.
Newly born, diseases of, 69; acute infectious diseases of, 82; acute pyogenic diseases of, 82; atelectasis, congenital, 74; asphyxia of, 69; blood in, peculiarities of, 839; diseases or accidents at birth, 30; facial paralysis in, 110; fatty degeneration of, 93; hemorrhages in, 96; hemorrhagic diseases of, 101; hyperpyrexia in, 122; icterus in, 77; infection of, 31; malformations of, 30; mastitis in, 117; ophthalmia of, 89; pemphigus in, 94; peritonitis in, 444; sclerema in, 121; skin of, 920; ulcer in stomach in, 342.
Night-terrors, 713.
Nipples, care of, during lactation, 169; fissure of, hematemesis from, 346; rubber, choice of, 196; care of, 196.
Nodes, lymph (see Lymph Nodes), 861.
Nodules, subcutaneous tendinous, in rheumatism, 1152.
Noma of face (see Stomatitis, Gangrenous), 285; of vulva, 662.
Nose, diseases of, 457; deformities of, in hereditary syphilis, 463; difficulty in blowing, with adenoids, 296; diphtheria of, 1030; discharge from, with adenoids, 296; foreign bodies in, 460; hemorrhage from, 462; in newly born, 1004; in scurvy, 238; in hereditary syphilis, 462, 1108; in late syphilis, 1122; polypi in, 461; pseudodiphtheria of, 301; sprays for, 60; syringing, 61.
Nurse, requisite qualities in, 10; wet (see Wet-Nurse), 176.
Nursery, general requirements for, 9.
Nursing, during acute illness, 215; during first days of life, 169; hours for, in newly born, 170; during illness, 177; importance of good habits of, 170; unsuccessful, symptoms of, 171; maternal, contra-indications for, 168.
Nursing-bottles, choice of, 196; care of, 196.
Nutrition, derangements of, 218; acute inanition, 218; malnutrition, 226; marasmus, 221; faulty, diseases due to, 231; importance in pediatrics, 127.
Nystagmus, 701; in cerebral hemorrhage, 109; in hydrocephalus, 775; in tuberculous meningitis, 750; with tumor of crura cerebri, 766.

Oatmeal water, 165.
O'Dwyer's intubation set, 1053.
Oil enemata, 65; in chronic constipation, 407.
Omphalitis, in newly born, 83.
Omphalomesenteric duct, 115, 349.
Onychia, syphilitic, 1111.
Ophthalmia, in newly born, 89.
Opisthotonus, 7; hysterical, 707; in cerebrospinal meningitis, 731; in birth paralysis, 782; meningeal hemorrhage, 109; in chronic basilar meningitis, 756; in marasmus, 224; in tuberculous meningitis, 752.
Opium, elimination of, in milk, 143; acute intestinal indigestion and diarrhea, 371, 372; in bronchitis, 484; preparations and dosage of, 53.
Oppenheim's disease, 827.
Optic nerve, atrophy of, in cerebral tumor, 764.
Orchitis, in mumps, 1018; in specific urethritis, 653; syphilitic, 1109; tuberculous, 1082.
Orthopnea, in chronic valvular disease, 597; in functional disorders of the heart, 608.
Osteogenesis imperfecta, 895.
Osteomyelitis, in newly born, 85; tuberculous, 918.
Osteoperiostitis, chronic, syphilitic, 1114.
Ostitis, primary, followed by joint disease, 906; simulated by scurvy, 238.
Otitis, acute, 938; etiology, 938; lesions, 939; symptoms, 940; complications and

sequelæ, 942; treatment, 944; cerebral abscess in, 759, 943; thrombosis of lateral sinus in, 943; facial paralysis in, 944; labyrinth in, 944; mastoid disease in, 942; meningitis in, 943; chronic, in late syphilis, 1122; in influenza, 1139; in scarlet fever, 965; in syphilis, 1109.
Oxyuris vermicularis (see Worms, Intestinal), 427.
Ozena, syphilitic, 463, 1122.

Pachymeningitis, acute, 723; chronic (internal), 724; syphilitic, 1108; meningeal hemorrhage from, 725; hemorrhagic, 724; pseudomembranous, 724.
Pack, cold, 52; hot, 59; mustard, 58.
Palate, cleft, 267; diphtheritic paralysis of, 833; hard, ulceration of, 279; in late syphilis, 1122; soft, lesions of, in hereditary syphilis, 463.
Pancreas, ferments of, 318; syphilis of, 1109; tuberculosis of, 1081.
Paracasein, formed from casein in stomach digestion, 150.
Paralysis, atrophic (see Poliomyelitis), 806; birth, 106, 780; atrophy and sclerosis following, 780; meningoencephalitis, 780; secondary degenerations following, 781; symptoms, 781; Erb's, 111; facial, 110, 836; in acute otitis, 944; hysterical, 706; in compression-myelitis, 805; multiple neuritis, 829; in myelitis, 803; of face, in newly born, 110; of the upper extremity in newly born, 111; peripheral, 106; (see also Neuritis, Multiple), 828; postdiphtheritic, 1037; pseudohypertrophic, 824; simulated by scurvy, 238.
Paralysis, infantile cerebral, 106, 779; acute acquired, 783; birth, 780; of intra-uterine origin, 779; varieties and symptoms, 779, 781, 783; prognosis, 786; diagnosis, 787; treatment, 787.
Paralysis, infantile spinal (see Poliomyelitis), 806.
Paraplegia, Pott's (see Myelitis Compression), 779, 804; spastic, 779.
Parotitis, epidemic (see Mumps), 1015.
Pasteurized milk, 154, 155.
Pathology, general considerations of, 41.
Pavor nocturnus, 713.
Peliosis rheumatica, 859.
Pemphigus, gangrenosa, 932; in newly born, 94; syphilitic, 1110.
Pepsin, in stomach secretion, 317.
Peptonized milk, preparation of, 157.
Percentages, in milk formulas, how to calculate them, 191.
Pericarditis, 588; acute in bronchopneumonia, 519; chronic, with adhesions, 593; diagnosis, 592; dry, 589; external, 589; in newly born, 85; in rheumatism, 589, 590, 1151; mediastinal, 589; prognosis, 591; purulent, 589; serofibrinous, 589; tuberculous, 589; with effusion, 589; with effusion of blood, 589; with lobar pneumonia, 529; with pleuropneumonia, 545; with transudation of serum, 588.
Pericardium, congenital absence of, 582; tuberculosis of, 1080.
Perinephritis, 648; acute peritonitis complicating, 445.
Peritoneum, diseases of, 444; hemorrhage into, in newly born, 101; in tuberculosis, 1081.
Peritonitis, acute, 444; etiology, 444; lesions, 445; symptoms, 446; treatment, 447; chronic, non-tuberculous, 448; with ascites, 448; fetal, cause of malformations, 349; in intussusception, 417; in newly born, 84; in suppurative appendicitis, 419; pelvic from gonorrhea, 658; tuberculous, 449; miliary, with general tuberculosis, 450; with ascites, 450; fibrous form, 451; with intestinal ulcers, 393; with lobar pneumonia, 539.
Pertussis, 1003; bronchopneumonia in, 1008; complications, 1008; convulsions, 1009; diagnosis, 1010; etiology, 1004; hemorrhages in, 1008; ileocolitis in, 1009; incubation, 1005; infective period, 1009; lesions, 1005; leukocytosis in, 1011; paralysis in, 1009; predisposition to, 1004; prognosis, 1011; prophylaxis, 1012; symptoms, 1006; treatment, 1012, 1015; vaccines in, 1014.
Peyer's patches, in typhoid fever, 1060; swollen, in acute ileocolitis, 359; tuberculosis of, 393; ulceration of, in ileocolitis, 379.
Pharyngitis, acute, 288; uvulitis in, 289; chronic catarrhal, syphilitic, 1108.
Pharynx, diseases of, 288; adenoid vegetations of vault, 296; with adenitis, 862; diphtheria of, 1024; diphtheritic paralysis of, 834; lesions of, in hereditary syphilis, 463; retropharyngeal abscess, 290; syphilitic ulceration of, 1108; syringing of, 61.
Phimosis, 650.
Phlebitis, of dural sinuses, 757.
Phosphorus, in rickets, 259.
Phthisis, chronic, 1078, 1095.
Physical examination, of the child, 34.
Pica, 719.
Pinworms (see Worms, Intestinal), 427; proctitis from, 432.
Pleura, effusion into, in acute nephritis, 632; tuberculosis of, 1075, 1080.
Pleurisy, 557; dry, 558; in acute bronchopneumonia, 504; purulent (see Empyema), 563; tuberculous, dry form, 558; with lobar pneumonia, 528; with serous effusion, 560; Grocco's sign in, 561.

Pleuropneumonia, 544; pericarditis in, 588, 590.
Pneumococcus, in bronchopneumonia, 495; lobar pneumonia, 495; peritonitis, 445; diphtheria, 1023, 1035; empyema, 563; acute meningitis, 744; malignant endocarditis, 604; pericarditis, 588.
Pneumonia, 492; anatomical varieties and classifications of, 493; broncho (see Bronchopneumonia, Acute), 497; catarrhal (see Bronchopneumonia, Acute), 497; chronic interstitial (see Bronchopneumonia, Chronic), 547; in newly born, 84; in typhoid fever, 1063; sources of infection, 496; varieties, classification of, 495; hyperacute, 524; hypostatic, 547; lobular (see Bronchopneumonia, Acute), 497; pleuro (see Pleuropneumonia), 544; syphilitic, 1107; tuberculous, 1086; course, duration, termination, 1087, 1088; diagnosis, 1094; physical signs, 1092; chronic, 1093.
Pneumonia, lobar, 526; etiology, 526; frequency of, 495, 526; complicating influenza, 1133; complications, 539; course, 539; abortive, 530; cerebral, 531; diagnosis, 540; lesions, 527; lysis, frequency of, 533; pathological differentiation from bronchopneumonia, 540; physical signs, 535; prognosis, 542; symptoms, 529; termination, 539; treatment, 543.
Pneumothorax, in pulmonary tuberculosis, 1080.
Poisons, gastritis from, 336, 338.
Poisoning, stomach washing, in, 63.
Poliencephalitis, acute, causing cerebral paralysis, 785.
Poliomyelitis, acute, 806; etiology, 807; diagnosis, 816; extent and distribution of primary paralysis, 813; electrical reactions, 815; lesions, 808; prognosis, 817; symptoms, 810; treatment, 818.
Polydipsia, in diabetes insipidus, 621; mellitus, 1156.
Polypi, nasal, 461; rectal, 435.
Polyuria, 621; hysterical, 707; in diabetes insipidus, 621; in diabetes mellitus, 1156.
Porencephalus, 723.
Pott's disease (see Spine, Caries of), 907.
Precordia, bulging of, 577, 600.
Pregnancy, effect of, on woman's milk, 143; effect of, on nursing child, 178.
Premature infants, management of, 11.
Prepuce, adherent, 650.
Prickly heat, 922.
Proctitis, 432.
Prognosis, general consideration of, 43.
Progressive muscular wasting diseases, the Werdig-Hoffman type, 822; peroneal type, 823.
Prolapsus ani (see also Rectum, Prolapse of), 430; from proctitis, 433; in ileocolitis, 382; in membranous ileocolitis, 385.
Prophylaxis, general consideration of, 50.
Protein, determination of, in milk, 140; function in diet, 129; in the feces, 320; of woman's milk, 131; of cow's milk, 148; percentages of, in modification of cow's milk, 191.
Protein milk, 161.
Pseudodiphtheria (see Membranous Tonsillitis), 300.
Pseudohypertrophic paralysis, 824.
Pseudoparalysis in rickets, 257; in scurvy, 235; in syphilis, 1114.
Puberty, delayed, in cretins, 884; in syphilis, 1123; effect of, on heart, in valvular disease, 597, 601.
Pulse, examination of, 36; in early life, 576.
Purpura, 854; arthritic, 859; blood in, 856; fulminans, 858; gangrenous, 858; hematemesis in, 857; hemorrhagica, 857; Henoch's, 857; primary, 855; rheumatica, 859; simplex, 854; symptomatic, 854; cachectic, 854; infectious, 854; neurotic, 855; mechanical, 855; toxic, 854.
Pyemia, in newly born, 82; of bone (see Arthritis, Acute), 850.
Pyelitis, 642.
Pyelocystitis, 642.
Pyelonephritis, 620.
Pylephlebitis, 439; cause of hepatic abscess, 439.
Pylorus, hypertrophic stenosis of, 321; diagnosis, 325; treatment, 326.
Pyogenic diseases, acute, in newly born, 82; general symptoms, 87; prophylaxis, 86; treatment, 88.
Pyonephrosis, following pyelitis, 642.
Pyopneumothorax, in pulmonary tuberculosis, 1080.
Pyuria, 620; in pyelitis, 642.

Quinin, dosage, 1147; methods of administration, 1147; scarlatiniform rash, 970.
Quinsy, 307.

Rachitis (see Rickets), 243.
Reaction, of degeneration, in Erb's paralysis, 113; in facial paralysis, 111; in multiple neuritis, 831; in poliomyelitis, 815, 817.
Rectal injections (see Enemata), 65; astringent, 389; oil, 407; saline, 389.
Rectal polypus, 435.
Rectum, diseases of, 430; administration of drugs by, 66; atresia of, 348; con-

genital obstruction of, 118; feeding by, 66; hemorrhage from ulcers of, 433; inflammation of (see Proctitis), 432; malformations of, 348; prolapse of, 430; ulcers of, 433.
Regurgitation of food, causes of, in young infants, 193; nasal, in diphtheria, 834, 1031, 1039.
Remittent fever, malarial, 1142.
Renal calculi, 646; renal colic, 647.
Rennet ferment in digestion, 317.
Respiration, artificial, methods of, 71, 72; Cheyne-Stokes, in cerebrospinal meningitis, 734; in meningitis, tuberculous, 750; paralysis of, in diphtheria, 834; rapidity and characteristics of, 476.
Respiratory system, diseases of, 457.
Rheumatism, 1149; diagnosis, 1153; treatment, 1154; chorea in, 694, 1152; endocarditis in, 595, 1151; erythema in, 1153; purpura in, 859, 1153; scarlatinal, 967; simulated by scurvy, 238; subcutaneous tendinous nodules, 1152; tonsillitis in, 306, 1152; torticollis in, 703, 1152.
Rhinitis, chronic, 461; hypertrophic, cause of asthma, 488; simple, 461; syphilitic, 462.
Rhinopharyngitis, acute, 457; in influenza, 1131; with adenoids, 296.
Rhinopharynx, diphtheria of, 1025; simple catarrh of, in acute otitis, 939.
Ribs, beading of, early symptoms in rickets, 250; resection of, in empyema, 571.
Rice water, 165.
Rickets, 240; etiology, 241; lesions, 244; symptoms, 248; calcium metabolism in, 256; course and termination, 256; acute, 256 (see also Scorbutus), 232; congenital, 256; convulsions in, 255; nervous symptoms of, 255; diagnosis, 256; from scurvy, 238, 257; prognosis, 258; treatment, 258; of deformities, 252; late, 256; spleen in, 246, 877.
Ringworm of scalp, 936.
Rötheln (see Rubella), 991.
Roundworms (see Worms, Intestinal), 425.
Rubella, 991; eruption, 992; treatment, 994.
Rubeola (see Measles), 975.
Rumination, 330.

Saccharomyces albicans, in thrush, 281.
Saline solution, as rectal injection, 389; subcutaneous injection of, in cholera infantum, 372; in acute inanition, 221.
Saliva, 315.
Salivation, in mumps, 1017; in ulcerative stomatitis, 279.
Salvarsan, 1129.
Salts, inorganic, in modification of cow's milk, 186; mineral, function of, in diet, 132; of cow's milk, 150; of woman's milk, 138.
Sarcoma, of brain, 762; of kidney, 641; of spinal cord, 819; of stomach, 345.
Scabies, 935.
Scalp, pustular eczema of, 926; ringworm of, 936; seborrhea of, 923.
Scarlatina (see Scarlet Fever), 952.
Scarlatiniform erythema, causes of, 970.
Scarlet fever, 952; albuminuria in, 966; angina in, 963; blood in, 963; cellulitis in, 964; complications and sequelæ, 963; desquamation, 957; diagnosis, 969; diphtheria in, 963, 969; duration of infective period, 954; eruption, 956; etiology, 952; heart in, 919; incubation of, 953; invasion, 955; joints in, 967; kidneys in, 966; lesions, 955; lymph nodes in, 964; mode of infection, 954; mortality in, 971; myocarditis in, 967; nervous system in, 969; other infectious diseases with, 969; otitis in, 965; predisposition to, 953; prognosis, 971; prophylaxis, 971; quarantine in, 971; relapses, recurrences and second attacks, 962; symptoms, 955, 963; surgical, 962; throat in, 963; treatment, 972.
Sclerema, 121; in cholera infantum, 365.
Scorbutus, 231; etiology, 232; lesions, 235; symptoms, 234; diagnosis, 238; treatment, 239; rickets with, 238; stomatitis in, 280.
Scrofula (see Adenitis, Tuberculous), 867; (see Tuberculosis), 1067.
Scurvy (see Scorbutus), 231.
Seborrhea, 923.
Senses, special, development of, 26.
Sepsis, in newly born, 82.
Serum-therapy of diphtheria, 1047.
Serum-therapy of cerebrospinal meningitis, 939.
Shiga bacillus (see Bacillus of Dysentery), 357, 374.
Shower bath, 57.
Sight, when developed, 25.
Singultus, 702.
Sinuses of dura mater, thrombosis of, 757; lateral, in otitis, 943.
Skin, diseases of, 920; of newly born, 920; care of, in newly born, 4.
Skull, asymmetry of, in birth paralysis, 780; in rickets, 249; sutures, syphilitic nodes on, 1118.
Sleep, disorders of, 712; disturbed, 7, 713; with hypertrophy of tonsils, 309; in intestinal indigestion, 397; in rickets, 248; with adenoids, 296; excessive, 714; inspection during, 33; proper periods of, 5.
Sleeplessness, 712.
Smallpox, protection against (see Vaccination), 997.
Smell, sense of, when developed, 27.

Snuffles, syphilitic, 462, 1110.
Spasm, carpopedal (see TETANY), 677; of larynx, 981; habit, 700; nodding, of the head, 701; rotary, of the head, 701.
Speech, disorders of, 710; when acquired, 27.
Spina bifida, 793; with congenital hydrocephalus, 773.
Spinal cord (see CORD, SPINAL), 796.
Spine, angular curvature of, in caries, 910; caries of, 907; physical examination, 909; causing compression of cord, 804; curvature of, in hip disease, 915; hysterical affections, referable to, 706; in rickets, 252; lateral deviation of, 911; Pott's disease of (see SPINE, CARIES OF), 907.
Spirocheta pallida, in syphilis, 1103.
Spleen, diseases of, 876; Banti's disease, 878; amyloid degeneration of, 878; enlargement of, 877; in acute disease, 877; in chronic cardiac disease, 593; in chronic disease, 878; in cirrhosis of liver, 441; in leukemia, 849; in malaria, 1143; in pseudoleukemic anemia, 844; in rickets, 246, 878; in secondary anemia, 842; in typhoid fever, 1061; with amyloid liver, 441; in diphtheria, 1061; in hereditary syphilis, 1107; in late syphilis, 1122; in tuberculosis, 1091; new growths and tumors of, 878; position and methods of examination, 877; weight, 876.
Sprue (see THRUSH), 282.
Sputum, means of obtaining, for examination, 1095.
Stammering, 711.
Staphylococcus, in furunculosis, 931; in acute bronchopneumonia, 496; in diphtheria, 1023; in empyema, 563.
Starch, objections to, as food of young infants, 132.
Status lymphaticus, 49, 891.
Stenosis, laryngeal, in acute catarrhal laryngitis, 469; in syphilitic, 473; of pylorus, 321; dilated stomach in, 341.
Sterilization of milk, 152; changes produced by, 152; at 212° F., 153; at low temperature, 153; indications for, 154; limitations of, 154; methods of, 155.
Still's disease, 902.
Stimulants, 54.
Stomach, diseases of, 315; absorption from, 318; bacteria of, 318; congestion of, in acute intestinal indigestion and diarrhea, 358; digestion in, 315; dilatation of, 304; in chronic gastric indigestion, 339; in rickets, 254; hemorrhage from, 345; in newly born, 104; in scurvy, 238; inflammation of (see GASTRITIS), 335; malformations and malpositions of, 321; ulcer of, in chlorosis, 844; tuberculosis of, 1081; tumors of, 345; ulcer of, 342; in newly born, 342; from acute gastritis, 342; tuberculous, 337; simple, perforating, 343.
Stomach washing, 62; indications for, 63.
Stomatitis, aphthous (see HERPETIC STOMATITIS), 277; catarrhal, 276; diphtheritic, 285, 1026; follicular (see HERPETIC STOMATITIS), 277; gangrenous, 285; gonorrheal, 284; herpetic, 277; in newly born, 84; parasitic (see THRUSH), 281; syphilitic, 284; ulcerative, 278; vesicular (see HERPETIC STOMATITIS), 277.
Stone, in the kidney, 646; in the bladder, 647.
Stools, blood in, from ulcer of stomach, 344; in catarrhal ileocolitis, 382, 384; in membraneous ileocolitis, 385; in intussusception, 416; in purpura, 857; fat in, 194, 360; green, explanation of, 360; in acute intestinal indigestion and diarrhea, 360; in cholera infantum, 363; in acute ileocolitis, 374, 376, 381, 382; indication of improper feeding, 192, 193; mucus in, in malnutrition, 224.
Strabismus, with tumor of crura cerebri, 766.
Streptococcus, angina (see MEMBRANOUS TONSILLITIS), 300; pyogenes, in acute bronchopneumonia, 495; in complications of scarlet fever, 963; in dermatitis gangrenosa, 932; in diphtheria, 1023, 1027, 1035; in empyema, 563; in measles, 984; in peritonitis, acute, 445; in pseudodiphtheria, 301; in scarlet fever, 952.
Stridor, in catarrhal spasm of larynx, 467; in acute catarrhal laryngitis, 469; congenital, 120.
Strophulus (see MILIARIA RUBRA), 922; (see URTICARIA), 934.
Stuttering, 710.
Sucking, 315; as a bad habit, 714.
Sudamina, 921.
Sudden death, chief causes of, 48, 49.
Sugar, cane, derivatives in digestion, 318; substitute for milk sugar, 132, 182, 185; milk, determination of, 140; percentage of, in woman's milk, 182; milk, derivatives in digestion, 318; percentages of, in modification of cow's milk, 181; solutions, rules for making, 191, 192; stools in, difficult digestion of, 369; symptoms of excess of, in food, 203, 204, 207.
Summer diarrhea, 357.
Suppositories, in chronic constipation, 407; proctitis from long use of glycerin, 432.
Suprarenal capsules, in syphilis, 1109; in tuberculosis, 1082.

Sutures, closure of, 22; premature ossification of, 24.
Swallowing, of foreign bodies, 346.
Sweating, in infants, 920; of head in rickets, 248; in tuberculosis, 1090.
Symptomatology, general considerations of, 31.
Synovitis, acute purulent (see ARTHRITIS, ACUTE), 900; scarlatinal, 967.
Syphilis, 1103; acute osteomyelitis in, 1106; bone lesions in, 1106; chronic osteoperiostitis in, 1114; dactylitis in, 1115; of larynx, 473; pseudoparalysis in, 1114; spleen in, 878; acquired, 1103.
Syphilis, hereditary, 1104; adenitis in, 866; bones, 1106; Colles' law, 1105; communicability of, 1105; diagnosis, 1124; etiology, 1104; evidences of, in fetus, 1106; hemorrhages, 1113; lesions, 1105; prognosis, 1125; prophylaxis, 1127; pseudoparalysis, 1114; rhinitis of, 462; spleen, 1107; symptoms, 1109; at birth, 1109; treatment, 1128; salvarsan, 1129; late hereditary, 1125; bones, 1128; skin, 1122; liver, 1107; nervous system, 1123; spleen, 1107; teeth, 1117; tertiary, chronic laryngitis in, 473; intubation for, 474.
Syringe, nasal, 61.
Syringing, nasal, 61; of mouth and pharynx, 61.
Syringomyelocele, 799.

Tachycardia, 608.
Tenia, cucumerina or elliptica, 423; nana, 424; saginata or mediocarnellata, 423; solium, 423.
Tapeworms, 423.
Taste, when developed, 27.
Teeth, 28; eruption of first set, 28; permanent set, 29; care of, 3; decayed (see DENTAL CARIES), 272; cause of adenitis, 862; delayed, in rickets, 254; Hutchinson's, in syphilis, 1118.
Temperature, at birth, 36; in childhood, 36; subnormal, 37; raised by artificial heat, 37; variations of, in health, 36; of nursery, 9.
Tenesmus, from proctitis, 433; in intussusception, 416; in membranous ileocolitis, 381; treatment of, 434.
Testicle, retraction of, with renal calculi, 647; syphilis of, 1107; undescended, 652.
Tetanus, in the newly born, 90.
Tetany, 677.
Therapeutics, general consideration of, 51.
Thomsen's disease, 702.
Thoraplasty, 573.
Thorax, description of, 476; measurements of, 20, 24; causes of deformity of, 24.
Threadworms (see WORMS, INTESTINAL), 427.
Throat, diseases of (see PHARYNX AND TONSILS), 288, 300.
Thrombosis, 612; cachectic, of dural sinuses, 757; in diphtheria, 1028, 1036; in infectious diseases, 613; inflammatory, of dural sinuses, 757; of internal jugular vein, 613; of lateral sinus in acute otitis, 943; of sinuses of dura mater, 758; of the aorta, 613; of the vena cava, 613; septic, of dural sinuses, 758.
Thrush, 281.
Thymus, enlargement of, causing convulsions, 49; in status lymphaticus, 891; tuberculosis of, 1081.
Thyroid extract in cretinism, 887.
Thyroid gland, congenital absence of, in cretinism, 882.
Tibia, deformities of, in rickets, 253; sabre-blade deformity in syphilis, 1121.
Tinea tonsurans, 936; treatment, 937.
Toes, clubbing of, in congenital heart disease, 583.
Tongue, diseases of, 269; congenital hypertrophy of, 268; epithelial desquamation of, 269; geographical, 270; inflammation of, 270; malformations of, 267; ulcer of frenum, 271.
Tongue-sucking, 718.
Tongue-swallowing, 271.
Tongue-tie, 268.
Tonics, 54.
Tonsils, diseases of, 300; anatomy of, 300; chronic hypertrophy of, 308; diphtheria of, 1024, 1029; hypertrophy of, cause of asthma, 488; hypertrophy of, in rickets, 255; removal advised in tuberculous adenitis, 872; with adenitis, 865; membrane upon, in scarlet fever, 963.
Tonsillitis, membranous (pseudodiphtheria; streptococcus angina; croupous tonsillitis), 300; diagnosis, 303; prognosis, 303; treatment, 304; bronchopneumonia in, 302; follicular, 305; diagnosis, 306; treatment, 306; in rheumatism, 1152; phlegmonous, 307; ulceromembranous (Vincent's angina), 304.
Tonsillotomy, 310.
Top-milk, 152.
Torticollis, 703; congenital, 703; from cervical Pott's disease, 703, 908; from hematoma of sternomastoid, 97; hysterical, 707; in phlegmonous tonsillitis, 307; in retropharyngeal abscess, 303; rheumatic, 307; spasmodic, 704.
Touch, when developed, 26.
Toxemia, in intestinal indigestion, chronic, 397; vomiting in, 329; in acute gastric indigestion, 329.
Trachectomy, for foreign body in larynx,

475; in laryngeal diphtheria, 1058; in retro-esophageal abscess, 314.
Transfusion, of blood, 68.
Trousseau's sign, in Tetany, 682.
Trypsin, 310.
Tubercle bacilli (see BACILLUS OF TUBERCULOSIS), 1071.
Tuberculin test in herds, 145; in diagnosis, 1099.
Tuberculosis, 1067; age, 1067; bacillus of (see BACILLUS OF TUBERCULOSIS), 1067; in milk, 145; bronchial lymph nodes in, 1095; clinical forms of, 1082; bronchopneumonia, 1075, 1086; chronic phthisis, 1095; chronic pulmonary, 1084; congenital, 1069; diagnosis of pulmonary, 1094; of bronchial glands, 1078; general, 1082; etiology, 1082; following measles, 987; following pertussis, 1012; frequency, 1067; general, in infants, 1082; in older children, 1082; hemoptysis, 1039; incipient, symptoms in, 1082; intestines, 391, 1081; intra-uterine infection, 1069; kidney, 639, 1081; lesions, 1073; mesenteric, 391; miliary, of the lungs, 1084; mode of infection, 1069; of larynx, 472; of lymph nodes, cervical, 867; of the skin, 1100; paths of infection, 1072; pericarditis in, 588; physical signs, 1092; pleura in, 559, 1080; predisposing causes, 1069; prognosis, 1101; prophylaxis, 1101; spleen, 1081; sputum, means of obtaining, 1095; treatment, 1102; tuberculin tests, 1099.
Tuberculous adenitis, 867; bronchial glands, 1078, 1095, 1098; meningitis, 747; nephritis, 639; ostitis, 905; pericarditis, 589; peritonitis, 449; pleurisy, 559; pneumonia, 1086, 1092.
Tumor, abdominal, in intussusception, 415; in stenosis of pylorus, 324; cerebral, 762; pituitary, 767; tuberculous, 1080, 1081; fatty, in cretinism, 884; of spinal cord, 819; mediastinal tuberculous lymph nodes, 1097; of spleen, 878, 1107.
Tunica vaginalis, hydrocele of, 654.
Turpentine stupe, preparation of, 58.
Tympanites in acute peritonitis, 446; in intestinal indigestion, 396; in rickets, 254; in typhoid fever, 1061.
Typhoid fever, 1058; bacillus of, in milk, 146; complications and sequelæ, 1063; diagnosis, 1064; etiology, 1067; lesions, 1060; prognosis, 1065; scarlatiniform erythema in, 970; symptoms, 1060; treatment, 1066; paratyphoid, 1058; fetal, 1058; infantile, 1058.

Ulceromembranous tonsillitis, 304.
Ulcers, catarrhal, of intestine, 377; follicular, of intestine, 377; following tuberculous adenitis, 870; of stomach, 342, 1081; tuberculous, of bronchial lymph nodes, 1097; tuberculous, of intestine, 391; tuberculous, of skin, 870; syphilitic, 1122; typhoid, 1060.
Umbilical vessels, arteritis, in newly born, 83; phlebitis, in newly born, 82; fistula, 115.
Umbilicus, hemorrhage from, in newly born, 103; hernia of, 116; inflammation of vessels, in newly born, 83; tumors of, 115.
Uremia, acute, in scarlet fever, 969; in acute nephritis, 631; in chronic nephritis, 637.
Urethra, hemorrhage from, in newly born, 105.
Urethritis, 653; gonorrheal, 653.
Uric acid, in early infancy, 616; infarctions, in kidney, 627; causing hematuria, 105.
Urine, arrest of secretion (see ANURIA), 621; albumin in, 617; blood in (see HEMATURIA), 619; composition of, 616; daily quantity of, 615; examination of, 41; hyperacidity of, in rheumatism, 1155; incontinence of, 662; with adenoids, 291; in diabetes, 1091; retention of, in myelitis, 779; in typhoid, 1013; in vesical calculus, 667; in infancy and childhood, 615; methods of collecting, 41, 615; microscopical examination of, 616; physical character of, 616; pus in (see PYURIA), 620; reaction of, 616; specific gravity of, 616; sugar in, 617; uric acid in, 616.
Urogenital organs, tuberculosis of, 1081.
Urogenital system, diseases of, 615.
Urticaria, 933; following diphtheria antitoxin, 1050; in intestinal indigestion, 397; papulosa, 934; scarlatiniform rash with, 970.
Uvula, bifid, 268; diphtheria of, 1024; elongation of, 289; cause of asthma, 488; edema of, 289; inflammation of, 289.

Vaccination, 997; choice of lymph, 997; methods of, 999; revaccination, 997.
Vaccinia, 997.
Vaccines, 57.
Vaginitis, 655; simple, 655; gonococcus, 657.
Vapor bath, 59.
Varicella, 994; symptoms, 995; diagnosis, 996; gangrenosa, 932, 995; treatment, 996.
Vegetables, third to sixth year, 212.
Veins, internal jugular, thrombosis of, 613; umbilical, 575.
Veins, abdominal, dilated, in cirrhosis of liver, 441; in thrombosis of vena cava, 613.

Ventricles, cardiac, relative thickness of, 577.
Vertigo, in cerebral abscess, 760; in cerebral tumor, 764; in functional disturbances of the heart, 608.
Vesical calculi, 667.
Vincent's angina (see Ulcero-Membranous Tonsillitis), 304.
Viscera, abdominal, transposition of, 350; frequency of inflammations of, 42; hemorrhages of, in newly born, 101; vitamins, 233.
Voice, hoarse or husky, with adenoids, 297; nasal, with hypertrophy of tonsils, 309; with adenoids, 296; in diphtheritic paralysis, 834.
Volvulus, fetal cause of malformations, 349.
Vomiting, 329, 331; from overfilling the stomach, 329; in acute gastric indigestion, 329; in hypertrophic stenosis of pylorus, 322; in acute intestinal obstruction, 329; in peritonitis, 329; in nervous diseases, 329; at onset of acute febrile disease, 330; from toxic substances in the blood, 330; reflex, 330; from habit, 330; chronic, 331; of blood, in ulcer of stomach, 343; stercoraceous, in intussusception, 414; cyclic, 331; treatment, 334.
von Pirquet's test for tuberculosis, 1099.
Vulvitis, gangrenous, 662.

Walking, causes which prevent, 25; delayed, in rickets, 255; when attempted, 25.
Wasting, in tuberculosis, 1090; simple (see Marasmus), 221.
Water, function of, in diet, 133.
Weaning, 177; time for, 177; indications for, 178; sudden, 177.
Weather, hot, prophylaxis against diarrhea in, 366.
Weight, 15; at birth, 16; curve during first few weeks, 16; curve of first year, 17; from second to fifth year, 18; of older children, 19; from birth to sixteenth year, 20; loss of, in acute inanition, 219; stationary, indications, 195; symptoms of unsuccessful nursing, 171, *et seq.*
Werlhof's disease (see Purpura), 854.
Wet dressings vs. poultices, 58.
Wet-nurse, in acute indigestion and diarrhea, 369; in gastro-enteric intoxication, 358; in acute inanition, 221; selection of, 176; dangers from syphilitic, 1127.
Wet-nursing, 176; vs. artificial feeding, 168; indications for, 169; disadvantages of, 166.
Wheal, in urticaria, 934.
Whey, 162; wine, 162.
White-swelling of knee, 916.
Whooping cough (see Pertussis), 1003.
Widal's test, in typhoid fever, 1064.
Worms, intestinal, 427; tape-, 424; round-, 425; thread-, 427.
Wrist, enlarged epiphyses in rickets, 253.
Wry-neck (see Torticollis), 703.

X-ray, in empyema, 565; in gastric retention, 324; in osteogenesis imperfecta, 896; in osteoperiostitis, 1120; in pneumonia, 537, 565; in syphilitic dactylitis, 1117; in syphilitic osteoperiostitis, 1115.

Zoolak, 161.